IMSA

INTERNATIONAL MEDICAL SCIENCES ACADEMY

April - June 2009
Vol. 22 No. 2

BOARD OF TRUSTEES

President
Dr. K. Jagadeesan

Vice-President
Dr. R.R. Thukral

Members
Dr. (Miss) S. Padmavati
Dr. Ramdas M. Pai
Dr. Sandip Mukherjee
Dr. K.B. Sharma
Dr. P.V. Chalapathi Rao
Dr. N.K. Ganguly
Dr. Harvinder S. Luthra (U.S.A.)
Dr. Shaheena Asif (Pakistan)

Central Executive Committee (CEC)

Dr. P.K. Dave
Chairman, India Region
Dr. H.K. Chopra
Secretary General
Dr. Kamlesh Chopra
Treasurer
Dr. P.D. Gulati
Editor - JIMSA
Dr. I.P.S. Kalra
Immediate Past Secretary General

International Advisors

Sir Roy Calne	**England**
Allistair D. Beattie	**Glasgow**
H. Klinkmann	**Germany**
Susan Lim	**Singapore**

Secretarial Correspondence :
INTERNATIONAL MEDICAL SCIENCES ACADEMY
National Medical Library Building
Ansari Nagar, Ring Road, New Delhi - 29
Tel./Fax: 26589660, 26588226
E-Mail : imsahq@ndf.vsnl.net.in
Website : www.imasonline.com
www.jimsaonline.com

PRESIDENT WRITES



Dear Fellows and Members,

The heat of summer will soon be on us and JIMSA comes as a whiff of air. Hansen' disease has made a dramatic downturn after the widespread use of multi drug therapy that is instituted now. However, health care delivery problems continue to taunt us. The benefits of advances in science must be made available to all including the less fortunate ones in a meaningful manner. Economic downturns and logistics may thwart our effort. Not withstanding all this, organizations like ours should be in the forefront to increase awareness and to facilitate good healthcare.

The panorama of topics that we have in our JIMSA will help to broaden our view and impel us to give all specialities due regard.

I am sure, you will enjoy reading this issue just as I have.

K. Jagadeesan

Dr. K. Jagadeesan
President, IMSA

Free Access

IMSA/ JIMSA WEBSITE

Free Access

www.imsaonline.com
www.jimsaonline.com

All fellows and members of IMSA can have **access** to the site and get information about its objectives, benefits to the fellows/members, chapters and their activities including seminars, refresher courses, rural CME;s etc. and also IMSACON - a regular annual event of international standard; *application form for enrollment as fellow/member can also be downloaded. Fellows - members and even not fellows - members can have access to full text in the quarterly journal - jimsa from July - Sept. 2003 onwards by putting their E-mail address under* ***'user name'*** *and using the* ***password 'UserJimsa'.***

Dr. Pinnamaneni Narasimha Rao International Award

Appeal by Vice-President IMSA



Dr. P. Narasimha Rao
Ex. President, IMSA World H.Q.



Dr. R.R.Thukral
Vice President IMSA World H.Q.

Dear Fellows and Members

You are aware late Dr. P. Narasimha Rao, an international figure both in academic and teaching had been the President of this prestigious organization for more than a decade from 1990 to 2002. He was President of Medical Council of India and Vice Chancellor of various Universities. He had to his credit several outstanding contributions to the medical fraternity till his death . He had been in close association with IMSA since its very inception in 1981. The Academy has flourished tremendously during his tenure as President . Keeping in view his status, services rendered to the mankind and on the insistence of senior Fellows, the Academy has established an International Award in his honour named 'Dr. Pinnamaneni Narasimha Rao International Award', on the lines of Dr. B.C. Roy National Award. Substantial funds are needed for this pestigious award. Initially, the family of Dr. P. Narasimha Rao has contributed a fair amount of money and has also assured to contribute more.

I appeal to all our Fellows and Members to contribute generously for this noble cause in the memory of this dedicated acadamecian - Dr. P. Narasimha Rao. A separate account has been opened for this Award.

(R.R.Thukral)

IMSA Chapter Activities

CME Tamil Nadu Chapter

12-4-2009 ***Dr.G. Jayaraj*** *: Growing Importance of Health Care and Industry*
10-5-2009 ***Dr. Mourougayan :*** *Scope of Plastic Surgery*
14-6-2009 ***Dr. Ashok Chacko :*** *Recent Advances in Gastrointestinal Endoscopy*

CONGRATULATIONS

Dr. Hem Chandra, FIMSA, Additional Medical Superintendent, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, UP has taken over as HOD, Hospital Administration on 27th June, 2009.

Change in Address

If the address of any Fellow/Member of IMSA has been changed , he may please intimate his latest address to IMSA, WHQ, New Delhi for future communication.

Dr.R.R.Thukral, Vice - President IMSA,WHQ

Election of Fellows and Members (April - June 2009)

Fellows

Dr. Dharmapuram Anil Kumar *(Hyderabad)*
Dr.D.Keshava *(Karnataka)*
Dr.Muthukumaran Rangarajan *(Coimbatore ,TN)*
Dr.Tarsempal Singh *(Amritsar)*
Dr.Shridhar Dwivedi *(Delhi)*
Dr.Jugal Kishor Sharma *(New Delhi)*
Dr. Pramod Kumar Gupta *(Noida ,UP)*
Dr.Binod K.Khaitan *(New Delhi)*
Dr. Sajal De *(Bhopal, Madhya Pradesh*
Dr. Uma Kumar *(New Delhi)*
Dr. Niraj Gupta *(New Delhi)*
Dr.T.S.Chandrasekar *(Chennai)*
Dr.Sheffali Gulati *(New Delhi)*
Dr.S.C.Tiwari *(New Delhi)*

Members

Dr. Puneet Kumar Agarwal *(New Delhi)*

IMSA Fellows/Members Directory 2007

Dear Fellows and Members

International Medical Sciences Academy has published Directory of IMSA Fellows and Members containing information about their mailing addresses, telephone nos. email addresses, wherever available. The Directory was released at the inaugural function of IMSACON 2007 held at Manipal, Karnataka in November, 2007. I shall request you to send a demand draft of Rs. 250 soon to enable us to send to you a copy of the Directory by post. You can also collect in person from IMSA office if you so wish.

Secretary General, IMSA

Suggestions to Enhance Image of Medical Profession and Improve Doctor-Patient Relationship

President, Vice President and Trustees of IMSA have stressed that IMSA must engage itself in enhancing the image of Medical Profession by organizing seminars/conferences on various issues relating to medical profession, medico legal, patient — doctor relationship protocol of drug trials and research etc. It was also desired that suggestions be invited from all fellows and members, for improving relationship among doctors and patients.The Fellows and Members are, therefore, requested to send their suggestions & ways and means to IMSA World Headquarter at New Delhi, for enhancing image of medical profession and improving doctor — patient relationship.

Secretary General, IMSA

REGISTER FOR IMSACON 2009

Annual Conference of International Medical Sciences Academy IMSACON 2009, 24th -25th October, 2009 at Government Medical College , Chandigarh.

Organizing Secretary: Dr.A.K.Attri ***Scientific Chairman:*** Dr. B.S.Chavan

Theme : "Recent advances in Medicine and allied specialities inMedicine"
Venue : Auditorium , Sarai Building GMCH Sector-32 Chandigarh, India
Host : Government Medical College, Chandigarh, India
Visa: Visa is required for foreigners for India and must be obtained before the visit.
Information Brochure on website: www.imsaonline.com
Avail Opportunity : "Apply for IMSA Fellowship"

For details contact:

Dr. A.K.Attri
Organizing Secretary,
Prof. & Head , Deptt. Of Surgery
Govt Medical College & Hospital, Sector-32, Chandigarh
imsacon-2009@gmail.com Mo.9646121561

Dr. H.K. Chopra
Secretary General, IMSA WHQ
2nd floor, National Medical Library
Ring Road, Ansari Nagar, New Delhi-110029
imsawhq06@gmail.com Mo.9811090204

JIMSA

JOURNAL OF INTERNATIONAL MEDICAL SCIENCES ACADEMY

April - June 2009
Vol. 22 No. 2

EDITORIAL BOARD

Editor
P.D.Gulati *(Delhi)*
Joint Editor
S.N.A. Rizvi *(Delhi)*
Asstt. Editor
Pradeep Chattree *(Delhi)*

MEMBERS

K.Jagadeesan (Chennai)
Sandip Mukherjee (Delhi)
D.D.S. Kulpati (Delhi)
K.B. Logani (Delhi)
Indira Bahl (Delhi)
S.K. Bhargava (Delhi)
I.P.S.Kalra (Delhi)
P.N. Renjen (Delhi)
H.K.Chopra (Delhi)
Tarun Gupta (Delhi)
Chintamani (Delhi)
H.S. Rehan (Delhi)
S. Trivedi (Delhi)
Achal Gulati (Delhi)
Ashok Grover (Delhi)

ADVISORY BOARD - National

K.B. Sharma (Delhi)
O.P. Gupta (Ahmedabad)
Habibullah Zargar (J & K)
P.M. Dalal (Mumbai)
G.S. Sainani (Mumbai)
S.A. Tabish (J & K)
M. Sachdev (Delhi)
Sukumar Mukerjee (Kolkata)
R.R. Thukral (Delhi)
R.K. Bali (Delhi)
S.S. Sethi (Delhi)
Rattan Singh (Delhi)
Sham Agarwal (Delhi)
P.S. Gupta (Delhi)
P.K. Dave (Delhi)
V.K. Dada (Delhi)
K.K. Malhotra (Delhi)
H.P.S. Sachdev (Delhi)
Kamlesh Kohli (Delhi)
T.K. Biswas (Kolkata)
R.R. Kasliwal (Delhi)
S.P. Aggarwal (Delhi)
Naresh Trehan (Delhi)
S.D. Jeyaraj (Chennai)
N.S. Neki (Amritsar)
Richa Diwan (Delhi)
M.Suresh Kumar (Chennai)
Sheela Chuttani (Delhi)
Prema Bali (Delhi)
Sudarshan K. Agarwal (Delhi)
Veera Hingorani (Delhi)
C.V. Raghuveer (Karnataka)

ADVISORY BOARD - INTERNATIONAL

Swaraj Singh (USA)
Leela Prasad (USA)
J.Heinrich Joist (USA)
Sir Roy Calne (U.K.)
Alistair D. Beattie (USA)
H. Klinkmann (Germany)
Susan Lim (Singapore)
J.D. Williams (U.K.)
Harvinder S.Luthra (USA)

Annual Subscription for non fellows & members

Inland	Rs. 500
Overseas	$ 200
Single copy	Rs. 150

FROM EDITOR'S DESK

Dear collaegues

India has almost half of the world's population of leprosy. With the enforcement of National Leprosy Eradition Programme, our country is set to achieve national health policy target of less than one per 100000 population; which is really commendable. This has been possible by strictly adhering to WHO global strategy-guidelines. The present issue contains an editorial by ***Prof. R.C.Sharma*** which amply highlights the recommendations for Leprosy control programme. The role of community surveillance, especially in the rural population, is another important facet covered in this issue. In another article, on hospital burden of minor head injuries, the need for an early CT scan and surgical intervention, if needed, is well emphasized by the authors. Cause and effect relationship of H-pylori and bronchial asthma has been discussed in another article. In update on benign thyroid disorder, authors have attempted to discuss the controversial issues relating to surgical intervention and have reccommended total thyroidectomy as a valuable option. The pandemic of the "Bird Flu" and its hazards are well highlighted in another update article, published in the issue. Value of immunosuppressive therapy in steroid resistant nephrotic syndrome is being recognized during last two decades; the place of non steroidal immunosuppressants is now well the established in some of the morphological types of glomerulonephritics including lupus nephritis, as discussed brilliantly in the therapy update contributed by **Prof. S.C. Tiwari.**

The present issue also contains several case reports coming from different specialities of the medicine and surgery. I am confident that some of the newer indiginously researched procedures in the field of gyneceology and obstetrics, reported by Dr. R.J.Sreenivasan, will be of interest to those interested in the field and will provide an impetus to several readers of JIMSA.

P. D. Gulati

JIMSA BEST PUBLISHED ARTICLE AWARDS 2008 - Declared

The following articles have been selected for the **"JIMSA Best Published Article Award 2008"** as per ranking given below:

1st Best Article Award : **"Efficacy of Herbal Medication for Echancing the Breast and Teats in Female Wistar Rats"** *R.Karunanithi, V.Pugalendi, TMR Paniker, K.Jagadeesan, M.Paul Koraoth.*

2nd Best Article Award : **"MRI Changes in Compressive Myelopathy in Fluorosis – Study of 18 cases from North West India"**. *Ashok Panagariya , Ravindra Singh, Paresh Sukhani, Bhawna Sharma.*

3rd Best Article Award : **"Preoperative Evaluation of Colorectal Carcinoma by Computed Tomography"** *Rajul Rastogi , Satish Kumar Bhargava, Satish Kachhawa.*

Each award consists of a medal, citation and cash prize: the awardee is required to preferably receive the award at the forthcoming IMSACON 2009 at Chandigarh on 24th & 25th October 2009 (contact urgently Dr.H.K.Chopra, Secretary General IMSAWHQ). In case he or she is unable to attend IMSACON 2009, he/she should intimate the editor JIMSA, accordingly.

Editor

Editorial Correspondence: *All corresspondance are to be addressed to* ***Editor JIMSA***
National Medical Library Building, Ansari Nagar, Ring Road, New Delhi - 110 029 India
Tel./Fax: 26589660, 26588226 E-Mail : imsahq@ndf.vsnl.net.in Website : www.jimsaonline.com

Copy Right *No part of this publication may be reproduced, or transmitted in any form or by any means, electronic or mechnical, including photocopy without written permission from the Editor.*
The Editor disclaims any responsibility or Liability for statements made and opinions expressed by authors.
IMSA is now on website and our address is ***www.jimsaonline.com*** */* ***www.imsaonline.com***
JIMSA is indexed by Experta Medica, Indian Science Abstracts/Chetna, Bibliographical Data base

RANBAXY
SUPER SPECIALITIES



In anemia due to CKD

CERITON

rec-Hum-Erythropoietin 2000 IU / 3000 IU / 4000 IU / 10000 IU PFS

For better life



RANBAXY
SUPER SPECIALITIES



In CMV Prophylaxis & Treatment

Ganguard

Guards against CMV





RANBAXY
SUPER SPECIALITIES



To Improve Graft Survival

BAXMUNE

Sharing Life



RANBAXY
SUPER SPECIALITIES



In Invasive Fungal Infections

Vorzu

Voriconazole 200 mg Tab/Inj

Your Valued Choice

EDITORIAL

STRATEGIC APPROACH TO OVERCOME THE BURDEN OF LEPROSY IN INDIA

Leprosy is the oldest disease known to mankind. It is a chronic infectious disease caused by mycobacterium leprae. Leprosy trends in prevalence and case- detection have varied widely among countries and within the country. India has almost half of the worldwide leprosy cases, distributed unevenly concentrated mainly in Southern states. There has been significant reduction in disease burden of leprosy in every country of the world including India. This achievement was made possible by implementation of multi drug therapy and intensified political commitment.

The Leprosy control programme in India was renamed in 1982 as the National Leprosy Eradication Programme [1]. The National Health Policy 2002 set the goal for elimination of leprosy. India is trying to achieve the elimination of leprosy as a health problem by reducing the prevalence to below l/l0,000 population[2]. It is possible to eliminate the leprosy but will take much longer time to eradicate the it where completely.

The most relevant epidemiological measure of the burden of leprosy is the incidence of disease. It is not feasible to measure the incidence of leprosy directly. Therefore, the "case-detection rate" is used as a proxy for the incidence. This may reflect under-reporting and thus the number of cases detected will be generally lower than the actual number. The other method could be related to the registered prevalence of leprosy eases which is useful indicator of leprosy elimination, but it is not adequate to reflect changes in epidemiological trend of leprosy. Special stigma related nature of the disease causing impairments and disabilities is another burden[3].

There is a clear consensus now that the world and countries are in a very favourable situation with regard to disease burden as measured by prevalence, transmission of disease as measured by case-detection and extent of disability due to leprosy as measured through the WHO grading system[4].

The WHO has recommended **global strategy** for further reducing the leprosy burden and sustaining leprosy control activities[5].

- Sustain leprosy control activities in all endemic countries.
- Use case detection as the main indicator to monitor progress.
- Ensure high-quality diagnosis, case management, recording and reporting in all endemic countries.
- Strengthen routine and referral services
- Discontinue the campaign approach
- Develop tools and procedures that are home/community-based, integrated and loeally appropriate for the prevention of disabilities/impairments and for the provision of rehabilitation services.
- Promote operational research in order to improve implementation of a sustainable strategy.
- Encourage supportive working arrangement with patients at all levels.

Based on these guidelines, India has taken initiative to accelerate leprosy elimination activities and to sustain quality leprosy services. The new paradigms in National Leprosy Eradication Programme focus on measuring disease burden, improving quality of services, integrated with primary Health Care system, prevention and management of impairments and disabilities, improving community awareness and involvement, support from National Rural Health Mission, programme monitoring and evaluation[6]. Implementation of these activities will result in reducing the leprosy case burden as well as minimize disease related stigma.

The progress achieved by Indian leprosy programme is commendable. The fall in case detection has been remarkable. Case detection rate came down from 3 1.1/10,000 during 2003 to 12.7/10,000 at the end of March, 2007. Prevalence rate of 2.4/10,000 at the beginning of 2004 plummeted to 0.72/10,000 population by March 2007[6]. India has made substantial progress in reducing the leprosy burden and has already achieved elimination of leprosy as a public health problem at national level. This is not the end to achieve the goal of leprosy elimination, another issue should be kept in mind that new cases keep coming even after elimination has been achieved. It appears that India, despite many hurdles, would not be the last country to reach the goal of leprosy elimination.

RECOMMENDED READING

1. ***Agarwal S.P.*** *Final push for elimination of leprosy in India. Indian JLepr 2005, 77;1l.V7*
2. ***Editorial.*** *The debate over leprosy elimination versus sustainability of leprosy services. Indian J. Lepr 2005, 77:299.*
3. ***Joshi P.L., Bharkakaty B.N, Throat D.M****. Recent developments in elimination of leprosy in India. Indian J. Lepr 2007, 79:107.*
4. ***Rao C.K.*** *Leprosy Elimination in India- so near. Indian J. Lepr 2005,73; 207*
5. ***WHO global*** *strategy for further reducing the leprosy burden and sustaining leprosy control activities. Indian J. Lepr 2006, 78:7.*
6. ***Thorat D.M and Joshi P.L.*** *Present status of NLEF in Indian and the paradigm changes. Indian J Lepr 2008, 80:69.*

Prof. R.C.Sharma

Sr. Consultat Dermatorlogist

Former Head , Dept. of Skin and VD

Lady Harding Medical College,

New Delhi, India

MANUSCRIPT SUBMISSION : FOR JIMSA

Check-list

(i) Copyright statement/declaration (not submitted or published elsewhere) signed by all the authors.

(ii) Three hard copies of manuscript with illustrations attached to each; **must send an electronic copy of text with photographes loaded on CD**.

(iii) **Title page :** Title of Manuscript, Name(s) and affiliation of author(s); Institution(s) and city(ies) address of corresponding author**(Tel; Fax; e-mail)**

(iv) The text of the article should contain **Abstract** highlighting objectives, methods, results, conclusions.

(v) Original Article(double-spaced on A-4 size paper): should contain introduction material & methods, results, discussion; **Indian Literature must be referred**, references numbered in text as they appear.

(vi) Update/Review/ Therapy update should have appropriate headings. with reference numbers in the text; Indian Literature cited, wherever available.

(vii) References: maximum number of references for update-30,original-20, Case reports-6:8.

(viii) Each table on seperate sheet; maximum number-4 in original article; 6 in update.

(ix) Photographs/ figures in envelope, each marked figure number on reverse with legends on separate sheet, numbers not to exceed 4 in original, 2 in case report.

(x) Statement signed by all authors regarding adherance to Standard ethical guidelines prescribed by ICMR 2000.

Viva

Complete Nephro Portfolio

DIALYSIS PORTFOLIO

Epofit

FE-10
Iron Sucrose Injection

Acutrol

TOR

Zalat

Lonitab

TRANSPLANT PORTFOLIO

Mycofit-S

Mycofit

Takfa

INTAS

INTAS PHARMACEUTICALS LTD.

Chirubhai Centre, Off. Nehru Bridge, Ashram Road, Ahmedabad - 380 009. INDIA.
Tel. : +91-79-26576855 Fax : +91-79-26576616. Email : personal@intaspharma.com

ORIGINAL

A COMMUNITY SURVEY OF HANSEN'S DISEASE IN RURAL BIHAR

Ranabir PaI, Neloy Sinha, Ankur Barua

Department of Community Medicine, Sikkim-Manipal Institute of Medical Sciences (SMIMS), Sikkim,India
Department of Dermatologv. MGM Medical College & L.SK. HospitaL Kishanganj Bihar-2

Abstract: *Residents of all 60 villages, who gave informed verbal consent to participate in this study were included in the sample and were investigated for presence of Hansen's Disease. Study population of 88973 individuals of all age groups. The collected data were tabulated and analyzed by using the statistical package SPSS (Statistical Package for Social Sciences) version 10.0 for windows. Prevalence rate of Hansen's disease was described in terms of proportion and its 95% confidence Interval. Prevalence of Hansen's disease was estimated to be 25.1 per 10,000 population (95% CI = 22-28/10,000 population). Multi-bacillary cases were 66.82% (95% CI=61%-73%) and childhood (<18 years) Hansen's disease cases comprised of 14.35% (95% CI=10%-19%) among them.*

INTRODUCTION

Since independence, India is striving for Hansen's disease control. India alone represents 64 per cent of the prevalence and 78 per cent of new case detection world wide[1]. The elimination level i.e. prevalence rate less than 1 case for 10,000 populations has been achieved in 16 states of India[2]. The WHO regime which is in vogue now has not gone through any field trial before its recommendation to use as back-bone in control as well as eradication programmes round the world[3].

MATERIALS AND METHODS

From January to June 2005, a study was conducted on 88973 population of the district of Kishanganj, Bihar by a multistage random sampling. Ten blocks of the district were selected by random sampling method. Then from each block, six villages (total 60) were selected by random sampling method. All households in 60 villages were screened. Every participant was interviewed and clinically examined and investigated after informed verbal consent was obtained and photographs of willing patients suffering from Hansen's disease were taken as part of documentation. The WHO criteria were applied for diagnosis of Hansen's disease. Approaches based on clinical classification were applied where reliable facilities for bacteriological examination of skin smears were not available and when classification was in doubt, patients were treated as multi-bacillary leprosy[4]. Data were tabulated and analyzed using the statistical software package of SPSS version 10.0. Results were calculated in the terms of proportions and their 95% confidence level (CL).

RESULTS AND DISCUSSION

In this cross sectional study, a total of 223 patients were diagnosed as suffering from Hansen's disease. The prevalence rate was determined to be of 25.1 per 10,000 population (95% Cl = 22-28 I 10,000 population). Multibacillary (MB) cases comprised of 149 (66.82%, 95% Cl 61%-73%). The proportion of males and females among MB cases were 104 (69.8%) and 45 (30.2%) respectively. Highest proportion of Hansen's cases (36.32%) were found in late adolescents and early adulthood, i.e., in the age group of (18-24) years. The median age of these patients was 23.8 years (lnterquartile range = 19.3years - 35.2 years). The majority (74.9 %) of cases were below 35 years of age. These findings were corroborated in textual facts[5].

It was also reported by other researchers that despite apparently curative antibacterial therapy, one quarter to one third of all patients often developed debilitating and permanent neurological deficits[5]. In this study, a high proportion 198(88.8%) of untreated fresh Hansen's cases with visible deformities of grade II and above (Table) was detected, where trophic ulcers were most common (28.8%); closely followed by ulnar clawing (21.7%). In this study, type I Lepra reaction was present among 9 (4%) of the patients while 5(2%) of them had Erythema Nodosum leprosum.

Table*: Visible Deformities(Grade-II) in patients suffering from Hansen's Disease*



CONCLUSION

There could be shortcomings on the part of local medical practitioners to misdiagnose some MB cases and treat them as PB cases. These inadequately treated MB cases must have remained infectious to the community for a long period of time. Another reason could be related to the potency of MDT which was introduced in 1983 as a quick-fix like solution for controlling the grave situation of Hansen's epidemic in developing countries without going through any extensive field or community trials[3]. Though Hansen's disease is eliminated in many parts of the globe and has recently lost our attention due to improvement in socio-economic conditions and extensive MDT management, this study serves as an eye opener to confirm that Hansen's disease still manifests as a disease of poor and rural people in developing countries like India.

REFERENCES

1. ***WHO(2002)*** *Weekly Epidemiological Records. No.1, Jan.2002.*
2. ***Govt. of India. Annual Report 2003-04****. Ministry of Health & familly Wefare, New Delhi-110024.*
3. ***Gelber R. H. Hansen's Disease, Inn: Kasper D.l. D.l., Fauci A.S. et. al,*** *Ed Harrison's Principles of Internal Medicine, 16th ed, Vol.1, Mcgraw Hill Medical Publishing Division, New York. 2005;966-971.*
4. ***WHO 1998,*** *Tech. Rep. Ser. No. 874*
5. ***Thomas H.R. et al. Hansen's disease.*** *In Freedberg IM et al Eds. Fitzpatricks Dermatology in general Medicine. Vol.26th ed Mcgraw Hill Medical Publishing Division 2003;202:1962*

Correspondence: **Dr. Ankur Barua**, Assistant Professor, Department of Community Medicine, Sikkim – Manipal Institute of Medical Sciences (SMIMS), 5th Mile Tadong, Gangtok – 737 102 India Fax.: 03592-231496, e-mail : ankurbarua26@yahoo.com

With Best Compliments
from RINON Division

UNITED BIOTECH PVT. LTD.

makers of :

AMINOS*Tablets new name of Tablet* **AMINESS-N**
(*Dialysis - sparing essential amino acid*)

LAZON
(*Metolazone 2.5 mg & 5 mg . Tablets*)

MYOTEC
(*Mycophenolate Mofetil 250 mg & 500 mg . Tablets*)

MYOTEC - S
(*Mycophenolate Sodium 180 mg & 360 mg . Tablets*)

TACROTEC
(*Tacrolimus 0.5 mg,1 mg & 5 mg . Capsules*)

UNIFER
(*Iron Sucrose 5 ml Ampoule*)

UNIPRAZ
(*Prazosin HCl 1 mg, & 2 mg Tablets*)

UNITERAZ
(*Terazosin HCl 1 mg, 2 mg & 5 mg Tablets*)

For queries:

Contact **Mr Deepak Arora**, 9811231278
Mr Avishkar Bhatt, 93106124292

GUIDELINES FOR MANAGING MINOR HEAD INJURIES

Amin Tabish, N.A. Lone, M.A. Wani, Abdus Salam

Departments of Accident & Emergency Service, Neourosurgery and Internal Medicine
Sher-e-Kashmir Institute of Medical Sciences, Srinagar (J&K) India

Abstract: *Globally, thousands of people sustain head injury everyday. Early diagnosis and appropriate management improves outcome but is sometimes more difficult to achieve than might be imagined,injury to the brain is most likely to result in death or permanent disability. Estimates of traumatic brain injury (TB1) incidence, severity, and cost reflect enormous losses to individuals, their families, and society. Authors prospectively studied 485 consecutive patients of traumatic brain injury, out of which 280 with GCS of 1 3, 4, and 1 5 were subjected to routine early CT scan of head after 4 hours of reporting to Emergency Patients with penetrating head injury were excluded. 15 % of patients had abnormal CT scans and only 4% needed surgical intervention Though a small number of patients harbour potentially lethal intracranial lesions yet. most of these cases are salvageable if diagnosed and treated early.There is a strong need for improved methods of initial assessment including early CT scan and are treatment subsequently lead to reduction in the number of unnecessary admissions and a more focussed head injury care.*

INTRODUCTION

Traumatic brain injury (TBI) is a non-degenerative, non-congenital insult to the brain from an external mechanical force, possibly leading to permanent or temporary impairments of cognitive, physical, and psychosocial functions with an associated diminished or altered state of consciousness. Mild head injury refers to a traumatically induced physiologic disruption of brain function as manifested by one of the following: a) Loss of consciousness; b) Recent loss of memory for recent events; c) Immediately before or after the accident alteration in mental state at the time of the accident; d) Focal neurologic deficits, which may or may not be transient.

The other criteria for defining ***mild TBI*** include GCS score greater than 12, no abnormalities on CT scan, no operative lesions and length of hospital stay less than 48 hours. The criteria to define ***moderate TBI*** include length of stay of at least 48 hours. GCS score of 9-12 or higher, operative intracranial lesion and abnormal CT scan findings. ***Severe TBI*** is indicated when the GCS score is below 9 within 48 hours of the injury[1-14].

According to the Center for Disease Control (CDC), approximately 1.5 million people sustain traumatic brain injuries throughout the United States each year; of those approximately 1.1 million or 75 percent sustain a mild TBI Each year 1.4 million oeople attend hospitals in England and Wales after suffering a head injury, of whom 150,000 are admitted to hospital[15]. The annual estimated incidence of TBI in the United States and European countries is 200 to 300 patients per 100,000 population[16,17] TBI accounts for approximately 52000 deaths (40%) of all deaths) from acute injuries in the United States. Incidence of mild TBI is about I3 cases per 100,000 people, of moderate TBI-15 and severe TBI-14. Inclusion of prehospital deaths increases that figure to 21 cases per 100,000 people[1-14]. It is estimated that 350,000 patients require hospital treatment for mild head injury each year in the United States, representing 80% of all patients admitted to hospital for head injury[18-19]. Similarly, about I million head-injury patients hospitalised each year in the United Kingdom, one-fiftieth of the population [20]). Head injury causes 10% of new Accident & Emergency admissions in the UK and accounts for about 25% of all emergency observation ward admissions, with 97% of patients admitted for less than 24 hours. Thus mild head injury causes a significant number of visits to emergency departments and a substantial number of hospital admissions in both the United States and Europe.

The decision regarding when to use CT imaging for patients with minor head injury carries significant clinical as well as economic implications. To help physicians identify patients who are at risk of intracranial damage following minor head injury, two sets of guidelines have been proposed: the NOC (the New Orleans Criteria) and CCHR (The Canadian computed tomography head rule). While the NOC applies only to patients with a GCS score of I 5, the CCHR may be used for patients with a GCS score from 1 3 to 1 5

Thc CCE-IR and the NOC are clinical decision rules promulgated to identify which head Injury patients should undergo CT imaging The NOC recommends head CT for patients with minor head injury and any of the findings including headache, vomiting, age, older than 60 years, drug or alcohol intoxication, persistent anterograde amnesia, visible trauma superior to the clavicle, seizure.

Findings that should prompt CT evaluation by the standards of the CCRR include GC score less than 15 at two hours or more after the injury, suspected open or depressed skull fracture, any sign of basal skull fracture, two or more episodes of vomiting, age, 65 years or older, more than 30 minutes of amnesia of events prior to the injury, automobile vs pedestrian crash, ejection from a motor vehicle, fall from more than three feet, or fall from five or more stairs. The CCHR does not apply to persons younger than 1 6 years old or those with a hypocoagulable state. The NOC and CCHR can Jentify patients at high risk of intracranial trauma following minor head injury. The NOC is designed to apply only to patients with a GCS score of 15, whereas the CCHR may be used for patients with a GCS score from 13 to 15. The NOC and CCHR appear equally sensitive in predicting the need for neurosurgical intervention following minor head injury.

MATERIALS & METHODS

We carried out this prospective cohort study in the Emergency Departments of Sher-eKashmir Institute of Medical Sciences (SKIMS), Srinagar (India). SKIMS is a 600- bedded University hospital (with 100 beds for Accident & Emergency Department including 20 beds for the Disaster Management) catering to approximately seven million out of 1, 11, 82,000 population of Jammu

Correspondence: **Dr. S.A. Tabish,** *Head, Accident & Emergency Department, Sher-e-Kashmir Institute of Medical Sciences, Post Box: 826, GPO, Srinagar- 190001, India*

& Kashmir State. SKIMS is the only referral centre in Kashmir for the treatment of head injury. During November 2004 to January 2005. we included consecutive adults who presented with head injury we did standardized clinical assessments before the CT scan. A total of 280 Patients with GCS of' 13,14 and 1 5 were subjected to routine early CT scan (Spiral) of head after 4 hors of reporting to Emergency Department Patients with penetrating head injury ere excluded. No patient with a focal neurodeficit was included in the study Patients with normal CT Scans were discharged from Accident & Emergence\ Department while patients \\ho had abnormal CT Scans were admitted in the hospital further management under Neurosurgery department None of the discharged patients was re-admitted during the study for initial head injury. The main outcome measures include need for neurological intervention and clinically important brain injury on CT.

RESULTS

A total of 1085 head injury patients have been admitted during 2004 that comprise about 3.05% of 35505 total admissions. The study included 280(57.7%) of TBI patients, out of which 180 had GCS of 15, 72 had GCS of 14 and 48 had GCS of 13.44 (15%) had abnormal CT scans (Table1). Patients with GCS of 15 had 11% abnormal CT scans in contrast to 29 per cent in patients with GCS of 13.

Table 1. *Abnormal CT scan in relation to GCS*

GCS	Number of patients	Number of abnormal CT scans
15	160	17(11%)
14	72	13(18%)
13	48	14(29%)

Mild head injury refers to a patient with head trauma who has GCS of 13-15 (20,22). Any other clinical, radiological or surgical finding has been an additional risk. Kraus etal have reported that 80% of head injury admissions in hospital are of mold brain injuries[23-25]. Although these patients comprise the majority of head injury admissions. The consensus on the treatment policy of these patients is still lacking. In a busy trauma centre these patients occupy the majority of beds and utilize the hospital resources 17-18% of these patients have abnormal CT scans[26-27] and only 5-6% need surgical intervention (Table2). Though a small number of patients harbour potentially lethal intracranial lesions yet most of these cases are salvageable with early diagnosis and proper treatment.

Table2: *Category-wise percentage of patients with CT findings and need for surgical intervention*

Mode of injury	No patients	No. of positive scans	Patients requiring surgical intervention
Motor Vehicle Accidents	158(56%)	17(38%)	2(11%)
Fall	78(28%)	13(29%)	1(8%)
Assault	36(13%)	8(18%)	3(37%)
Flying objects	8(3%)	5(11%)	2(40%)

Early CT scanning can detect potentially lethal intracranial lesions and will reduce unnecessary hospital admissions.

Majority of patients were victims of motor vehicle accidents comprising 56 percent followed by falls 28 percent, assault 36 percent, flying objects like cricket ball etc 11 percent.

The abnormal CT scans when correlated with mode of injury that the highest number of abnormal scans (38%) patients hit by flying objects - assault and motor vehicle accidents. This may be directly related to the impact of force hitting the head. A total of 72 scans were abnormal *(Figure)*. Most common was single contusion around 4% patients needed surgical intervention (4 patients with depressed fracture, 3

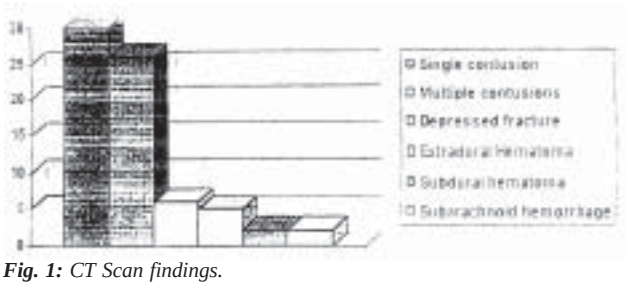


Fig. 1: *CT Scan findings.*

cases with extradural hematoma and, I case with contusion)

Forty-four (44) patients admitted after positive CT scan finding, accounted for 197 bed days. If all patients with brain trauma (including 236 with normal CT findings) had to be admitted for observation it would require additional 415 bed days; it saved 236 bed days for the hospital leading to availability of beds for new emergency admission

DISCUSSION

Minor injuries include patients having loss of consciousness, post traumatic amnesia with a GCS or more than13 in the United States, incidence of mild TBI is about 131 cases per 1,00000 people incidence of moderate TBI is about 10 cases per 100000 people. Incidence of severe TBI is about 14 cases per 100000 people more than 80 percent of TBI admission are mild and the average length of hospital stay was 265 days.

Recommendations for management of these patients vary from routine admission and observations without CT scan to mandatory CT scanning and admission to CT scanning without admission, The timing of the intant CT scan is important it has been shown that 14 to 20 percent of patients admitted with a diffuse brain injury may develop a mass resion within 12 to 24 hours after injury. Thus the danger is particularly evident when patient s with mild head injuries under go every year.

Difficulties encountered in the management of these patients include large number of such patients presenting as brain injuries and a high number of abnormal intracranial lesions which can be missed it routine CT scan is not done. Moreover, in appropriate admission of all patients withy head injury will need additional bed days in the presence of limited resources.

There is wide variation in the indications for obtaining a CTScan. One approach recommends scanning the vast majority of mildly TBI patients withy the only indication being loss of consciousness (LOC) or a period only in the presence of clinical deterioration[26,27,35] and a local neurological deficit. Some have suggested a combinationh of clinicoradiographic parameters, such as CT for all patients with a GCS score of 13 or 14 or a skull fracture. In patients with a GCS score of 15, the decision is left to the discreation of the treating neurosurgeon[38]. Some authors recommends CT on the presence of a skull fracture, persistent headache or vomiting or clinical deterioration [20,30] Purely clinical indications for CT are mild TBI with PTA or LOC, local neurological deficit, vomiting, headache, a serious injury mechanism[36,38] of Master criteria[38].

This SKIMS hospital adopted the policy of routine CT scanning of mild brain injury patients and selective admission of patients with abnormal CT scans. The authors observed that around 15 percent patients had abnormal CT scans. Similar results (1-18%) have been reported elsewhere[33,39,40]. Some authors have recommended routine admission and observation without CT scanning on the basis that it resulted in more efficient use of CT scanning. The authors behave that it results in large number of unnecessary admission that leads to over utilization of scarce resources that could otherwise be directed

to genuine admission.

Patients with admission GCS score of 13,14, or 15 have, minor or mild head injuries. A patient with mild head injury compolicated by an intracranial lesion has more severe sequalae and disability compared with a patient with anuncomplicated injury. In patients with a GCS score of 15 but LOC or PTA, diffuse headache, or vomiting a CT scan of the head must be obtained as soon as possible. In patients with admission GCS score of 14 or 15 but with skull fractures or neurological deficit CT Scan must be obtained immediately. The risk of missing delayed hematomas is overwhelming by the benefits of early hematomas detection. A CT scan should be obtained preferably with 4 hours of injury. Klauber et al[42] have reported a high mortality among very low risk patients in at least one hospital due to inadequate staffing which is a serious concern. One most question the reluctance to employ routinely early CT scanning in all cases of mind brain in jury because the policy of routine hospital admission for all patients of mild head injury results in more hospital charges[43]. Routine admission does not guarantee neurological observations, and the cost difference of CT scanning and X-ray films as screening devices are relatively less than cost of prolonged hospitalization lost wages, medical complication and expenses resulting from man practice.

Current use of cranial computed tomography (CT) for minor head injury nis increasing rapidly, highly variable and inefficient. The Canadian CT Head Rule (CCPR) and New Orleans Criteria (NOC) are previously developed clinical decision rules to guide CT use for p0atients with minor head injury and with Glassow Coma Scale (GCS) scores of 1500 15 for the CCHR and a score of 15 for the NOC. Howe3ver, uncertainty about the ethical performance of these rules exists. For patient with minor head injury and GCS score or 15, the CCHR and the NOC have equivalent high sensitivities for need for neurosurgical intervention and clinically important brain injury, but the CCHR has higher specimen for important clinical outcomes than does not the NOC, and its use may result in reduced maging rates Then adopted NOC decision rule appears valid for use in all patients with minor head inbjury whon are 16 years or older and have a GCS score of 13 to 15, irrespective of loss or consciousness. However, further research is needed to identify patients with neurocraman injury who do not require neuro surgical intervention but may benefit from emergency CT scanning and to determine the optimal trade off between sensitivity and specincityn not

REFERENCE

1. ***North Am 1991*** *Aug :15(3): 591-604 Medicine*
2. ***Chot DW****: tonic dependence of glutamate neurotoxicity. J. Neurosci 1987 Feb. 7(2) 309-79 (Medicine.)*
3. ***Rourkes M.A. Esenberg HM, Jane, JA, et al****. The Traumatic Coma Data Bank Design, methods and baseline characteristics. J. Neurosurg 1991:75:S8-S13.*
4. ***Gennareii IA, Thibault L.E. Adams JH****: Diffuse axonal injury and traumatic coma in the primate. Ann Neurol 1982 Dec.: 12(6): 564-74.*
5. ***Randay AL****: Pathophysiology In: Maridon DW, ed. Traumatic Brain Injury NY Tmeme Medical Publishers :1999-=29-38.*
6. ***Kraus Jr. Mc Arthur DL****: Epidemiologic aspects of brain injury. Neurol Clin 1997 May; 14(2):435-50.*
7. ***Kraus Jr. Black, MA, Hessol N, et al****: The incidence of acute brain injury and serious impairment in a defined population. Am J. Epidemiol 1'984 Feb. 119 (2) 180-201. Medicine.*
8. ***Masson P. Thicoipe M. Aye P****:Epidemiology of severe brain injuries a prospective population based studyJ. Trauma 2001 September:51(3)481-9.*
9. ***Ommaya AK, Grubb RL Jr. Naumann RA****: Coup and contre coup injury observations on the mechanics of visible brain injuries in the rhesus monkey. J. Neurosurg 1971 Nov. 35(5): 503-16.*
10. ***Regner A. Alves LB, Chemale I*** *Neurochemical characterization of traumatic brain injury in humans J. Neurotrauma 2001 Aug. 18(8) 783-921 Medicine.*
11. ***Silbernagl S. Lang F****: Neuromuscular and Sensory Systems. Colour Atlas of pathophysiology NY Thieme Medical Publishers : 2000-298-361.*
12. ***Thomhill S. Teasdale GM, Murray GD*** *Disability in young people and adults one year after head injury prospective cohort study BMJ 2000 Jun 17: 320 (7250) 1631-5. Medicine.*
13. ***Woo BH Nesathurai S****:Overview. The Rehabilitation of people with Traumatic Brain Injury Vol.1 Backwell Science 2000:5-12.*
14. ***Zink BJ*** *Traumatic brain injury Emerg Med. Clin North Am 1996 Feb. 14(1) 2115-50 Medicine*
15. ***NCC-AC.*** *TheRoyal College of surgeons of England Head InjuryTriage assessment, investigation and early management of head injury in infants., children and adults NCC-AC,2003.*
16. ***Kraus, J. Romer C. Holst H et.al****. Prevention critical care and rehabilitation of Neurotrauma Stockholm, WHO collaborating Centres for Neurotrauma, Karolinska Institute, 995.*
17. ***N. Jennet B.*** *Epid emiology of head injury J. Neurol Neurosurg Psychiatry 60:362-369, 1996.*
18. ***Kraus JF and Nourah P.*** *The epidemiology of mild uncomplicated brain injury J. of Trauma 1988:28 (2): 1637-1643.*
19. ***Stiell IG, Wells GA, Wandemheen K, et.al.*** *Ann Emerg Med. 30:14-22, 1997.*
20. ***Servadei F & Merry S*** *Mild Head Injury in Adults Youman's Neurological Surgery Saunders, 5th ed. 1996. Vol.4:5065-5080.*
21. ***Barclay L, Vega C Canadian CT*** *Head Rule May, Be Better Than New Orleans Criteria for CT of Minor Head Injury CME Medscape September,27, 2005.*
22. ***Servader P.*** *CI examination in minor head I jury Lancet 1'991:21:788.*
23. ***Naus Jr. Black MA, Hessol N, et.al****. The incidence of acute brain injury and serious impairment in a defined population. Am J. Epidemiology 1984:119(2) 188-201.*
24. ***Kraus JR. Peek C. Willams A.*** *Compliance wit the 1991 California Motorcycle rennet ose law, Am J. Public Health 1995: 85:96-99.*
25. ***Kraus Jr. Mc Arhur DL****:Epidemiologic aspects of brain ijury. Neurol Clin 1996 May, 14(2) 435-501 (Medicine)*
26. ***Dacey RG, Alves WM, et.al.*** *Neurosurgical complications after apparently minor head injury J. Neurosurgery 1986: 65:203-210.*
27. ***Risang JNK Yeung T. Yu, ALM, et.al****. The utility of head computed comography after niminal head I bjury J. Trauma 1999:46:268-270.*
28. ***Gomez PA, Lobato RD, Ortega JM, et.al****. Mild head injury: differences in prognosis among patients with GCS score of 13 to 15 and analysis of factors associated with abnormal CT findings Br. J. Neurosurgery 1996:10:453-460.*
29. ***Servader P. Merry G.S.*** *Mild Head injury in Adults. In Youmans Neurological surgery rem edition. Volume 4 Saunders WB 1966 pp 5065-5079.*
30. ***Stein SC, Ross SE****: The value of CT scans in patients with low risk head injuries neurosurgery 1990: 26:637-640.*
31. ***Jeret JS, Mandell M et.al.*** *Clinical predictors of abnormality disclosed by CT after mmd head trauma. Neurosurgery 1993:32:9-16.*
32. ***Sherman C et.al.*** *Miod Head Injury. A plea for routine early CT scanning. The Journal of Trauma 1992:33 (1):11-13.*
33. ***Nagy KK, Joseph KT, Krosner SM, et. al****: The utility of head computed tomography after minimal head injury J. Trauma 1999:46:268-270.*
34. ***Miller JD, Murray L.S. Teasdale GM,*** *Development of a Traumatic intracranial hematomas after a minor head injury. Neurosurgery 1990:669-673.*
35. ***Haydel MJ Jones, PA, Mills TJ, et.al.*** *Indications for CT in patients with minor head injury N. Eng. J.Med. 2000:343:100-105.*
36. ***Gomez PA, Lobato RD, Ortega JM, et.al.*** *Mild head injury, differences in prognosis among patients with GCS score of 13 to 15 and analysis of factors associated with abnormal CY findings Pr. J. Neurosurgery 1996 : 10:453-460.*
37. ***Nagurney JT, Boreuk P, Thomas SH*** *Elder patients with closed head trauma a comparison with on elder patients Acad Emergy Med 1998 :5:678:684.*
38. ***Shackford SR et.al.*** *Clinical utility of CT scanning and Neurological Examination in the management of patients with minor head injuries Jr. of Trauma 1992:33(3)385-393.*
39. ***Todd Hard, Morris D.*** *Kerstein Inadequately of Bed side Clinical indications in identifying significant intracranial injury in trauma patients 1993:32(3) 359-362.*
40. ***Mohanty SK Thompson W. Rakower S.*** *Arc CT scans for Head Injury patients always necessary? The journal of Trauma 1991:"31(6) 801-805.*
41. ***Klauber M.R, Marshall LF and Leiersen TG et. al*** *Determinants of Head InjurybMortality importance of low risk patients J of Neurosurgery 1989 24(31)*
42. ***Max W. Mac kenzie EJ, Rice DP*** *Head injuries cost and consequences. Journal of Herad Traun a Rehabilitation 1991 P: 6 : 76-91.*
43. ***Stiel IG, Clement CM Rowe BH, Schull MJ, et.al.*** *Comparison of the Canadian CT Head Rule and the New Orleans criteria in patients with minor head in jury Lancer 2001 May 5,357 (9266):1391-6.*
44. ***Ian G. Stiell, Catherine M. Clement, Brian H. Rowe, et.al.*** *Comparison of the Canadian CT Head Rule and the New Orleans criteria in patients with minor Head Injury JAMA 2005-294:1511-1518.*

In Hypertension Control



Olmat

Olmesartan medoxomil 20 / 40 mg tablets

Quick onset of action...
...Lesser clinical events

Once-a-day

Avas

5 mg
10 mg
20 mg

Atorvastatin tablets

Controls LDL-C...
Achieves NCEP Goals

For the use only of a Registered Medical Practitioner or a Hospital or a Laboratory

CARSYON

MICRO LABS LIMITED
27, Race Course Road,
Bangalore - 560 001

ROLE OF HELICOBACTER PYLORI IN BRONCHIAL ASTHMA : A CASE CONTROL STUDY

Nirmal Jain, G.N. Sexana, J.P. Rishi

Upgraded Department of Medicine, S.M.S. Medical College & Hospital, Jaipur, India

Abstract: 30 cases of asthma along with 20 unrelated controls were included in this study after strictly following inclusion and exclusion criteria. Both cases and controls were subjected to clinical examination and battery of investigations, including PFT, upper Gl endoscopy and specific tests for H. pylori. Cases with asthma have significantly high prevalence of H. pylori (73.3%) as compared to controls (60%). Sensitivity and specificity of rapid urease test for H. pylori is more than antral histopathology.(90% v/s 68%). After treatment of H. pylon cases had significant improvement in asthmatic symptomatology in the form of no exacerbation decreased need of medications, decreased night time exacerbation along with decreased in serum IgE levels and significant improvement in GIT symptoms.

INTRODUCTION

Bronchial asthma is a disease characterized by episodic airflow obstruction which is partially reversible; lung inflammation, particularly in the airways and bronchial (airways) hyperresponsiveness[1]. Etiopathogenesis of asthma is still poorly understood and treatment is mainly based on symptoms rather than underlying disease process, so there is always a need to look for new causes which may be associated and/or causing asthma.

As H.pylori is also associated with many extragastric conditions[2] like allergic diseases of skin e.g.urticaria, it may be possible that H.pylori may be linked directly to asthma as asthma is also an allergic disorder of respiratory tract.

We undertook this case control study to know the prevalence and correlation of H.pylori in patients of bronchial asthma.

MATERIAL AND METHODS

30 patients of bronchial asthma who fulfilled at least one of the following criteria[3] (a.) An increase in FEF1 of at least 15% after bronchodilator therapy. (b.) Spontaneous diurnal variability of peak expiratory flow rate (PEFR)/FEV1 of >20%. (c.) Clinical features consistent with diagnosis of asthma. `20 unrelated controls attending the allergy clinic, medical OPD and wards of SMS Medical College and Hospital, Jaipur (Raj.) were included in study and assessed by detailed history, clinical examination and relevant lab investigations.

Inclusion Criteria: 1.) All patients with bronchial asthma who fulfill at least one of the following criteria (a)An increase in FEV1 of at least 15% after bronchodilator therapy; (b) Spontaneous diurnal variability of peak expiratory flow rate; (c) Clinical features consistent with diagnosis of asthma. 2.) Patients willing to undergo upper GIT endoscopy. 3.) Patent between age of 15-65years 4.) Patients who were clinically stable at the time of entry into this study.

Exclusion Criteria (1) Patient not fulfilling diagnostic criteria of asthma. (2) Serious concomitant medical or surgical illness related to lung, liver, kidney or brain., (3) Immunocompromised patient. (4) Pregnancy. (5) Patient who had history of previous esophageal or pulmonary surgery (6.) Patient on antireflux medication.

Subject were studied as: Asthma and GIT characteristic of patients with Asthma GIT Questionnaire upper GIT endoscopy along with gastric antral biopsy for the evidence of H.pylori by means of rapid urease test (helikochek test) and histopathological examinations with special staining for H.pylori. Along with this, spirometry pre and post bronchodilator and other relevant investigations like s.lgE, TEC, CBC, DLC, X-ray chest PA view were done.

Those found positive for H.pylori by either rapid urease test and/ or histopathological examination were given 14 days of standard course of anti H.pylori treatment and later on followed for five month to see improvement in asthmatic and GIT symptomatology by means of history, examination and investigations. In those 14 days patients (only cases) were kept on rescue medications in the form of inhaled bronchodilators e.g. salbutamol on SOS (as and when required) basis.

RESULTS

Out of 30 cases, maximum 9(30%) were in 35-44 yrs. of age group. It shows maximum cases were in middle part of Gaussian distribution. 5(16.6%) cases were in each of 25-34,45-54,55-65 yrs. of age group. 6(20%) cases were in 15- 24 yrs. of age group. In control group they were distributed almost equally among all age groups. Mean age of cases was 38.03 yrs. and 35.86 yrs. of controls.

Out of 30 cases, 13(43.3%) had both, rapid urease test as well as antral biopsy histopathology, positive, while 8(26.6%) had both negative. 2(6.6%) had only antral biopsy histopathology positive and 7(23.3%) had only rapid urease test positive (Table 1).

Table 1.: *Combined rapid urease test and antral biopsy Histopathology results in cases*

Both Histopathology & Rapid Urease Test Positive		Both Histopathology & Rapid Urease Test Negative		Histopathology Positive But Rapid Urease Test Negative		Histopathology Negative But Rapid Urease Test Positive	
No.	%	No.	%	No.	%	No.	%
13	43.3	8	26.6	2	6.6	7	23.3

Out of 20 controls, 6(30%) had both, rapid urease test as well as antral biopsy histopathology, positive, while 8(40%) had both negative. 2(10%) had only antral biopsy histopathology positive and 4(20%) had only rapid urease test positive(Table 2).

Table2: *Combined rapid urease test and antral biopsy Histopathology Results in Controls*

Both Histopathology & Rapid Urease Test Positive		Both Histopathology & Rapid Urease Test Negative		Histopathology Positive But Rapid Urease Test Negative		Histopathology Negative But Rapid Urease Test Positive	
No.	%	No.	%	No.	%	No.	%
6	30	8	40	2	10	4	20

Correspondence: **Dr. Nirmal Jain,** *K-86, Badhwar park Railway colony Colaba, Mumbai*
e-mail: drnimal_1979@ yahoo.co.in

Table3: *Response to anti H. Pylori treatment in cases*

Asthma symptomatology				GIT symptomatology			
Improved			Not improved	Improved			Not improved
No Exacerbation	↓ Exacerbation	↓ Need of rescue medications		Epigastric pain	Heart burn	Post prandial fullness	
5 (25%)	11 (55%)	13 (65%)	4 (20%)	10/11 (91%)	7/7 (100%)	9/9 (100%)	1 (9.8%)

Out of 20 cases who came for follow up 5(25% had no exacerbation of asthma while 11(55%) had decreased exacerbation and 13(65%) had decreased need of rescue medications. Likewise in GIT symptoms epigastric pain relieved in 10(91%) while heart brun decreased in 7(100%) cases. There was no response in asthma symptomatology in 4(20%) cases and in GIT symptomatology in 1(9.9%) case.

Out of 20 controls, 12 were positive for H.pylori (rapid urease test/ histopathology/both).They were treated with 14 days of standard course of anti H.pylori treatment. On follow-up 11 controls had significant improvement in their GIT symptoms.

In study group: upper GI endoscopy examination in asthmatic cases, it showed that 15(50%) had normal study while 13(43.3%) had antral gastritis. GERD was present only in 1 patient in this group.

In control group; In this group 13(65%) had upper GI endoscopy normal while just 4(20%) had antral gastritis (Table 4).

Table4: *Upper GI endoscopy findings in cases and control*

FINDINGS	CASES		CONTROLS	
	Numbers	Percentage	Numbers	Percentage
Normal Study	15	50	13	65
Antral gastritis	13	43.3	4	20
Esophagitis/GERD	1	3.3	-	-
Peptic ulceration	1	3.3	1	5
Others Duodenitis & Hiatus hernia	-	-	2	10
Total	30	100	20	100

DISCUSSION

This study was done to find out the incidence of H. Pylon in patients with asthma and tried to delineate any causal relationship between them so that this may lead to new dimensions in diagnostic approach of asthma and may help in developing new therapeutic interventions that may enable us to prevent the development of disease and associated irreversible changes in airway functions.[4]

lgE levels in cases ranged from 75 lU/mi to 20001U/ml.(mean 655 lU/ mi) while controls ranged from 68 lU/mi to 20001U/ml (mean 386 lU/ mi) IgE is the hallmark of atopic diseases, such as asthma, allergic rhinitis and atopic dermatitis. —treatment of H. pylon, serum IgE level decreased significantly. (p value<O.05).

When combined rapid urease test and antral histopathology were assessed cases out of 30 cases, 22(73.3%) cases were positive by both or either modality, 15(68.18%) had positive histopathology; while 20(90%) had When both tests were combined, we had 6 (30%) controls positive for H. pylon by both modalities of tests, 8 (40%) were negative in both while 2 (10%) had positive histopathology but negative rapid urease test and 4 (20%) had negative histopathology but positive rapid urease test.

In our study, out of 30 cases total 22(73.3%) were positive for H. pylon while out of 20 controls, total 12(60%) were positive for H. pylon (p value< 0.05) similarly Karel'skaia et a16 also found H. pylon invasion in 89.2% patients with chronic allergic diseases (asthma, urticaria) but study of Nijevitch et a17 had only 52.4% H. pylon invasion detected histologically in children with chronic asthma. However Tsang et al8 did a study to know the seroprevalence of H. pylon in asthma. They found that H. pylon seroprevalence was not significantly different between asthmatic and control subjects. (p value>0.05)cases, were given 14 days of standard anti H. pylon treatment, meanwhile continuing rescue medications for asthma. After 14 days of treatment 20 patients turned up for follow up. Out of these 20, 9 patients refused to undergo endoscopy again due to some or other reason and as in our institution we have no facility of noninvasive tests like urea breath test, we have to rely on patients' history and symptomatology.On follow up, out of 20 cases, 5(25%) had almost complete relief from asthmatic symptoms. They had almost nil exacerbation and their post bronchodilator response was markedly improved. 11(55%) cases showed significant improvement in the form of decreased nocturnal episodes of shortness of breath. Their post bronchodilator response was improved and they had decreased requirement of bronchodilator therapy. Rest 4(20%) cases showed no improvement. So after giving 14 days of standard anti H. pylon treatment, 16(80%) had improvement in symptomatology of asthma irrespective of bronchodilator treatment (rescue therapy) which is clinically significant (p value<0.05) Out of 20, 11 had epigastric pain before treatment, which was relieved in 10 (90.1%) cases while heart burn and post prandial fullness was relieved in all the patients, clearly suggesting that H. pylon was responsible for these GIT symptoms. Cases who had improvement in GIT symptoms also had significant improvement in asthmatic symptoms.

CONCLUSION

Apart from an increased incidence of H. pylon in patients of bronchial asthma, further proof of role of H. pylon in bronchial asthma came from the fact that with the eradication of H. pylon, asthmatic symptomatology improved in t80%) of the patients. Thus it is believe that though it is a small study, our finding may have important implications in clinical practice and these results may form the basis of future screening program and preventive strategy in bronchial asthma.

REFERENCES

1. ***Pascual R.M. et al:*** *uAirway remodeling contribute to the progressive loss of lung function in asthma: An overview". Journal Allergy and Clinical Immunology 2005;1 16:477-486.*
2. ***Roussos A et al.*** *Helicobacter pylon infection and Respiratory diseases :a review. World J Gastroenterology 2003 ;9(1): 5-8.*
3. ***Field SK et al.*** *Prevalence of gastroesophageal reflux symptoms in asthma.CHEST1 996; 109,316-322.*
4. ***Timothy D et al.*** *Mapping susceptibility genes for asthma and allergy. Journal Allergy and Clinical Immunology 2000; 1 05:S477-81.*
5. ***Mutius EV. Influences in allergy*** *: Epidemiology and the environment. Journal Allergy and Clinical Immunology 2004; 113:373-79.*
6. ***Karel'skaia IA et al.*** *Helicobacter pylon infection in patients with chronic hives and asthma. KIm Med (Mosk)2005;83(3):58-61.*
7. ***Nijevitch AA et al.*** *Helicobacter pylon infection and reflux esophagitis in children with chronic asthma. Journal of Clinical Gastroenterology 2004;38(1): 14-18.*
8. ***Tsang KW et al.*** *Helicobacterpylori sero prevalence in asthma. Respiratory Medicine 2000;94(8):756-59.*

Next Issue Highlights ***SPECIAL ISSUE: Urolithiasis :Current Perspective***

Guest Editor:Prof. N.P.Singh

- ***Urogenital Anatomy-How it is prone for Urolithiasis?***
- ***Radilological Investigations in Urolithiasis***
- ***Urolithiasis in Children***
- ***Epidemiology of Renal stones***
- ***Clinical features of Urolithiasis***
- ***Workup of a patient with Urolithiasis***
- ***Lithopripsy for Urolithiasis***
- ***Surgical Management including PCNL***
- ***Aternate Remedies for Urolithiasis***

EVALUATION OF IMMUNOSUPPRESSIVE THERAPY IN CASES OF STEROID RESISTANT IDIOPATHIC GLOMERULONEPHRITIS

Manju Sharma, N. Nand*, Viral Sangwan*, S.Binani***

**Departments of Medicine & Nephrology,*

***Department of Biochemistry Pt.B.D. Sharma, PGIMS, Rohtak – 124001, Haryana, India*

Abstract: *The present prospective randomized study was carried out on 30 adult cases of steroid resistant idiopathic membranous (MGN) or membrano proliferative glomerulonephritis (MPGN) and the effects of immunosuppressive therapy were evaluated. Thirty (30) patients with progressive deterioration of renal functions were included which were divided in to two groups. After baseline investigations and pre-informed written consent, 20 cases of group A were given azathioprine (1-2 mg/kg / body weight) and prednisolone (0.5 mg/kg/body weight) for a minimum period of 6 months and the renal functions were evaluated at monthly intervals, Group B cases (10 patients) served as controls. After six months of immunosuppressive therapy, creatinine clearance increased in group A, where as it decreased slightly in group B and the differences were statistically significant (P<0.001). The proteinuria decreased significantly in group A patients (P<0.01) where as in Group B it increased (P>0.05). Cholesterol did not change significantly in the two groups. All the patients tolerated the drug well. In group A, 20% patients had complete remission and rest 80% had partial remission, where as in group B, 40% patients had stablilzation of renal functions, 40% had no change and 20% deteriorated. Hypertension and persistent proteinuria were associated with poor4 prognosis.*

Conclusions: It is therefore, concluded that immunosuppressive therapy induced remission in a significant number of patients. However, a large study is needed to confirm the long term survival in such patients.

INTRODUCTION

Chronic glomerulonephritis is leading cause of chronic renal failure. The primary mechanisms, involved in pathogenesis of idiopathic glomerulonephritis are largely antibody mediated. 1 However, there is considerable evidence that cell medicated immune reactions are also involved. High rates of remissions have been observed with steroid and cytotoxic drugs and combination of azathioprine and prednisolone has been reported to beneficial in idiopathic membranous nephropathy specially with deteriorating renal functions. 3-5 However, a few studies have not found the use of azathiprine to be beneficial. 6-8. Therefore, the treatment of steroid resistant idiopathic glomerulonephritis is not only frustrating but is also controversial. It was in this context that the present study was planned to evaluate the effects of this immunosuppressive therapy in case of steroid resistant idiopathic glomerulonephritis.

MATERIAL & METHODS

The study was carried out in 30 adult cases of steroid resistant idiopathic glomerulonephritis (SRIGN). Only cases with MGHN and MPGN with deteriorating renal functyions with glomerular filtration rate (GFR)< 80.0-ml/min and serum creatinine >2/5 mg% were included in this study. Patients with secondary gloerulonephritis were excluded. The diagnosis of SRIGN was based on histopathological examination and relevant investigations. The patients group consisted of two groups. Group A cases were given a combination of azathioprine (1-2 mg/kgbody weight) and prednisolone (0.5 mg/kg/body weight) for a minimum period of 6 months and the renal functions were evaluated at monthly intervals. Group B cases (10 patients) served as control and it included only those cases of glomerulonephritis who dropped out of the study or refused for their inclusion. Remission was defined as complete when renal functions returned to normal (serum creatinine decreased to < 1.5 mg%) and proteinuria decreased to non nephritic range. Partial remission was defined when there was an improvement in the renal function but proteinuia persisted <3.5 gm/ day. No response was indicated by non change in renal functions or an increase in the serum creatinine by less than 50% of the basal value. Deterioration of renal functions was indicated by a further rise in serum creatinine levels more than 50% of base line value.

RESULTS

The mean age of the patients was 33.4±13.4 years. There were 18 males and 12 females. In group A, there were 14 cases of MPGN and 6 of MGN where as in group B, eight cases were of MPGN and 2 cases of MG. The basal biochemical parameters including age, the degree of renal failure, proteinuria etc., were alike in the two groups (Table-1)

Table-1: *Showing Baseline Renal Profile in the two groups*

Renal Parameter	Group A	Group B	P value
Blood Urea (mg %)	128.5 ±34.7	132.5 ±19.4	>0.05
Serum Creatinine (mg%)	4.3± 0.9	3.97± 0.48	>0.05
GFR (ml/min)	19.9 ±8.9	30.05 ±2.39	>0.05
Proteinuria (g/day)	4.4± 1.6	2.47± 0.5	>0.05
Serum Chlestol (mg%)	280.5± 96.0	264.2± 74.2	>0.05
Serum Uric Acid (mg%)	6.1 ±1.5	5.7 ±1.2	>0.05
Serum Protein (gm%)	5.8± 1.4	5.9± 1.2	>0.05
Serum Calcium (mg%)	8.4± 0.5	8.2± 0.4	>0.05
Serum Phosphate (mg%)	4.9± 0.6	4.8± 0.5	>0.05

Table 2: *Renal Functions Following Immunosuppressive Therapy*

Parameter	Group A before therapy	Group B After therapy	P paired
Blood Urea (mg %)	128.5 ±34.7	64.0 ±20.1	<0.001
Serum Creatinine (mg%)	4.3± 0.9	1.8± 0.6	<0.01
GFR (ml/min)	19.9 ±8.9	52.0 ±22.5	<0.001
Proteinuria (g/day)	4.4± 1.6	2.5± 1.6	<0.01
Serum Chlestol (mg%)	280.5± 96.0	236.5± 59.5	<0.05
Serum Calcium (mg%)	8.4± 0.5	9.01± 0.62	>0.05

Following immunosuppressive therapy there was a significant rise in GFR and fall in serum creatinine, blood urea, serum cholesterol and proteinuria (Table-2) in group A patients as compared to group B. 20% patients achieved complete remission and rest 80% were in partial remission following six months therapy, where as in Group B 40% patients had stabilization of renal functions, 20% deteriorated and 40% had no change. Therefore, percentage of patients going in to remission (complete + partial) was significantly higher in Group A as compared to group B. Further an attempt was made to find out the factors associated with poor outcome. The patients who had complete remission had significantly lower systolic blood pressure, as well as base line biochemical parameters like blood urea, serum creatinine and cholesterol as compared to those

Correspondence: **Prof. Nitya Nand,** 3/7J Medical Enclave, Rohtak-124001 e-mail nitya_49@yahoo.com

Table-3: *Baselines Characteristics of patients who went into remission (complete / partial) after immunosuppressive therapy*

Parameter	Complete remission	Partial remission	P value
Age (years)	31.0±12.5	32.8±13.3	>0.05
Sex (Male: Female)	1:1	1:3:1	>0.05
Duration (months)	33.0±11.5	28.2±22.4	>0.05
Systolic Blood Pressure (m Hg)	125.0±12.9	148.0±15.5	<0.05
Anemia	None	9	-
Oliguria (< 400 ml / d)	1	8	<0.01
Blood Urea (mg %)	117 ±12.9	127.3 ±35.5	>0.05
Serum Creatinine (mg%)	3.8± 0.3	4.4± 1.1	>0.05
Serum Chlestol (mg%)	231.8± 59.3	298.3±107.1	>0.05
Serum Calcium (mg%)	8.2±1.05	8.1±0.73	>0.05
Proteinuria (g/day)	4.3± 2.1	4.5± 1.5	>0.05
GFR (ml/min)	19.4± 7.3	19.5± 10.0	>0.05
Kidney size (cm)	9.0± 0.41	8.8±0.36	>0.05

who did not respond (Table-3) Further early amelioration of proteinuria was associated with better prognosis and outcome as compared to those who had late decrease in the urinary excretion of protein. When the type glomerulonephritis was taken in to consideration, it was observed that 25% of MPGN cases had complete remission and 75% had partial remission, where as all the cases of MGN had partial remission and none of the cases had complete remission. However, no histopathological markers of bad prognosis could be identified. Fixed drug reaction, hematemais and activation of pulmonary tuberculosis were seen in one case each in Group a and none in Group B.

DISCUSSION

In MPGN, the lesion is due to glomerular localization of preformed circulating immune complexes, where as in MGN, the lesion is due to a reaction of circulating antibodies with intrinsic glomerular antigen. The appearance of population of large radius pores, leading to increase in fractional IgG clearance : local deposition and activation of complement mediated injury are the major parts of the cascade. Fibrosis and sclerosis leads to further progression of renal disease. Therefore steroid and cytotoxic drugs, which have anti inflammatory action, are though to be effective in halting the progression of renal disease.9 In addition, steroids also effects the renal hemodynamics leading to increase in GFR. The fall in proteinuria is though to be due to the direct action of corticosteroids on glomerular basement membrane. 10. An alternative hypotheis is that they have anti interleukin action and relieve proteinuria by interfering with the cytokine cascade.11

Immunosuppressive therapy produces clinical benefits by producing several alterations in the immune system of the body. These included the inhibition of helper, inducers and cytotoxic T lymphocytes. They also influence the cell released mediators of inflammation and fibrogenesis 12. Azathioprine, is chiefly use in patients who are resistant to steroid and have frequent elapses following withdrawal of steroids. The action of this drug is at different level than steroid therefore, addition of azathioprine result in optimization of therapeutic response with minimum iatrogenic mortality and morbidity. There are a few studies available where the combination of azathioprine and prednisolone have been found to be beneficial in SRIGN, especially in MGN. 3-5,13. However, there are conflicting reports as well where the combination was not found to be beneficial in SRIGN,3,7. Therefore the literature on the subject is not only scant but is also controversial hence this study. 5 In the present study all patients tolerated the drug well. In group A, 20% patients had complete remission and rest 80% had partial remission, where as in Group B, 40% patients had stabilization of renal functions, 40% had no change and 20% deteriorated. Hypertension and persistent proteinuria were associated with poor prognosis. Two of the previous studies, 6-7 which did not find any beneficial effects were not well planned and were retrospective; hence their results can not be compared. The number of the patients in Barker study 5 were only 4 where as Brown et al4 reported similar observation as reported by us. On the other hand the recent 10 year follow up study 8 reported no beneficial effects.

However, in this study also they did not analyze the bad prognostic factors which might be operating in their patients. A number of factors like advancing Age 14, long duration of disease 17, presence of systolic or diastolic hypertension 16, heavy proteinuria 17, higher basal value of serum creatinine and urea 18, late amelioration of proteinuria and presence of certain histopathological markers like crescents and tubulointerstitial inflammation etc. are known to be associated with poor outcome, 19-20 Hypertension accelerates glomerular injury through hemodynamic mechanism, greater degree of proteinuria, extra capillaryproliferation and glomerulosclerosis. Further its presence may denote fluid over load or actually advanced renal disease. In addition, hypercoagulability and hyperlipidemia may also independently lead to progression of renal disease. Hyperlipidemia through complex mechanisms of macrophage infilitration and interstitial cell proliferation, contributes to the progression of glomeruloscelerosis In the present study, only bad prognostic factor was the presence of significantly elevated systolic blood pressure in the non responders. Although a number of histopathological markers have been described to be associated with poor out come. However, except for the type of glomerulpathy, no pathological marker could be identified in the present study. The presence of crescents and a large number of crescents and a large number of non functioning (sclerosed) glomeruli, may in fact denote a serve renal pathology rather than a prognostic market. Although the number of patients included in the present study were small and the duration of the study and follow up were 6 months and a year. However, the result of this study no suggest that it is essential that a good work up of the individual case is of utmost importance to predict the long term survival in these cases and every case of SRIGN with deterioriating renal functions deserves immunosuppressive therapy.

REFERENCES

1. ***Camussi G. Kitaaazawa K, Andres G.*** *Mechanisms of tissue injury induced by immune Complexes formed in circulation or in situ. Diagn Immunol 1985:3:109.*
2. ***Couser WG,*** *Mechanisms of glomerular injury in immune complex disease. Kidney Int. 1985: 28: 569-3.*
3. ***William PS, Bone JM*** *Immuno supression can arrest progressive renal failure due to idiopathic membranous glomerulonephiritis. Nephrol Dial Transplant 1989:4:181-6.*
4. ***Brown JH, Douglas AF, Murphy BG Hill CM, McNamere PT.*** *Treatment of renal failure in idiopathic membranous nephropathy with azathioprine and prednisolone. Nephrolo Dial transplant 1998:13:443-8.*
5. ***Backer LR, Tucker B, Macdougall IC, Oomen R.*** *Treatment of progressive renal failure and nephritic syndrome with azathioprine and prednisolone. Postgrad Med. J. 1997:73:647-8.*
6. ***Muirhead N.*** *Management of IMN:Evidence based recommendations. Kideny Int. 1999:70 (Suppl.) 847-55.*
7. ***Ahuja M. Goumenous D, Shortland JR, Gerakis A.*** *Treatment of IMN Am J. Kideny Dis. 1999:3:521-9.*
8. ***Goumenos DS, Ahuja M, Davlouros P et.al.*** *Prednisolone and azathioprine in membranous nephropathy; a 10 year follow up. Study. Clin. Nephrolo 2006: 655(5) 317-23.*
9. ***Ponticelli C.*** *Progrnosis and treatment of membranous nnephropathy (Nephroloogy Forum). Kideny Int. 1986:29:927-40.*
10. ***Travis RH, Sayers G. Adrenocorticotropic hormone, adrenocorticosteroids and their synthetic analogs. In: Goodman LS, Gillman A. Editors.*** *The pharmacological basis of therapeutics. 3rd New York The Mach Millan Company 1965:p.1628-9.*
11. ***Misra RP, Berman LB*** *Studies on glomerular basement membrane. Effects of steroid on membrane chemistry and its protein permeability. Lab Invest 1972:26:666-70.*
12. ***Kincaid S.P.*** *Treatment of chronic MPGN with impaired renal function Med. J. Aust 1972:2:589-92.*
13. ***Willaims PS, Bone JM, Rustom R.*** *Progressive versus indolent idiopathic membranous nephropathy, Quart J. Med 1997:211:1854-55.*
14. ***William H.*** *Idiopathic focal segmental glomeruloscloris. Seminars in Nephrology, 23, 2003:183-193.*
15. ***Iitaka K. Ishidate T.*** *Idiopathic membranous glomerulonephiritis in Japanese children . Ped. Nephrol 9:1995:272-77.*
16. ***Chadban SJ, Atkins RC.*** *Glomerulonephritis. Lancet 365:2005:1797-806.*
17. ***Isbel N.M.*** *Glomerulonephritis Management in General practice Australian Family Phy. 34; 2005:907-13.*
18. ***Alexopoulos E, Stangou M.*** *Factors influencing the course and the response to Treatment in primary focal segmental glomerulosclerosis. Nephrol Dial Transplant 15:2000: 1348-56.*
19. ***Little MA Dupont P.*** *Severity of primary MPGN rathr tha M PGN type determines renal survival and post transplant recurrence risk. Kidney Int. 69(3. 2006:504-11.*
20. ***Schmitt H, Bohle A Reineke T. Meyer Eichberger D. Voge W.*** *Longter prognosis of membranoproliferative glomerulonephrisits type 1 Nephron 1990:55:242-50.*

Future Special Issuses/ Symposia

Special Issues :
- Urolithiasn: Current Perspective
- Emerging Infections: Indian Perspective
- Challenges of Diabetes in the Developing World
- Organ Transplantation: Current Scenario
- Critical Care: What is relevant to our needs
- Current trends of Ophthalmology

Symposia :
- Menorhagia: Management Strategies
- Advances in Orthopedic Surgery
- Advances in Endourology
- Advances in Gynaecology

CASE REPORT

AMBIGUOUS EXTERNAL GENITALIA OF AN ADOLESCENT REARED AS FEMALE

Ranabir Pal*, Ankur Barua*, Pradip K Mohanata, Amrita Gosh*****

Departments of Community Medicine, Surgery, ***Pharmacology*

Sikkim Manipal Institute of Medical Sciences 5th mile, Tadong, Gangtok, Sikkim-737102. India

Abstract: *A 12-year-old adolescent, with ectomorphic innocent feminine appearance, presented with ambiguous genitalia with associated congenital absence of right thumb, scoliosis, severe anaemia, primary amenorrhea. She had normally developed breast without any other secondary sexual charactenshcs. She complained of recurrent episodes of abdominal pain and burning micturition with fever for the last 3 years. There was no vaginal orifice. In the genital area there was a pair of ill developed labia majora fused at upper end with a whirling pattern containing inside the urethral opening. The lower end of genitalia had loose skin folds containing adequate amount of rugae with a raphe mimicking the texture of scrotal skin. Ultrasound imaging studies confirmed that she had structures like ovary and uterus with hematometra. Renal scan confirmed agenesis of the left kidney and hypo-plastic right kidney with parenchvmal insufficiency.*

INTRODUCTION

Ambiguous genitalia is a congenital physical abnormality where the outer genitals do not have the typical appearance of either sex; it is difficult to classify the infant (by physical examination) as male or female. So, inter sex disorders associated with phenotypic males and females may be diagnosed much later. In very rare instances, the physical appearance may be fully developed as the opposite of the genetic sex (e.g., a genetic male may have normal female appearance).[1]

The Mayer-Rokitansky-Kuster-Hauser Syndrome refers to a group of associated findings that include unilateral renal agenesis or ectopia, ipsilateral mullerian defects and vaginal agenesis. Approximately 15% have contralateral vesicourethral reflux. Here, the müllerian agenesis second in frequency only to gonadal dysgenesis as a cause of primary amenorrhea. It can be caused by mutation in the genes encoding anti-müllerian hormone (AMH) or its receptor (AMHR). Women with this syndrome have a 46XX karyotype, secondary sexual characteristics of female and normal ovarian function, including cyclic ovulation, but have absence or hypoplasia of the vagina. The uterus usually consists of only rudimentary bicomuate cords, but if uterus contains endometrium, cyclic abdominal pain and accumulation of blood may occur. One third of men with this syndrome have abnormalities of urogenital tract and one tenth with skeletal anomalies, usually involving the spine.

CASE HISTORY

The patient Nazma Akhter, 12 years old adolescent was brought to kolkata for consultation from a remote village of Chittagong in Bangladesh during January 2006 with the chief complaints of recurrent and sever abdominal pain since last four years, few episodes of buming pain during passing urine since last three years, occasional bleeding from single opening in the genital area as her genitalia was not of quite normal shape and size. There were no pubertal changes. She has no feminine looks with axillary and pubic hair along with smaller breasts in comparison to her other daughters. There was no history of menarche or regular periods except occasional trickling of blood was present along with severe pain in loin that made her bedridden and she had recurrent bouts of low grade fever.

Birth history: Her mother recalled that she had no illness or had not taken any form of medication during this pregnancy which ended with a full-term delivery. The birth history was uneventful as home delivery with no proper ante-natal, intranatal and post-natal cares. This child was not given any vaccine till date and no growth monitoring was done.

Family history: The patient was the third child among six siblings- 2 older and 2 younger sisters and 1 younger brother. There was no history of similar complaints among other family members. In the paternal and maternal tree also there was no similar problem. The mother had reported fetal wastage once but there was no neonatal death in family. There was no history of maternal virilisation, suggesting an androgen—producing maternal tumor. Parents of this patient have never practiced any form of contraceptive methods till date.

On clinical examination the girl appeared scared, anxious, severely pale, and thin built, of medium stature with ectomorphic features, in general with an infantile feminine look. Her weight was 26 Kgs with height of 127 cms, There was absence of right thumb, cyanosis, jaundice, oedema, clubbing. There was scoliosis to left in dorsal region and to the right in lumber region. The abdomen was scaphoid shape with definite tenderness in the hypogastric region. Though the patient gave a history of reeling acute pain in the loin and lower abdomen, but there was no such tenderness or severe pain or mass or organomegaly on superficial and deep palpation in the abdomen. There was no abnormal finding in systemic examination. Rectal examination did not reveal the cervix and uterus to confirm the internal mullerian structures. The anal aperture was apparently normal in appearance.

Secondary sexual character examination: No age specific secondary sexual feature was present viz, no adolescent change in voice, adolescent increase in width of girth of pelvis and buttock, no axillary or pubic hair (Tanner stage 0). There was well developed breast with areola (tanner stage 3).

External genitalia examination: A pair of ill developed labia major like structure was visible. The size and degree of differentiation of the phallus was noted, since variations may represent with an irregular clitoromegaly or hypospadias. A protruding mass similar to hypertrophied clitoris of 1 cm long was overhanging the external genitalia. The external genitalia of this age without any pubic hair and infantile pelvic girth were unusual for this age in an adolescent (Figure-1).

On lithotomy posture, a single aperture was found clinically in the region between mons pubis and anal opening which was clinically noted as external urethral meatus. The latter was almost wholly covered by a structure similar to be called labio-scrotal folds. This could be separated

Correspondence: **Dr. Ankur Barua**, Assistant Professor, Department of Community Medicine, Sikkim – Manipal Institute of Medical Sciences (SMIMS), 5th Mile Tadong, Gangtok – 737 102 India Fax.: 03592-231496, e-mail : ankurbarua26@yahoo.com



Fig. *A pair of ill developed labia majora like structure seen with a protruding mass similar to hypertrophied clitoris with urethral opening.*

apart to note the lone aperture seated deeply. The fold of skin contained adequate amounts of rugae with a raphe. This clinically was similar to texture of scrotal skin has given an appearance of a scrotum without any solid structure like testes. This was unlike fatty smooth feelings of labia majors or minor. Rugose scrotal or labioscrotal folds with increased pigmentation suggested the possibility of increased corticotropin levels as part of adrenogenital syndrome. This fold of skin was fused at upper end with a whirling pattern apparently resembling clitoris. On pressing the meatus, a few drops of blood oozed out with dribbling of urine.

Investigations: Routine hematological studies revealed that the hemoglobin level of the patient was 4.2 g m/dl, MCHC 14.4 percent. Her serum creatinine level was O.7mmol/L.

Imaging Studies: Congenital absence of right thumb and scoliosis to the left in dorsal region and to the right in lumber region was confirmed through radiography. A Renogram with ^{99}mTc-DTPA, dose 4mCi was done and the report revealed; poor uptake in the right kidney and nil uptake in the left kidney.

Renal scan report confirmed that this patient had an agenesis of left kidney along with a hypoplastic right kidney, consistent with insufficiency ' of renal parenchyma.

Abdominal ultrasound was performed and structures resembling uterus and ovaries were noted. Uterus was of normal size with evidence of collection inside (haematometra). Genitography, CT scanning, MR and diagnostic laparoscopy, exploratory laparotomy, gonadal biopsy were advised, but due to financial constraints did not give consent.

Follow-up: This patient was treated for endstage renal failure dialysis was initiated, but could not be continued because of financial constraints; renal transplant was not possible due to lack of donor and financial constraints, the patient expired during the 8th month of follow up.

DISCUSSION

Intersex conditions are among the most fascinating conditions encountered by the clinician. In most cases today, clinicians can promptly make an accurate diagnosis and counsel parents on therapeutic options. However, the paradigm of early gender assignment has been challenged by the results of clinical and basic science research, which show that gender identity development begins in utero. While the techniques of surgical genital reconstruction have been mastered, the understanding of the psychological and social implications of gender assignment is poor.[23,4]

Intersexual individuals are treated in different ways by different cultures. In some cultures intersexuals were induded in larger third gender or gender-blending social roles along with other individuals. In most societies, intersex individuals have been expected to select one sex, and adhere to its gender role. Since the rise of modem medical science in Western societies, intersexuals with ambiguous external genitalia have their genitalia surgically modified to resemble either male or female genitals. But there are increasing calls for some critics had condemned the practice of performing corrective surgery on the genitals of intersexuals as an act of female genital mutilation. Corrective surgery is done purely for aesthetic or social purposes. It may lead to negative consequences for sexual functioning in later life, but many intersex individuals had resented the surgical intervention. Some so had opted for sexual reassignment surgery later in life. [14,5]

Medico-legal-Pitfalls: Treatment for intersex states is controversial; the controversy revolves around issues of gender reassignment. Though some intersex activists and health care professionals have called for a moratorium on gender reassignment and genital surgery until studies have been completed on the long-term effects of such surgery, but many had opposed the proposed moratorium.

Infants born with ambiguous genitalia present a true medical and social emergency. Salt-wasting nephropathy occurs in many of the infants born with ambiguous genitalia. If unrecognized, the resulting hypotension can cause vascular collapse and death. Male infants with this syndrome may be phenotypic ally normal and hence, the diagnosis might be missed. Modem treatment of infants with ambiguous genitalia involves a team-oriented approach. The goal is to provide appropriate medical support and counseling regarding care and therapy.[4,5] Evaluation of ambiguous genitalia requires a multidisciplinary.

Hence, from the medico-legal standpoint, the best approach to managing these cases of ambiguous genitalia is to provide parents with adequate information and guide them to make rational decisions.

REFERENCES

1) ***Behrman RE., Kliegman.R.M., Jensen.H.B., Eds***, *Nelson Textbook of Pediatrics, 17th ed,2004,Saunders-An imprint of Elsevier; 582:1945.*
2) ***Behrman R.E., Kliegman.R.M., Jensen.H.B., Eds, Nelson*** *Textbook of Pediat,ics,17 ed,2004,Saunders-An imprint of Elsevier; 529:1783-1785.*
3) ***Can- BR., Bradshaw K.D., in KasperD.L. Ed*** *Harrison's Prnciple of Internal Medidne.l6th ed.2005, McGraw Hill, Medical Publishing Division; 326:2204..*
4) ***Ghai O.P., Gupta P., Paul V.K. Ed.*** *Delopment and diffetentiation of Gonads, Ghai Essential Pediatrics,6th ed, revised & enlarged,2004:17.14:488*
5) ***Gartlen A.,*** *Gynecological Diseases in Campbell A.G.M., Mcintosh N., Eds. Foifar and Ameil's Textbook of Pediatrics, 5 ed, Churthill LMngstone, 1998; 18; 966.*

LITERATURE REVIEW

Recovery of renal function after 90 d on dialysis: implications for transplantation in patients with potentially reversible causes of renal failure

Samira Siddiqui, Michael Norbury, Sue Robortson, Alison Almond and Chris Ises. Clinical Transplant 2008,2, 133-137

Late recovery of renal function in patients requiring dialysis is a well recognized but uncommon phenomenon. Moves to increase the number of live donor transplants and the recognition that early transplantation is associated with better graft survival means it is possible that patients who are going to recover renal function may be transplanted unnecessarily. Prospective survey of patients receiving dialysis for more than 90 d in south west Scotland from 1 January 1994 to 31 December 2005. Routine measurement of residual renal function by combined urea and creatinine clearance allowed us to detect late recovery whenever this occurred. Eight of 202 (4%) patients recovered sufficient renal function to stop dialysing after 90-d treatment. The likely cause of the renal failure in five of these patients was atheroembolism. One with atherosclerotic renovascular disease had been stented and would have received a live related renal transplant had his sister not had second thoughts about the procedure. It may be sensible to postpone transplantation in patients with certain types of renal failure, perhaps particularly patients with renovascular disease who have recently undergone a failed revascularization procedure.

BACLOFEN IN THE MANAGEMENT OF INTRACTABLE HICCUPS –A CASE REPORT

N.S. Neki

Department of Medicine, Govt. Medical College/Guru Nanak Dev Hospital, Amritsar (Punjab), India

Abstract: *Many drugs and remedies are currently available for the treatment of hiccups; baclofen is good drug in relieving the intractable hiccups, where all other known durgs fail to provide relief. Hiccup is a sudden, involuntary contraction of inspiratory muscles (predominantly diaphragm) terminated by abrupt closure of glottis thereby stopping inflow of air and producing a typical sound. The basic defect seems to be stimulation of hiccup reflex arc vagus nerve, phrenic nerve and a hiccup centre in the upper spinal cord. Hiccup attacks usually last for few seconds or minutes and occur at brief interval. However intractable hiccup may be prolonged for months or years also. A case of intractable hiccup treated with baclofen producing complete relief is reported here.*

INTRODUCTION

Hiccups are particularly troublesome and frustrating both for the patient and the treating physician. They result from an involuntary clonic spasm of the respiratory musculature (chiefly of the diaphragm but also the intercostal muscles) with sudden interruption or a sequel to anaesthesia, diaphragmatic ileus or paradiaphragmatic irritation, paralytic ileus, alcoholism, cerebrovascular accidents, encephalitis, brain tumour, psychogenic, metablic causes including renal failure, hepatic failure, diabetic ketoacidosis, respiratory filure and electrolyte disturbances. Other causes include phrenic nerve irritation caused by pericardities, mediastinitis, compression by tumour and surgery of thorax or upper abdomen.

CASE REPORT

A 28 years educated male patient presented with hiccup of 3 weeks duration. He was having these troublesome episodes since 2 years. The attacks occurred at an interval of 3-4 months and each attack lasted for 1-2 months, thereby interfering wih his daily routine activities. He was nonalcoholic with no family history of hiccups. On questioning he admitted having taken many drugs like domperidon, metoclopramide, chlorpromazine, amitryptiline, diazepam, haloperidol and cisapride etc. with no relief. Laboratory profile revealed Hb 10,8gm dl, TLC and DLc within normal limits, FBS 95mg/dl. The profile of liver, kidney and serum electrolytes was normal, other investigations like x-ray chest, ECG, echocardiography, ultrasound abdomen, upper GI endoscopy, barium swallow and meal CT scan of head and abdoen all were essentially normal. He was started on tablet baclofen in doses of 5 to 10 mg twice daily for two days. His hiccup disappeared on 3rd day and the drug was continued for four weeks in tapering doses. He was followed up for 6 months with no adverse effects especially drowsiness.

DISCUSSION

Various remedies used for treatment of idiopathic hiccups include breathing into a closed paper bag, holding breath and increasing pressure on diaphragm, swallowing dry granulated sugar or ginegar, pharyngeal irritation, sips of iced water, stomach washes, tongue traction and lifting uvula etc. other drugs used with variable success include chlorpromazine, haloperidol, metochlopropamide, antacids, quinidine phenytoin, valproic acid, amitryptiline and nifedipine etc. But baclofen has been shown to be a novel drug for the management of intractable hiccups[1.2.3]

Baclofen is an analogue of the naturally occurring neurotransmitter gama aminobutyric acid (GABA), but unlike GABA it can cross the blood brain barrier. It is a potent natural depressant acting at bicuculline insensitive GABA receptors of GABAb receptors, which are widely distributed in the brain, Unlike other GABA receptors, they are metabotropic and are coupled to heterotrimeric serpentine which increase conductance in k+ efflux and increase in Ca2+ efflux producing an inhibitory post synaptic potential resulting in hyperpolarization of neurons, GABAb receptors inhibit the release of glutamate and aspirate. Baclofen is well absorbed after oral ingestion and has to be given twice or thrice daily. It can also be given directly into the space around the spinal cord(intrathecally) in order to minimize the adverse effect especially sedation. The pharmacological effects are mainly spinal in the daily therapeutic doses of 20-80 mg and higher doses are needed to hae effects on higher centres of brain 85% of the drug is excreted unchanged in the urine and needs dose modification in patients of end stage renal disease (ESRD) inorder to prevent toxicity[4.5] Uncommon adverse effects include nausea, vomting,diarrhea, headache, abdominal and chest pain, fatigue, pruritis, skin rashes, syncope, headache, palpiation dryness of mountiand taste disturbances. Clinical manifestations of overdose include absence of reflexes, vomiting, hypotonia, marked salivation, drowsiness visual disturbances, seizures respiratory drepression and coma[6]. Apart from ESRD other conditions predisposing to the development of toxicity with relatively low doses of baclofen include, elderly pregnancy, lactation, epilepsy, psychosis, respiratory and hepatic impairment.The toxicity is treated with atropine[7] baclofen should be tapered gradually to avoid withdrawal syndrome manifesting as hallucinations, seizures, pshychosis or even status epileptius[8] particularly if high doses are used.

CONCLUSION

Baclofen decreases the number and severity of hiccup spasms and relieves associated pain, cramps and muscle rigidity as observed in the present case, so it is a very good drug for the treatment of intractable hiccups before attempting surgical therapy.

REFERENCES

1. ***Yaqboob M, Prabhu P, Ahmad R.*** *Baclofen for intractable hiccups. lancet 1989; 2: 562-63.*
2. ***Patial RK.*** *Baclofen in the treatent of intractable hiccups Journal of Association of Physicians of India 2002; 50:1312-13*
3. ***Lewis j Hiccups.*** *In: griffith's 5 minute clinical consult. Dambro MD, Editor, 3rd edition, Baltimore: willliams and Wilkans1995;486-7.*
4. ***Chen KS, Bullard MJ , Chenn YY, Lee S.*** *Baclofen toxiticity in patient with severely impaired renal function. Ann Pharmacother 1997; 31: 1315-20*
5. ***Aisen Ml, Diet ZM, NcDowell F, Kutt H.*** *Baclofen toxiticity in patients with severely impaird renal function insufficiency. Arch Phys Med Rehabil 1994; 75: 109-11*
6. ***Physician Drug Hand Book,*** *8th Ed. Jan Fawett, Md Rush (Eds)-Prebyterian St. Luke's Medical Centre 100-1.*
7. ***Ferner RE.*** *Atropine treatment for Baclofen Overdose. Postgrad Med J 1981; 57: 580-81.*
8. ***Terrence CF, Fromm GH.*** *Complications of baclofen withrdrawl. Arch Neurol 1981; 38: 588-89.*

Correspondence: **Professor N.S. Neki,** *Department of Medicine, Govt. Medical College/Guru Nanak Dev Hospital, Amritsar (Punjab) India. e-mail : drnsneki_123@yahoo.com*

La Renon Introduce...

Laretol Injection
(Calcitriol Injection 1mcg/ml)

For effective management
of "Secondary Hyperparathyroidism"



Eido-Fe
(Vitamins Supplement Tablets with Elemental Iron)

The Power of 4

1. Vitamins Supplement
2. Folic Acid
3. Methylcobalamin
4. Oral Elemental Iron



Our Product Range...

Lacarnit Tablet	Levocarnitine Tablet 330 mg
Lacarnit Injection	Levocarnitine Inj. 200 mg / ml (5 ml Amp)
Lamino	Essential Amino Acids Tablet
Kchek	Calcium Polystyrene Sulphonate Powder 15 gm sachet
Laretol	Calcitriol SGC 0.25 mcg
Laretol Injection	Calcitriol Injection 1 mcg/ml
Lanum	Calcium Acetate Tablet 667 mg
Eido	Vitamins Supplement Tablet
Eido-Fe	Vitamins Supplement Tablet with Elemental Iron

La Renon Healthcare
C-21, Marutinandan Villa-2, B/h. Arohi Bungalows, Before Sun City, Bopal,
Ahmedabad-380 058, Gujarat, India.
Telefax : +91-2717-243352, E-mail : info@larenon.com www.larenon.com

CASE REPORT

COMPUTED TOMOGRAPHY IN THE DIAGNOSIS OF CHRONIC GASTRIC VOLVULUS

Rajul Rastogi, Satish Kumar Bhargava, Shuchi Bhatt, Sumeet Bhargava

Department of Radiology And Imaging,University College of Medical Sciences & Guru Teg Bahadur Hospital, Dilshad Garden, Delhi-110095, India

Abstract: *Gastric volvulus is a rare entity, which when acute can be potentially life threatening. On the other hand, chronic volvulus may be detected incidentally or may result in intermittent epigastric pain and abdominal fullness following meals. Organo axial variety is twice as common than mesenteroaxial variety. We report a case of an uncommon mesenteroaxial volvulus of the stomach that was incidentally detected on computed tomography of the abdomen and review the pathogenesis, classification and diagnosis of this rare entity.*

INTRODUCTION

Gastric volvulus is defined as an abnormal rotation of the stomach of more than 180°, creating a closed loop obstruction.[1] Stomach may rotate as much as 180° without becoming obstructed; more than 180° usually results in gastric obstruction. Emergent surgical intervention is indicated for acute gastric volvulus while for chronic gastric volvulus, surgery is performed mainly to relieve the symptoms and to prevent further complications.

CASE REPORT

A 35 years old male presented with a nonspecific complaint of episodic abdominal bloating since many years, which is mainly postprandial. Mild epigastric pain was also associated. The distension and pain was unresponsive to treatment. The patient had taken treatment for the tuberculosis of the chest 2 years back. Clinical examination did not yield any significant abnormality. Laboratory tests were unremarkable. Plain radiograph of abdomen revealed raised left dome of diaphragm with a large air fluid level underneath it. Computed tomography (CT) of abdomen was then performed to detect any occult disease of the gastrointestinal or hepatobiliary system and to determine the cause of the elevated left dome of diaphragm (fig 1-4). It revealed an over distended stomach in an upside down position. The gastroesophageal junction (GEJ) was normally positioned. The gastroduodenal junction was positioned abnormally higher than the GEJ and was to the left of spine. The left dome of the diaphragm was elevated and the left crux was thinner compared to the right crux. There was no associated diaphragmatic defect. Spleen and other viscera on the left side of the abdomen were abnormally high placed due to elevated left dome. Based on the above findings the radiological diagnosis of the chronic mesenteroaxial gastric volvulus with eventration of the left dome of diaphragm was made. The diagnosis was confirmed at surgery, which was performed to relieve the symptoms and to prevent further complications after which the patient remained asymptomatic.

DISCUSSION

The stomach is anchored at the cardia and tethered by the gastrocolic, gastrohepatic, gastrophrenic and gastrosplenic ligaments. Dalggard[2] demonstrated that it is easier to rotate a fluid-filled stomach than an empty one, and that a normal stomach cannot rotate more than 180º



Figure 1: Plain radiograph of the abdomen shows raised left dome of diaphragm with a large air fluid level underneath it



Figure 2: Axial contrast enhanced CT image shows superiorly displaced viscera on left side due to raised left dome

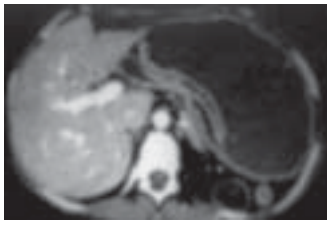


Figure 3: Axial contrast enhanced CT image shows over distended stomach filled with water and food material with gastroduodenal junction crossing the midline anterior to the spine



Figure 4: Coronal MPR CT image shows the upside down appearance of the stomach, elevated left dome of diaphragm, thin left crux and raised viscera on left side

Correspondence: **Prof. Satish Kumar Bhargava**, 201, Fancy Apartments, 19, Vasundhara Enclave, Delhi
Tel: 011-22629011, 9818312946, Email : skbhargava9731@rediffmail.com

unless the gastrosplenic or gastrocolic ligaments are divided.

According to etiology, gastric volvulus is classified as either type 1 (idiopathic – two-third) or type 2 (congenital or acquired – one-third). Type 1 is due to the laxity of the gastric ligaments and is seen in elderly when any cause of gastric distention can predispose to excessive rotation, setting the stage for volvulus.[3] A major cause for type 2 is a diaphragmatic defect that allows stomach displacement in to atypical spaces. In adults, the commonest defect is a paraesophageal hernia.[4] Other predisposing factors include diaphragmatic injury or surgery, congenital hernias, and diaphragmatic eventration associated with phrenic nerve paralysis, left lung resection, or pleural adhesions. Intrinsic lesions (e.g., gastric ulcer or neoplasm) or extrinsic pressure from adjacent masses can alter normal anatomy, and may predispose to volvulus.

According to Singleton,[5] gastric volvulus is classified based on the axis around which the stomach rotates,:

- ***Organoaxial***

➢ The stomach rotates around an axis that connects the GEJ and the pylorus with the greater curvature rotating (most often) anteriorly.[6]. The antrum rotates in opposite direction to the fundus of the stomach.

➢ It is the commonest variant, seen in approximately 59% of cases.[7] and is usually associated with diaphragmatic defects.

- ***Mesenteroaxial***

➢ The stomach rotates around the transgastric axis i.e. a line connecting the middle of the lesser and the greater curvature. The antrum rotates anteriorly & superiorly so that posterior surface of the stomach lies anteriorly and the greater curve lies most superiorly.

➢ It comprises 29% of cases.[7] Gastrosplenic ligament laxity is the predisposing factor, and this form often occurs without concomitant diaphragmatic defects.[4] Complete obstruction and strangulation are unlikely and spontaneous detorsions with recurrent acute episodes may occur. Most cases of chronic volvulus are related to this type.

- ***Combined / Mixed / Unclassified***

➢ This rare form exhibits features of both mesenteroaxial and organoaxial volvulus or remains unclassified

➢ It comprises the remaining 12% of volvuli

Ten to 20% of gastric volvulus occur in children, usually before one year, and are often secondary to congenital diaphragmatic defects. The condition is uncommon in adults < 50 years. Males and females are equally affected.

Gastric volvulus manifests as an acute abdominal emergency or as a chronic intermittent problem. The presenting symptoms depend on the degree of twisting and the rapidity of onset. Chronic volvulus, which accounts for 2/3 of adult cases, may be asymptomatic, may be reported as an incidental finding on radiography, barium studies or computed tomography[8], or may present with vague, spontaneously resolving epigastric pain, abdominal fullness following meals, early satiety, dyspnea, and sometimes chest discomfort. Dysphagia occurs if the GEJ is distorted. Because of the nonspecific nature of the symptoms, patients are often investigated for other entities as cholelithiasis and peptic ulcer disease.

Imaging by chest radiograph may reveal a retrocardiac gas-filled viscus in cases of intrathoracic stomach. Plain abdominal radiograph may reveal a massively distended viscus. In mesenteroaxial volvulus, the stomach appears spherical on supine abdominal radiographs while erect films often show double air fluid level; inferior representing the fundus & superior representing the displaced antrum.[9] A helpful finding is a nasogastric tube arrested at the GEJ secondary to obstruction. On an upper GI series, the GEJ is usually below the diaphragm, the distal portion of the stomach appears cephalad, and a "beak" may appear where the GEJ is normally located.[10] CT abdomen not only delineates the exact type of volvulus better than barium studies but also helps in detecting the complication as well as the cause of volvulus.

Organoaxial volvulus is difficult to image, particularly if no diaphragmatic defect is present. Plain films may show a horizontally oriented stomach with a single air fluid level and a paucity of distal gas. An upper GI contrast study may show a low lying GEJ and a horizontal or inverted stomach with the cardia and pylorus at the same level.[11] If the pylorus is obstructed secondary to organoaxial torsion, there may be marked gastric dilatation with "beaking" of the contrast material at the site of torsion. CT abdomen delineates the abnormality better. In cases of organoaxial volvulus, plain films and contrast studies may both be falsely negative.

Although the treatment of gastric volvulus is surgical, endoscopic reduction can be attempted in selected patients.

Strangulation and necrosis occur most commonly with organoaxial gastric volvulus (5-28% of cases). Gastric emphysema and perforation occurs secondary to ischemia and necrosis. Perforation can also complicate endoscopic reduction. The nonoperative mortality rate is reportedly as high as 80%. With advances in diagnosis and management, the mortality rate from acute gastric volvulus has been brought down to 15-20%; the mortality rate from chronic gastric volvulus ranges from 0-13%.

REFERENCES

1. ***Tanner, N.*** *Chronic and recurrent volvulus of the stomach. Am J Surg 1968; 115:105-9*
2. ***Dalgaard, JB.*** *Volvulus of the stomach. Acta Clin Scand 1952; 103:131-6*
3. ***Askew, AR.*** *Treatment of acute and chronic gastric volvulus. Ann R Coll Surg 1978; 60:326-8.*
4. ***Wasselle, JA, Norman, J****. Acute gastric volvulus: pathogenesis, diagnosis and treatment. Am J Gastroenterol 1993; 88:1780-4*
5. ***Singleton, AC.*** *Chronic gastric volvulus. Radiology 1940; 34:53-61.*
6. ***Teplick, G, Haskin, M.*** *Surgical radiology. 3rd ed. Philadelphia: W.B. Saunders; 1980. p. 482.*
7. ***Milne, LN, Hunter, JJ. Gastric volvulus****: 2 cases and review of the literature. J Emerg Med 1994; 12:299-306.*
8. ***Chiechi, MV, Hamrick-Turner, J, Abbitt, PL.*** *Gastric herniation and volvulus: CT and MR appearance. Gastrointest Radiol 1992; 17: 99-101*
9. ***Gurney, JW, Olson, D.*** *The gastric bubble: roentgen observations. Radiographs 1989; 9: 467-85.*
10. ***Llaneza, PP, Salt, II WB****. Gastric volvulus more common than previously thought? Postgrad Med 1986; 80: 279-88.*
11. ***Kilcoyne, RF, Babbitt, DP, Sakaguchi, S.*** *Volvulus of the stomach: a case report. Radiology 1972; 103:157-8*

ETHICAL GUIDELINES FOR BIOMEDICAL RESEARCH

The need for uniform ethical guidelines for research on human subjects is universally recognised. It has acquired a new sense of urgency as the critical issues in the area of biogenetic research involving human subjects have become acute. Apart from the mandatory clinical trials on new drugs, a number of diagnostic procedures, therapeutic interventions and prevention measures including the use of vaccines, are being introduced which involve human subjects. Further the advent of new medical devices and radio-active materials and therapeutic benefits of recombinant DNA products have added a new dimension to the ethical issues that need to be considered before evaluating these for their efficacy, utility and safety.

Any research using the human beings as subjects shall bear in mind the following **principles** of : (i) **essentiality**, (ii) **voluntariness, informed consent**, (iii) **non exploitation**, (iv) **privacy** and **confidentiality**, (v) **precaution** and **risk minimisation**, (vi) **professional competence**, (vii) **accountability & transparency**, (viii) **maximisation of public interest** and **distributive justice** (ix) **institutional arrangements** (x) **public domain** (xi) totality of **responsibility** and (xii) **compliance**.

Recent advances in the field of **Assisted Reproductive technologies, organ transplantation, Human genome analysis** and **gene therapy** promise unquestionable benefits to mankind. At the same time, they raise many questions of law and ethics, stimulating public interest and concern.

(Source : ICMR Publication 2000)

PING PONG BALL IN A HEFTY HEART – A CASE REPORT

Rabindranath Das*, Naryayan Goutam**

**Department of Medicine, Manipal Teaching Hospital & **Department of Cardiology ,Padma Nursing Home Pokhara , Nepal*

Abstract: *We describe here a very unsual case of a large free floating left atrial thrombus due to critical mitral stenosis detected by incidental echocardiography, in whom symptoms and complications of thrombus were absent. The patient's only symptom was mild dysponea attributed to smoking. She had no syncope or arrhythmia induced embolic event. Owing to nan-availability of cardiothoracic facility, she was given anticoagulant therapy which resulted in intracerbral haemorrahge and death.*

Key words: *Ball thrombus mitral valve disease, asymptomiatic, anticoagulant, intracerebral bleed.*

INTRODUCTION

Mural thrombus in left atrium or its appendage is a common finding in mitral valve disease with arrhythmia but potentially fatal free foating ball valve thrombus is rare. The thrombus can produce abruptly obstructed mitral valve or catastrophic emblic event. Here we report an unusual case of large left atrial floating thrombus in a long standing case of critical mitral stenosis without arrhythmia or any complication but she died shortly after prophylactic anticoagulant therapy out of intracerebral bleed.

CASE HISTORY

This 53-year old smoker, presented with breathlessness for last one year. She had no syncope or palpitation. There was no history of childhood joint pain or sore throat. Routine physical examination revealed apical middiastolic rumble with presystolic accentuation and pansystolic murmur. She had early diastolic murmur at Erb's area also. Her temperature was 98.4oF, pressure 110/60 and pulse 88/min, regular, All other physical examination was unremarkable. Other laboratory investigations including liver, thyroid function haemorgram, coagulation profile and biochemistry were unremarkable. Electrocardiogram showed right axis deviation, clockwise rotation and right ventricular hypetrophy. Chest skiagram showed cardiomegaly and mitralization of heart. Transthoracic colour flow doppler echocardiography revealed: LA 60 .4 mm, Ao27.7mm, LVID54 mm (diastole) and 37.5 (systole). EF 57%, mitral vlave area 0.69cm^2 and no evidence of valvular vegetation. There was an echogenic large left atrial snow ball like mobile mass resembling a trhombus which rolled and tumbled around the left atrium and bounced off the atrial walls suggesting a free floating thrombus in a case of mild mitral regurgitation, moderate aortic regurgitation and severe mitral stenosis. As there was no cardiothoracic facility available she was put on heparin followed by warfarin with a international normalized ratio of 3.5 On the 6th day of hospital stay, the patient suddenly went into deep coma had convulsion with decorticate posturing. Computerized tomographic scan revealed massive heamorrage in both basal ganglia with ventricular extension the patient died on 10th day of hospitalization.

DISCUSSION

Left-atrial thrombus is an infrequent complication of mitral valve disease. Ball thrombus originates as mural thrombus on interatrial septum. Thrombus enlarges slowly to form a projecting mass being connected with a pedicle from atrial wall. As the bulbous end of thrombus enlarges due to fibrin and platelet deposition, the pedical lengthens and thins out and eventually separates or fragments from the main ball thrombus. Thrombus spins freely in dilated left atrium and acquires characteristic smooth polished appearance[1]. Transient embolism or stroke occurs during thrombus development. Our patient did not experience any such event before hospital admission. In hospital she was diagnosed clinically to have obstructive airway with valvular heart disease, confimed by transthoriacic colour flow doppler study. Her dyspnoea and preferred supine posture were attributed to emphysema and the panystolic murmur was ascribed to tricuspid regurgitation owing to pulmonary hypertension and right ventricular hypertrophy. Due to non-availability of surgical option, she was put on prophylactic warfarin after initial heparin therapy with normal target range of activated partial thromboplastin time and international normalized ration [3 to 3.5]; on the 6th post hospitalization day she suddenly developed haemorrhagic carebrovascular accident, confirmed by brain scan and died on 10th day. Prior to advent of surgical treatment of mitral stenosis nearly 20% of some time during the course of the disease and about 10-15 % of this group died from its complications[2]. Before the era of anticoagulant therapy and surgical treatment, approximately one fourth of all fatalities in patient with mitral vlave disease were secondary to thromoembolism[3]. Embolzation correlates inversely with cardiac putput and directly with patient's age and size of left atrium, 80% of patients with mitral stenosis in whom systemic emboli develop are in atrial fibrillation and this risk of recurrent events exceeds 10% per year. If embolization occurs in patients with sinus rhythm, the possibility of transient atrial fibrillation and underlying infective endocarditis should be considered[4]. Rarely abrupt left atrial outlow tract obstruction from a large pedunculated ball vlave thrombus in variable body position may cause syncope or sudden death, requiring emergency surgical intervention which gives a survival benefit of more than 90%[5]. Free floating ball thrombus is a rare and dramatic finding seen on echocardiography in patients with mitral valve disease and may even persist after valve replacement and adequate anticoagulation[6]. Here the clinical diagnosis was difficult as there were few symptoms and no complications of rheumatic mitral valve disease. Seconly, obstructive airway disease which misled the clinician until she had undergone the colour flow Doppler echocardiogram cliniching diagnosis of multiple valvualr lesions and large free floating ball valve thrombus. Clinically she had presystolic, middiastolic, early diastolic and pansystolic murmur. She had neither varied symptoms like dyspnoea[7] nor any change of murmur in different body position. It was unfortunate that poor socioeconomic condition was also responsible for her premature death.

ACKNOWLEDGEMENT

We are thankful to Prof. V.M. alurkar, professor of cardiology, Manipal teaching hospital, Pokhara, Nepal in preparing this manuscript and also greatful to Dr. Naresh Goyal DM(Cardiology), associated professor, cardiology department, G.B. Pant hospital, New Delhi for his valuable suggestion in the final proof reading of this article.

REFERENCES

1. ***Fraser, A.G. angelini, GD Ikram, S. Butchart, E.G.*** *Left atrial ball thrombus: echocardiography features and clinical implication Eur Heart J. 1988; 9 672-7*
2. ***Nielson, G.H. Galea, E.G. and Houssack, K.F.*** *Thromboembolic complications of mitral vlave disease. Aust. N.Z.J. Med 8.372, 1978*
3. ***Lie, J.T. and Entman, M.L.*** *Hole-in-one Sudden death mitral stenosis and left atrial thrombus Am Heart J. 91, 798 1976*
4. ***Braunwald E*** *Valvular heart disease In Braunwald heart disease, Braunwald (Edr) Vol 2 5th Edition, Prism book pvt. Ltd, Bangalore, India 1997 1007-1076*
5. ***Blanche, C Chaux, A Kass R.M. Helfenstein, J Sugarman, G Free*** *floating ball valve thrombus in the left atrium after mitral valve replacement; successful surigical management Ann Thorac Surg 1985 39 566-8*
6. ***Schen T Lejemtel TH Gacia J. Gordon GM Spevack DM*** *Free floating left atrial ball thrombus following mitral vlave replacement Echocardiography 2005 May 22(5) 438-40*
7. ***Grandmougin D Letourneau, T Favre JP Barral X.*** *Paroxysmal postural dyspnea related to a left atrial ball thrombus Ann Thorac Surg 2002 Nov 74(5) 1691-4.*

Correspondence: **Dr. Rabindranath Das**, *Department of Medicine, Manipal Teaching Hospital, Pokhara, Nepal* ***e-mail:*** *das_rabindranath@ hotmail.com*

The most comprehensive range of
Nephrologicals

TRANSPLANTATION

siromus
The Non-Nephrotoxic **Immunosuppressant**

Tacrolimus
Tacromus
The **Superior CNI** for a better tomorrow

Azathioprine 50 mg
Zymurine
The "FIVE" rupee Azathioprine

CHRONIC RENAL FAILURE

Erythropoetin
ZYROP 1K|2K 3K|4K
The World's Only Thermostable EPO

Nexiron
Iron Sucrose Injection
The Safest Next Generation Iron

Sevelamer Hydrochloride 400 mg / 800 mg
Zynagel
The *non-calcium* phosphate binder

PHOSTAT
Calcium Acetate 667 mg
The Ideal Phosphate Binder

Calcitriol 0.25 mcg.
Calcit
The *Most* Active Vitamin D_3 Analogue

K-BIND
Calcium Polystyrene Sulphonate
The No.1 Potassium Binder

ZavineX
INTERFERON ALFA 2B
The **Unique Interferon** with **TSA**

Renal Specialist
Committed to a better & brighter tomorrow

In service of Medical Profession

Zydus Biogen
We, Better Life

"Zydus Tower", Satellite Cross Roads, Ahmedabad 380 015. India.
Phone : +91-79-26868100 (20Lines) Fax : +91-79-268 66 453
www.zyduscadila.com

NEUROCYSTICERCOSIS PRESENTING AS ISOLATED THIRD CRANIAL NERVE PALSY - A CASE REPORT

C.M. Sharma, Ravindra Singh, R.S. Jain, Shailesh Dixit, B.L. Kumawat, Anjani Sharma

Department of Neurology, SMS Medical College, Jaipur, Rajasthan, India

Abstract: *Neurocysticercosis has varied clinical presentation amongst which isolated oculomotor nerve palsy is infrequently presenting clinical feature. Here, we report at case of acute unilaterial oculomotor nerve palsy. The diagnosis of neurocysticercosis was established by presence of characteristic granulomatous lesion in mid brain and strongly positive ELISA for cysticercal antigen in CSF and serum. Patient responded well to medical therapy.*

INTRODUCTION

Neurocysticerosis (NCC) is the most common neurological parasitosis and continues to be a serious health problem in several states of India with poor socioeconomic status and deficint civic facilities.cysticerci have a predilection for CNS,eyes and striated muscles. The clinical spectrum of NCC is variable and ranges from asymptomatic form to symptomatic forms.The commonest presentation is presentation is partial or secondary genesralized seizures.Here,we report a case of NCC presenting as acute onset isolated unilateral oculomotor nerve palsy.

CASE REPORT

A 22 year old right-handed man presented with three days history of headache followed by drooping of left eyelid (Fig.1). There was no history of vomiting, blurring of vision, fever or trauma. On examination patient was normotensive, general physical & musculoskeletal examination was unremarkabale. Neurological examination was normal except left side partial ptosis with unilateral dilated pupil which was sluggishly reactive to light, with restriction of adduction in left eye.

Routine Investigations like hemorgram, blood glucose and other biochemistry were normal X-ray or chest and soft tissues of thigh did not reveal any abnormality. ELISA for cysitcercal antigen in CSF and serum was positive. Serological tests for tuberculosis and HIV were negative. C.T scan brain showed a ring enhancing lesion in the left mid brain with hyper dense nodule within it and perilesional edema (Fig-2) M.R.I. brain confirmed it to be a cysticercus lesion.

The patients was primed with oral steroids (Prednisolone 40 mg per day) & received albendazole (15 mg/kg/day). The neurological deficit started improving within seven days of treatment and complete recovery was seen after 15 days of treatment. Follow u CT scan after eight weeks showed complete resolution of lesion.

DISCUSSION

The clinical spectrum of NCC is variable and depends on the location, size, number of lesions and the magnitude of hosts inflammatory re3action against parasite in brain[2]. Other proposed mechanisms are ischemia secondary to arterial inflammation and occlusion produced by cysticercal arachnoidities[5]. The common sites of involvement in brain are cerebral parenchyma, meninges and ventricles. Brain stem and cerebellum are infrequently affected and are usually part of disseminated form of neurocysticercos[4] is but the incidence of isolated solitary cyst in brainstem is rare[3,6,7,8].

The usual presenting features of NCC are partial or secondary generalized seizures. Signs and symptoms or raised intracranial pressure and mental changes are also been and clinical presentation like begin intracranial hypertension is not uncommon. Many patients show focal neurological



deficit, which is a result of parenchyma presence of cysticerci[4].

Cysticerci are seen in unusual locations with interesting clinical findings. Mesencephalic syndrome described by Sawhney et al9 has spectrum of clinical features including upward gaze palsy, convergence retraction nystagmus, eyelid retraction, pupillaryu abnormalities and skew deviation. The underlying pathology may be necrosis or location of cyst in dorsal mid brain itself.

Our patient was interesting in view of location of the lesion in dorsal mid brain resulting in acute onset partial coulomoter nerve palsy with papillary involvement The possible mechanism for deficit in this patient may be the lesion itself. The rapid improvement could because of reduction in perilesimal edema. Thought other common causes of acute third nerve palsy are diabetes mellitus, vacuities due to intra cranial infection and posterior communicating artery aneurysm. The papillary involvement in ptosis should prompt early neuron imaging studies to exclude the space occupying lesion.

REFERENCES

1. ***Delbrutto OH, Sotelo.*** *J Neurocysticercosis A update Rev. Infect Dis 1988 10.1057-87*
2. ***McCormick G.F.*** *Cysticercosis a review of 230 patients. Bull clin neurosici 1985:50,76-101*
3. ***Rajshekhar V.E. Joseph, R. Kachnara et al****-Neurocysticercosis presenting as a Solitary cyst in the brainstem-A case report: An of Indian Academy of neurology 1999 vol. 2:99-100.*
4. ***Del Brutto OH****: Cysticerosis- cerebrovascular disease, review: J Neurol, Neursourg, Psychiatry : 1992 55: 252-54.*
5. ***Levy, A.S. Lillehei, K.O Rubinstein, D Stears, J.C. subarachnoid*** *Neurocysticercosis with occlusion of major intracranial arteries. A Case report J. Neurosurgery 1995 36 183 186.*
6. ***Chotmongkolv' suranyauisuth K Limpaawettana P. et.al*** *superior divisional occulomotor palsy caused by midbrain neurocysticercosis. Parasitol Inmto 2006 sept 55(3) 223-225.*
7. ***Garg RK. Karak B****, Neurocycticrecosis presenting as midbrain syndrome, Journal AssocPhysicians India 2000 May: 48(5) 533-5*
8. ***Singh N N, Verma, R, Pankaj B K et al****: Neurocysticercosis presenting as weber syndrome, Neurology India 2003 Dec.51(4) 551-52*
9. ***Sawhney IMS, singh G, Lekhra OP et al****, Uncommon presentation of cysticerosis, J, Neurolsci: 1988: 154:94-100.*

Correspondence: **Dr. C. M. Sharma,** *150 Hari Marg, Civil lines, Jaipur, India* ***Phone:*** *0141- 2225684*

Presenting

The proven therapy to delay progression of

Chronic Kidney Disease

Ketosteril®

Protects and Preserves Renal Function



- **Provides nitrogen sparing effect**
- **Reduces hyperfiltration of nephrons**
- **Improves metabolic complications**

Recommended for all patients with:

- **Proteinuria, even micralbuminuria**
- **Creatinine clearance < 50 ml/min**

Delays the onset of dialysis and Prolongs life expectancy

Dose: 1 tab/5kg bw/day



Fresenius Kabi

Caring for Life

For further detailed information please contact
Fresenius Kabi India Pvt. Ltd.
Heritage House, 6-E, Ramabai Ambedkar Road, Pune - 411001, India
Ph. : 91-20-26053602-7 Fax : 91-20-26138258
www.fresenius-kabi-india.com

CASE REPORT

SILDENAFIL IN THE TREATMENT OF PRIMARY PULMONARY HYPERTENSION – A CASE REPORT

N.S. Neki

Department of Medicine, Govt. Medical College/Guru Nanak Dev Hospital, Amritsar (Punjab), India

Abstract: *Pulmonary arterial hypertension (PAH) is a life threatening, progressive and often fatal, debilitating disorder with a grave prgnosis. The increased pulmonary artery(PA) pressure found in PAH is due to disturbances in key vascular mediator pathways including relative deficiencies of vasodilators like nitric oxide (NO) and prostacycline, as well as increased production of vasoconstrictors like endothelin and thromoboxanes. Various vasodilators such as bosentan, eporosternol, iloprost and sildenafil have a definite role in PAH inhibitor, which is stabilization of cyclic guanosine monophosphate (C GMP), resulting in increase in NO at the tissue level thereby causing pulmonary artery vasodilatation. Sildenafil is more potent acute pulmonary vasodilator than inhalded metric oxide (NO). In addition sildenafil has been found to exert additive effects on pulmonary hemodynamics when used in combination with inhalded iloprost or NO. A case of severe pulmonary arterial hypertension managed effectively with sildenafil is being reported here.*

INTRODUCTION

Pulmonary arterial hypertension (PHA) includes primary pulmonary hypertension (PPH) with no apparent cause, PAH secondary to congenital shunt lesions, connective tissue disorders, portal hypertension, drug induced and HIV related PAH. It is a progressive disease, affects predominantly women in early life and is characterized by progressive breathlessness, exertion limitation, frequent decline and failure of right ventricle. It is estimated that PAH affects 100,00 patients worldwide. Although many treatments are available, but there is no known cure[1,2] The current therapeutic options available for PPH include calcium channel blockers anticoagulants, eporostenol (prostacycline), iloprost, bosentan and sildenafil[3]. Sildenafil a selective PDE-5 inhibitor is a potent pulmonary vasodilator and is effective in patients of moderate to severe PH[4]. Otral sildenafil has been found to be clinically effective in PPH refractory to epoprostenol[5].

CASE REPORT

A 25 years old female nondiabetic, non hypertensive presented with worsenining exertional dyspnoea being unabale to cycle for 100 yards (NYHA) class III) and increasing fatigue sisnce 5 years. There was no history of orthopnoea, syncope and hemoptysis. She complained of swelling of feet and case since last 3 months. She denied any history of exposure to chemicals, appetite suppressants or collagen vascular disease. No member of ther family had such problem. Her physical examination revealed poor body built, pulse 110/min, regular, BP 100/60 mmHg, JVP raised with prominent a and v waves with bilaterial edema. There was no cyanosis or clubbing. Cardiovascular examination revealed right ventricular heave, signs of tricuspid regurgitation (TR) and ejection systolic murmur in the pulmonary area. Right ventricular S3 gallop suggestive of right ventricular failure was audible. Her laboratory findings revealed Hb 9.2 G/d1, TLC &DLC were within normal limits. Total collagen profile was normal and HIV non reactive. ECG showed evidence of sinus tachycardia with features of right arterial enlargement with right ventricular hypertrophy with strain pattern and right axis deviation suggestive of pulmonary hypertension. X – ray chest revealed right heart enlargement with dilatioation of main pulmonary artery as well as right descending pulmonary artery. Echocardiography revealed dilated right sided chambers and main pulmonary artery. Lung functions showed only mild airflow limitation. Doppler echocardiography demonstrated evidence of TR and PAH. The estimated systolic pulmonary artery pressure was 110 mmHg and right atrial pressure of 22 mmHg. Left ventricular systolic function was preserved.

The patient was started on dioxin, diuretics, warfarin (anticoagulant) and oral sildenafil given 25 mg thrice daily. Gradually the dose of sildenafil was titrated to 150 mg/day. The patient started improving within 3 weeks of therapy and showed progressive improvement over the next three months. On a follow up at 6 months, she was able to cycle 200 yards (NYHA Class II) comfortably. Echocardiography showed a significant reduction in systolic PA pressure from previous 110 mmHg to 90 mmHg after treatment.

DISCUSSION

PPH is characterized by progressive elevation of mean pulmonary artery pressure (PAP) more than 25 mmHg at rest, right ventricular failure and death[2]. Current therapeutic options for PPH mostly include limitation of physical activity, long term anticoagulation and vasodilator therapy Calcium channel antagonists are the main stay of therapy, but only 25-30% of patients respond. Major advancement in the treatment of PPH is the development of two orally active agents bosentin (endothelin receptor antagonis) and Sildenafil which are selective pulmonary vasodilators. Sildenafil inhibits cyclin GMP specific phosphodiesterase enzyme resulting in smooth muscle relaxation via a nitric-oxide dependent increase of GMP further producing decrease in elevated PAP [3,4]. Other treatment options include epoprostenol (prostacycline) infusion and heart lung transplantation, which are limited by nonavailability in many parts of India, clinical difficulties, complications and high costs [5,6] . The dose of sildenafil for PPH remains unknown. Studies in awake limbs7 with PPH demonstrated a dose related (serial dose 12.5 mg, 25 mg and 50 mg) reduction of PAP by 21% and 43% and pulmonary vascular resistance by 18%, 23% and 45% respectively. But in a study by Malhotra S et al[8] using oral sildenafil 25 mg thrice daily titrated to 150 mg/day, there was reduction in PAP to the tune of 20 mmHg and in the present study it was again 20 mmHg. Sildenafil was tolerated without any tolerance and side effects. When administered in combination with inhaled nitric oxide (NO) augments and prolongs the pulmonary vasodilatory effect of NO and prevents the rebound pulmonary vasoconstriction which occurs after NO inhalation in the absence of sildenafil. It acts synergistically with inhalded iloprost to cause strong pulmonary vasoilattation in PPH[9].

REFERENCE

1. *Mehta S. Sildenafil for pulmonary arterial hypertension : exiting but protection required. Chest 2003, 123, 989-92*
2. *Sastry BK, Narasimhan C, Reddy NK et al. A study of clinical efficacy of Sildenafil in patients with primary pulmonary hypertension, Indian Hear J 2002, 54 410-4*
3. *Kothari SS, Bahal VK. Primary pulmonary hypertension-an update. Indian Hear J 2002, 54, 255-60*
4. *Karatza, Aa Bush A, Magee AG. Safety nad efficacy of sildenafil therapy in children with pulmonary hypertension Int J Cardiol 2005, 100 267-73*
5. *Katakoka M, Satoh T, Manabe T et al. Oral Sildenafil improves primary pulmonary hypertension refractory to epoprostenol. Cir J 2005, 69 461-5*
6. *Higgenbotam TW, Spiegelhaltor D, Scott JP et al. Prostacycline (epoprostenol) and heart lung transplantation as treatment for sever pulmonary hypertension. Br Hear J 1993 70 366-70*
7. *Weimann, J Ullrich R, Hromi J et al. Sildenafil is a pulmonary vasodilator in awake with acute pulmonary hypertension Anaesthesiology 2000.92 1702-12*
8. *Malhotra S, Punia VPS Oral Sildenafil in the management of primary pulmonary hypertension JAPI 2004, 52 670-72*
9. *Ghofrani HA Wiedeman R, Rose F et al, Combination therapy with oral sildenafil and inhaled iloprost for sever pulmonary hypertension. Ann Intern Med 2002, 136 515-22.*

Correspondence: **Professor N.S. Neki,** *Department of Medicine, Govt. Medical College/Guru Nanak Dev Hospital, Amritsar (Punjab) India. e-mail : drnsneki_123@yahoo.com*

CYRIN™

Cyclosporine Capsules USP



The Critical **Success Factor**
in Transplant Therapy

LIMUS™

SIROLIMUS



The transplanted
Kidney's Lifeline!

MYGRAFT™

Mycophenolate Mofetil

Partnering
Graft Survival



EPOTIN™

Recombinant Human Erythropoietin Injection



Adding years
to Life...

SUCROFER™

Iron Sucrose Injection USP



The
SAFE & EFFECTIVE
IV Iron

Claris

Committed to Nephrology...

Corporate Towers, Ellisbridge, Ahmedabad 380 006, India.
Tel : +91-79-26563331 Fax : +91-79-26408053/26565879
Email: crcproducts.cerp@clarislifesciences.com www.clarislifesciences.com

AN UNUSUAL CASE OF ABDOMINOCYESIS FOLLOWING EARLY FUNDAL RUPTURE OF UTERUS

Purvita Dam, Dinesh Pathak, Sebanti Goswami, Sudhir Adhikari

Department of Obstetrics & Gynecology, Medical College, Kolkata, India

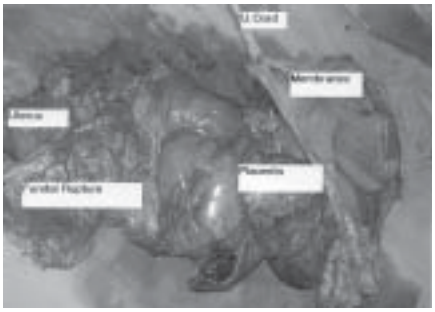
Abstract: *Abdominocyesis is characterized by its morbid pathological process and fetal salvage. In this case, after a is exceedingly rare condition and prolonged period of amenorrhoea, the patient conceived; pregnancy, continued as a secondary abdominal pregnancy after fundal rupture and despite repeated sonology, the condition went undiagnosed. Laparotomy revealed an amniotic sac containing a healthy baby with precarious attachment of placenta and membranes within the peritoneal cavity. Supracervical hysterectomy was performed in view of the wide rupture of the fundus of the uterus. Healthy mother and son were discharged subsequently.*

INTRODUCTION

Existence of primary implantation of fertilized ovum in the peritoneal cavity is questionable. Abdominocyesis is almost always secondary, primary sites being tube, ovary or even an uterine rent. Minor ailments of pregnancy are often exaggerated. Absence of Braxton Hicks contraction and persistent abnormal attitude of fetus should provoke a suspicion. Diagnostic error in sonography may go upto 40%. Hence a clinically conscious provides alead; surest evidence is on laparotomy. Catastrophic hemorrhage and fetal malformation are the primary complications associated with continuation of pregnancy. Ideal surgery involves removing the entire sac-fetus, placenta and membranes. However difficulty encountered often may result in leaving behind the latter two and await aseptic autolysis risking secondary hemorrhage, intestinal obstruction and infection. Maternal mortality with the advances of surgery has been reduced to 5%, but perinatal mortality and morbidity remain as high as 90%.

CASE REPORT

Mrs G.P., a 26year old gravida 2, para1, living issue nil was admitted through the out patient department with amenorrhoea of 9 months duration and clinical as well as radiological diagnosis of transverse lie with central placenta praevia. On examination her pulse was 84/min and blood pressure 130/82mmHg. Previously she was admitted in June 2005 with severe pain abdomen and marked pallor with sonological findings of oligohydramnios. She received 4 units of A+ve blood and was discharged from hospital after conservative management. This was followed by regular antenatal check up. An ultrasound revealed single live intrauterine gestation of 30weeks in transverse lie, placenta completely covering internal os, scanty liquor (Fig.). Her obstetrical history revealed a previous term IUFD with obstructed labor managed by craniotomy subsequent to which she developed a vesico-vaginal fistula that was successfully repaired vaginally 10years back. Subsequently she developed secondary amenorrhoea for 8 long years, treated outside both medically and surgically(D/C); she conceived immediately after the inception of menstruation for the second time. Since her previous antenatal records and previous ultrasound pointed towards term, we decided to go for elective caesarean section with two units of blood in hand keeping the placental position in mind once she started complaining of decreased fetal movements. Laparotomy was done on 16/9/05, which surprisingly revealed intraabdominal gestational sac on incising the parietal peritoneum. Baby was delivered having Apgar score 10 and further exploration revealed an uterus lying low down with ruptured fundus. Placenta was on the internal os anterior and posterior wall of uterine cavity and also on posterior wall of uterus with partial attachment on left broad ligament. Cord had membranous attachment the sac was attached to omentum and transverse colon. Placenta was completely removed, membranes partially without damaging the omentum and gut. Supracervical hysterectomy was performed and a total of 4 units of blood transfused both intra and post operatively. Apart from mild depression of right temporal bone the baby was absolutely normal. Mother and son were discharged on the 8th postoperative day. Follow up at 6 weeks confirmed a normal healthy infant with appropriate milestones and an absolutely healthy mother.



DISCUSSION

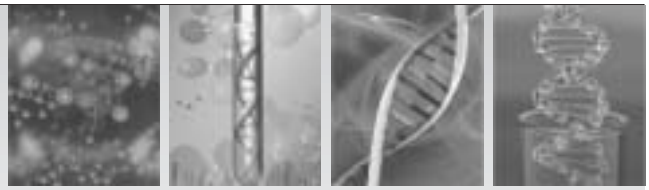
Secondary abdominal pregnancy or abdominocyesis is a condition in which the embryo or fetus continues to grow in the abdominal cavity after its expulsion from the tube or other site of its primary development. Incidence is 1 in 3000. Weakening of uterine musculature at fundus possible resulted from previous craniotomy or subsequent D/C. Bladder was relatively free even after repair of VVF. The risk of maternal mortality is 7.7 times greater than tubal pregnancy and 90 times higher than an uterine pregnancy.[1] Recurrent abdominal pain, vomiting in the second and third trimesters, and painful fetal movements are frequent complaints. Often, fetal parts are easily palpated, with the fetus high in the abdomen in a transverse or oblique lie. Anemia is occasionally noted late in the first or second trimester; this is due to intra-abdominal bleeding at the time of partial placental separation as was seen in our case. An unusual lie that does not change over time is also highly suggestive. Misleading ultrasound may be due to stretched omentum over the gestational sac giving the appearance of uterine wall.[2] Fetal abnormalities are seen in 30-95% of survivors and are related to oligohydramnios and to the constricted extrauterine environment, which results in pressure deformities.[3,4] Our baby fortunately survived with minimal deformity.During laparotomy, if the blood supply to the placenta can be safely secured, removal of the placenta completely usually results in good postoperative recovery. In a series of 101 cases, the placenta could be removed completely in 60% and most authors now agree that removal of placenta if straightforward should be the optimal approach. Removal of pelvic organ on which implantation has occurred may be necessary like hysterectomy or salpingo-oophorectomy.[5] Abdominocyesis results in very poor fetal salvage but in our case timely intervention has allowed a very succesful and satisfactory outcome.

REFERENCES

1. *Atrash HK, Friede A, Hogue CJR. Abdominal pregnancy in United States, its frequency and maternal mortality. Obstet Gynecol 1987;69:333.*
2. *Chamberlain G. Caesarean Section. In: Turnbull's Obstetrics. Editors: G. Chamberlain, P.J. Steer, Assc Editors: G.Breart, M. Johnson, A. Chang, J.P. Neilson 2001; 37: 603-604.*
3. *Hallatt JG, Grove JA. Abdominal pregnancy: a study of twenty-one consecutive cases. Am J Obstet Gynecol. 1985; 152: 444-449.*
4. *Rahman MS, Al- Suleiman SA, Rahman J, et al. Advanced abdominal pregnancy. Obstet Gynecol 1982; 59: 366-72.*
5. *Costa SD, Presley J, Bastert G. Advanced abdominal pregnancy. Obstet Gynecol Survey. 1991; 46: 515-525*

Correspondence: Dr. Purvita Dam, *26, Sarat Chartterjee Road, Kolkata, India (M) : 09830184589* ***e-mail:*** *purvik_mdgoal@yahoo.com*

Emcure Research **leads again** in the management of hyperphosphatemia in CKD



1st Indigenous Sevelamer Carbonate

Sevelamer Carbonate

Sevcar™ 400 / 800

added advantage of carbonate buffer

- Increases serum bicarbonate
- Reduces events of metabolic acidosis
- Enhances treatment of uremic osteodystrophy
- Reduces chances of protein catabolism
- Better GI tolerability than Sevelamer Hydrochloride



Emcure RENAL CARE
www.emcure.co.in

partners in health, healing & hope

For the use of a Registered Medical Practitioner or a Hospital or a Laboratory only.

CASE REPORT

SWYER JAMES (MACLEOD'S SYNDROME)

MMA Kamili, GH Hassan, AH. Manzoor. M. Yaseen, Ishrat F Omer, Tajamul

Department Of Medicine & Associate SMHS Hospital

Government Medical College, Srinagar, Kashmir-190010, India

Abstract: Macleod's Syndrome is a disorder of unilateral emphysema, bronchiecasis and loss of lung volume following bronchiolitis occurring or patchy bronchitis in infancy or childhood. We describe a case who presented with polycytemia and corpulmonale; thisunusual presentation has not been reported so far.

INTRODUCTION

This is a ratre disorder characterized by unilateral Emphysema of a lung or resulting from localized bronchiolitis or bronchitis following viral infections or tuberculosis during infancy or child hood. Radiologically there is unilateral hyper translucency with diminished pulmonary vascular markings and normal or reduced lung vlume and bronchiectasis [1-4]. The disorder was initially described by Swyer and James in 1953 2 and by Macleod a year 4 and subsequently few more cases were reported. We describe the syndrome in a 16 year old male who presented with polycythemia and cor pulmonale possibly representing the first case of such nature in literature.

CASE REPORT

A sixteen year old male was admitted with history of productive cough and dyspnea since isx months of age with frequent hospitalizations. On examination he was grossly cyanosed, with pectus carinatum and features of corpulmonale with right heart failure. On investigations he had hemoglobin of 18.5 gm/dl and hematocrit of 66%. Chest radiograph revealed evidence of bilateral bronchiectasis, emphysema and reduced lung volume on rith side and cadiac enlargement of right ventricle (figure1). Electrocardiogram revealed right axis deviation p-pulmonale, and R in V1 and V2 High resolution computed tomography (HRCT) showd bilateral bronchiectases, and hyper lucent lung fields with emphysema on right side (Figure2) Echocardiography demonstrated pulmonary arterial hypertension, tricuspid regurgitation and other findings of cor pulmonale. On pulmonary function testing he had mild obstruction. The serum levels of alpha one antitrypsin were normal and all other investigations including serum electrophoresis and immunoglobulin levels were normal. Arterial blood gas analysis revealed desaturation (68% and respiratory acidosis. The patient was managed with oxygen, diuretics, phlebotomies and other supportive measures. He improved symptomatically and clinically and is under regular follow up.

DISCUSSION

On account of rarity, there is paucity of literature regarding the disorder. It seems likely that this disorder starts between six months to five years of age after viral broncholitis or tuberculosis, and is characterized by cough, dyspnea, hemoptysis and clinical manifestations of bronchiectases [1.3.4]. The affected lung is usually subnormal in size because of the subsequent underdevelopment of lung after bronchiolitis. If the disorder occurs early in life, the lung fails to grow normally and remains smaller than the opposite lung but if it occurs later in childhood, the lungs may be of normal size [1.4]. On histopathological examination there is pancinar emphysema, with fever alveoli than normal. The pulmonary artery is often hypo plastic on angiography possibly due to local hypoxemia. There is often hypertrophy of the pulmonary artery walls and a decreased number

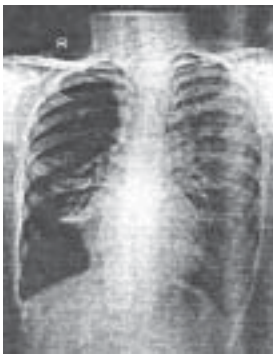


Figure-1*:Postero anterior view of chest readiography showing bilateral bronchiectatsis, emphysema on right side and features of cor pulmonale.*



Figure-2 :*HRCT showing bilateral bronchiectasis and emphysema on the right side*

of branches. On pulmonary function testing there is often evidence of airway obstruction and an increase in the residual volume. Bronghography demonstrates diffuse bronchiectasis with absence of filling of the terminal bronchioles. Radionuclide one scanning shows decreased ventilation and persuasion in the affected area. HRCT is considered as investigation of choice for identifying the characteristic changes [2.5]. The disorder needs to be differentiated from compensatory emphysema, where the collapsed opposite lobe can by visualized and the vessels in emphysematous lobe are fanned out. The occurrence of cor pulmonale as in our case is most likely due to hypoxic vasoconstriction following emphysema and the diminished vascular cross sectional area from bronchiectasis [6] In future further studies are needed regarding etiopathogenesis, treatment modalities, and prognosis of the disorder.

REFERENCES

1. ***Seaton A, Seaton D, Leitch AG****. Chronic Broncjitis and Emphyseama in Crofton and Douglas's Respirtatory Diseases: London 4th Ed 1990 490-525.*
2. ***Swyer PE, James Gow.*** *A case of unilateral pulmonary emphysema, Thorax 1953; 8 133-136*
3. ***Macleod WM*** *Abnormal treansradinancy of one long. Thorax 1954; 9:147-149*
4. ***Marti-Bonmati L, Perales FR, Catala F etal****. CT findings in Swyer-James syndrome, Radiology 1989; 477-480*
5. ***Swartz MN*** *Bronchiectasis in Fishman AP Elias JA Fishman JA (Eds), Fishman's Pulmonary Diseases and Disorders 3rd Ed, Mc Graw Hill: New York 1998; 2045-2070*
6. ***Behera D,*** *Cor pulmonale In Text book of Pulmonary Medicine 1st Ed, Jay Pee brothers; New Delhi 1995: 394-402.*

Correspondence: Dr. M.M.A Kamili, *Professor and Head, Department of Medicine,Government Medical College, Srinagar, Kashmir-190010, India*

GILLES DE LA TOURETTE'S SYNDROME:

A COMMONLY MISDIAGNOSED NEUROBEHAVIORAL DISORDER

Kuljeet Singh Anand, Jyoti Garg, Yogesh Valecha, Ranjit Singh Makar

Department of Neurology, Dr. RML Hospital PGIMER, GGS-IP University, New Delhi-110001, India

Abstract: *We report two cases of Gilles de la Tourette syndrome Case 1, thrity year old male, presented with complains of episodes of finger drumming and titubation. Upon further enquiring into the history, the patient recalls history of multiple tics occurring daily since childhood. There was history of obsessive compulsive bhaviour and palilalia. Case 2 is that of a ten year old boy who also presented with multiple motor tics. The parents ggave history suggestive of obsessive compulsive behavior, attention deficit hyperactive disorder and coprolalia. In both case the routine haematological and biochemical investigations were within normal limits. CT scan head and EEG were normal. Based on the clinical presentation a diagnosis of Tourettes syndrome was made and treated with antpsychotic. Both the patients had significant improvement within 1 month of therapy.*

INTRODUCTION

Tourette syndrome (TS) is a neuropsychiatric disorder with onset usually in the first two decades of life characterized by a combination of multiple motor tics and at least one phonic tic. Phonic tics may be simple sounds (simple phonic tics such as grunting or sniffing) or whole words (complex phonic tics). In the latter case they are called complex phonic tics whose most characteristic example is coprolalia (searing or emission of obscenities). Tics are often associated with behavriorl abnormalities such as hyperactivity and attention deficit disorder, obsessive compulsive disorder and others as was seen in both of our cases.

CASE REPORTS

Case 1: A 30 year old male, presented with episodes of involuntary movement of the fingers of the right hand in the form of repeated tapping and turning of the head. Upon further enquiring in to the history, the patient recalls similar involuntary movements, which were identified as tics, occurring since childhood. He first initially noticed the tics when he was 6 years old and had complains of frequent eye blinking . Later he developed tic in the feet and hands. These occurred daily and would cause significant embarrassment. He used to cover up by putting his hands in the pocket and start pacing. He faced difficulty in learning, academics and work performance due to lack of concentration. He also had complex vocal tics and used to repeat ones own utterances particularly the last word (palilalia). There as history suggestive of obsessive compulsive behavior in the form of repeated hand washing due to fear of contamination. Both motor and phonic tics used to exacerbate during times of stress such asexaminatons and interviews, so much so that in one instance he was sent out of the examination hall, suspecting that he was cheating in the exam. He was also refused many jobs and was fired twice from his job owning to his problem. He as born following a fulterm normal delivery and had normal developmental milestones. No family history of any psychiatric or neurological illness. On examination his higher mental functions were intact. His mood was congruent with his affect. There was no abnormality in the thought process. There was no cranial nerve involvement. No sings of Motor, sensory and cerebellar involvement. He had a normal gait. Haematological and biochemical investigations were unremarkable. Ct scan head and EEG were normal. Based on the classical presentation a clinical diagnosis of Tourette;s syndrome was made and the patient was started on antipsychotic medication Resperidone (1mg twice daily). He responded significantly with disappearance of tics within 1 month of treatment. The quality of life has also significantly improved and patient in now performing well academically and proressionally.

Case2.: A ten year lod boy who was brought by the parents with complains of requent eye blinking, head jerking and shoulder shrugging. The parents recall, they first noticed the problem when he was 7 years old. It started with eye blinking and later the lateral head truning and shoulder shrugging. These motor tice used to occur daily. The child was also very fidgety and would get distracted very easily. His teachers complained about his fidgidity and that he lacked concerntraion during his teaching sessions. His academic performance was poor. He was also very impulsive with a low threshold for engaging into fights with his friends. There was history suggestive of obsessive compulsive behavour, felt that his hands are dirty and contaminated to the extent that he suded to wash them 20-30 times a day His parents also complained that their son had started using abusive language,. swearing words for no reason to everyone including his parents because of which he ass punished may a times but was still unable to control himself. All this caused a lot of frustration and embarrassment and now he had very few friends. He as born following a ful term normal delivery. No history suggestive of any perinatal insults and had normal developmental milestones. He has two elder brothers and one younger sister, none of whom exhibit any fetures of aneurobehavioural disorder. On examination his higher mentalfunctions and cranial nerves were intact. No sings of motor, sensory cerebeelar involvement and had normal gait. Upon investigations he had normal haematological and biochemical exams. CT scan head and EEG were normal. A diagnosis of tourette syndrome was made and he was started on haloperidol. He showed significant improvement with 6 weeks with decreased frequency of motor and phonic tics but still had some features of attention deficit disorder. His academic performance has improved and is now getting good grades in school.

DISCUSSION

Tourette syndroms (TS) named after George gilles de la tourette who in 1885 coned this syndrome of persistent but changing tics and coprolalia beginning in childhood. It is a neuropsychiatric disorder with onset usually in the first two decades of life characterized by a combination of multiple motor tics and at least one phonic tic. This syndrome is often misdiagnosed as a psychiatric illness2 A. family

Correspondence: Dr. K.S. Anand, *Senior Neurologist & Head of Department, Phone: 23404517*

history is sometimes obtained, and partial expression of the trait may occur in siblings or offsprings of patients. In most families with chronic tic disorders, there is an autosomal dominant mode inhertiaance with variable penetrance that is gender related. Although the disorder has been reported in association with chromosomal abnormalities, in vast majority of cases the chromosomes are normal3 boys are affected much more commonly than girls. On the basis of the etiology, ST is classified into two catergories. idiopathic and symptomiatic symptomatic TS can occur after head inury encephalitis stroke, rheumatic fever, and drug intake (Methylphenidate, neuroleptics, opioids, antiepileptics), Motor tics commonly affect the face and may consist of repetitive snfiffing, winking blinking, elevation of eyelids eye closure, pursing of lips or fcial twitching, patients eventually develop several different motor and phonic tics, the latter frequently taking form of grunts, barks, hisses, sighs, throat clearing, coughing, phonic tics may be simple sounds (simple phonic tics such as grunting or sniffing) or whole words (complex phonic tics) In the latter case they are called complexphonic tics whose most characteristic example is coprolalia (swearing or emission of obscentities) It must be emphasized that although possibly the most characteristic symptom of Tourette syndrome4 coprolalia is not mandatory for the diagnosis of this codition. The pathophysiology of coprolalia and other complex phonic tics remains ill defined. The tics may change in location, severity, complexity and character with time are worsened by emotional stress and can be suppressed voluntarily for short periods of time. Tics are often associated with behavioral abnormalities such as hyperactivity and attention deficit disorder, obsessive compulsive disorder and others. These co morbid conditions offer interference in learning, academic and work performance. it has a life long course with remissions and exacerbations. Many patients have non specific electroencephalographic abnormalities of no diagnostic significance . Treatemnt responses is seen with high potency antipsychotic durgs including pimozide, haloperidol and respiridone.

There have been many cases reported in the Indian literatute. A recent study by Anand et al5 showed the clinical profile of the cases reported in India is similar to those reported from the western counties with the exception of coprolalia and ADHD, the incidence of which is higher in the cases reported in India. The pathophysiology is obscure and no structural pathology has been reconginzed. A dopaminergic excess has been suggested by the clinical observation that the tics may respond to treatement with dopamine blocking drugs.

In another report by Duggal eta al6, they have studied the Bereitschafts potential, movement related cognitive potential indicative of motor preparation, in three patients with Tourette syndrome and have implicated quasi volitional nature of tics and that they are not totally involuntary, Desai et al7 described the use of metoclopramide, a potent dopamine antagonist, in patients with Tourette syndrome.

Kumar s8 reported Neurofibromatosis type1 manifesting with Tourette syndrome, a rare presentation, and pointed towards the pathological involvement of caudate and lentiform nuclei in the genesis of tics in TS,

CONCLUSION

Gilles de la Tourette's syndrome is often misdiagnosed as a case of psychiatric disorder, as was the scenario in our patient. These patients are subjected to unnecessary, expansive investigations and treatment before the corret diagnosis is made, Depression developed in our patients, due to the social embarrassment cause by the tics, which could have easily been avoided with the early recognition of this syndrome.

REFERENCES

1. *Enoch MD Ball HN Gilles de la tourettes syndrome in Uncommon psychiatric syndromes London Arnold 2001 134-136*
2. *Micheli F Gatto M, Grshavik O Gilles de la Tourette syndrome, Clinical features of 75 cases from Argentina Behavioural Neurol 1995 18 75 80*
3. *Merskey H Gilles de la tourettes syndrome Brit J psychiat 1974 125 593*
4. *Robertson MM Banerjee S Kurlan R et al the tourette syndrome diagnostic confidence index development and clinical associations Neurology 1999 53 2108 2112*
5. *Anand KS Gurtoo Anil Garg Sandeep et al Gilles de la tourettes syndrome case series of 14 patients from India Ann Ind Acad Neurol 2005 8 49 52*
6. *Duggal H.S Haque Nizamie S Bereitschaftspotential in Tic Disoreders a Preliminary Observaton Neurology India 2002 50 487 489*
7. *Desai AB Boongaji DR Satoskar RS metoclopramide in Gilles de la Tourettes syndrome (a case report) J Postgrad Med 1983 181 183*
8. *Kumar S. Neurofibromatosis type – 1 mainifesting with Tourette syndrome, Neurol India 2005 53 361 362*

DRUG PROFILE

Rifaximin

Pharmacokinetics: Rifaximin, a semi-synthetic, nonsystemic antibiotic. Rifaximin can be administered with or without food. Systemic absorption of Rifaximin was low in both the fasting state and when administered within 30 minutes of a high fat breakfast. There is no evidence of accumulation of Rifaximin following repeated administration for 3 days (9 doses). Peak plasma Rifaximin concentrations after 3 and 9 consecutive doses ranged from 0.81 to 3.4 ng/mL on Day 1 and 7.83 + 4.94 ng-h/mL on Day 3. Rifaximin is suitable for treating systemic bacterial infections because less than 0.4% of the drug is absorbed after oral administration. Animal studies have that 80% to 90% of orally administrated Rifaximin is concentrated in gut with less than 92% in the liver and kidney, and less than 0.0 1% in others tissues. In adults with infectious diarrhiea treated with Rifaximin 800 mg daily for three days at Rifaximin is excreted primarily in the feces. After oral administration of 400 mg. ***Indications:*** Escherichia coli infection of the guity to the main indication Rifaximin to healthy volunteers approximately 97% of the dose was recovered in feces, almost entirely as unchanged drug, and 0.32% was recovered in the urine. ***Special Populations***: ***Geriatric*** : The pharmacokinetics of Rifaximin in patient> 65 years of age has not been studied. *Pediatric:* The pharmacokinetics of Rifaximin has not been studied in pediatric patients of any age. *Renal Insufficiency:* The pharmacokinetic of Rifaximin in patients with impaired renal function hasbeen studied. *Hepatic Insufficiency*: Rifaximin plasma concentrations of 13.5 ug/mL. were detected in hepatic encephalopathy administrated Rifaximin 800 mg three times daily for 7 days. Drug-Drug Interactjns, In an in vitro hepatocyte induction model Rifaximin was shown to induce cytochromic P450 3A4 (CYP3A4), an isoenzyme which Rifampin is known to induce. Two clinical drug interaction studies have been conducted using midazolam, Rifaximin has not shown to significantly affect intestinal of hepatic CY3A4 activity. The oral contraceptive showed study that the phannacokinetic of single dose of ethinyl estradiol and gestimate were not altered by Rifaximin ***Dosage and Administration:*** Rcifax tablets can be administered orally with or without food. For travelers diarrhea, the recommended dose is one 200 mg tablet taken three times a day for 3 days. ***Adverse Effects***: Common side effects include nausea, flatulence, constipation, vomiting, pyrexia, headache, LyIi/abdominalsain, rectal tenesmus, defecation urgency. ***Warning:*** The drugs is in effective in cases of travelers, diarrhea due to Campylobactojejunis; Should be discontinued if diarrhea persists; antibiotic therapy should be considered. Pseudomembranous colitis has been reported with use of antibiotics due to the overgrowth of nonsusceptible organisms; should superinfection occur during therapy, appropriate measures should be taken.

CONTROVERSIES IN THE SURGERY OF BENIGN THYROID DISORDERS

A.N. Sinha, Dalbir Singh Sandhu

Department of Surgery, Rockland Hospital, Qutab Institutional Area, New Delhi 110016, India

Abstract: *Use of total thyroidectomy (TT) for differentiated thyroid carcinoma is controversial, but even more controversial is the use of TT for benign thyroid disease. The utility of TT for benign thyroid diseases aims at avoiding the risk of leaving behind abnormal thyroid tissue, a potential source of disease recurrence. Suppressive hormonal treatment with L-thyroxin after partial thyroid resection often does not prevent re-growth in the thyroid remnant. It is also well known that re-operation greatly increases the risk of injury to the parathyroid glands and recurrent laryngeal nerves. TT is considered extremely effective in patients with Grave's disease with progressive exophthalmos. However, for most surgeons, benign thyroid disease does not represent an indication for TT. Most surgeons prefer to perform subtotal thyroidectomy for such benign condition as multinodular goiter and hyperthyroidism even when they have to leave grossly abnormal tissue in the neck owing to the complication rate associated with this procedure. With the emergence of surgical endocrinology as superspeciality, total thyroidectomy has been particularly relevant in endemic regions where patients present with a long standing large nodular goiter with virtually no normal thyroid tissue. Total thyroidectomy has therefore, been considered a valuable option for treating benign thyroid disorders.*

INTRODUCTION

Disorders of thyroid gland affect 3% to 5% of population; these occur as the 2nd most common endocrine disease next to diabetes mellitus. Benign goiter is endemic in the northern belt of India, Germany accounts for 90,000 to 1, 00, 000 operations per year. Goiter surgery is one of the most common surgical procedures.

In recent years surgeons are performing an increasing proportion of total thyroidectomy in comparison to other types of thyroid operations. In Australia, the proportion of total thyroidectomy in relation to all thyroid operations ranges from 10.5% in a survey of general surgeons upto 55% elsewhere by surgical endocrinologists.[2]

With the advent of anesthesia, antisepsis and appropriate surgical instruments, and due to the pioneering work of Kocher, Billroth, Halsted, Lahey and De Quervain and others — thyroid surgery has become a safe treatment modality with an acceptable low risk profile, offering many patients good prospects of cure. Now a days, death from thyroid surgery is rare. The incidence of the most common long — term complications, recurrent laryngeal nerve palsy and hypoparathyroidism, is genuinely low. The reported complication rates in the literature vary ranging from 0% for primary surgery to 20% for reoperative surgery for permanent RLN injury and from 0.5% to 11.0% for permanent hypoparathyroidism.'

ANATOMICAL CONSIDERATIONS

The clarification of the anatomy and blood supply of the parathyroid gland by Haisted and Evans, the development of appropriate instruments by Kocher, Spencer Wells, Halsted, Dunhill and others and the technique of ultraligation of vs. of the thyroid by Halsted laid the foundations of successful thyroid surgery (Figure 1). Although these developments occurred about a century ago, some of the advances were not well applied by all surgeons operating on the thyroid during the middle 50 yrs of the century. The improvement in results attributed to best modern practice has accompanied the recognition of endocrine surgery as a superspeciality and the leadership of the members of the International Association of Endocrine Surgeon.[2]

CONTROVERSIES IN SURGICAL MANAGEMENT

Controversy exists about the type of thyroid surgery with extent of resection, the operative frequency of recurrent goiter and Grave's disease, the experience of the surgeon, the hospital operative volume on post operative complication rates. Controversy also exists regarding: (i) Can total and subtotal thyroidectomies be performed safely without loss of parathyroid function or injury to recurrent laryngeal nerves? (ii) What is the minimum surgery to be performed Thyrotoxicosis?



Fig.1: Twigs of inferior thyroid artery specific for thyroid gland should should be ligated and not those for parathyriods. This will maintainblood supply of parathyroid glands.

TOTAL V/S SUBTOTAL THYROIDECTOMY

Total Thyroidectomy has been accepted for primary management of ***thyroid cancer***. Because of the reportedly high incidence of complication associated with this procedure, it was never accepted as a viable treatment option for management of benign thyroid disorders. With the advent of a safe surgical technique and recognition of pitfalls of subtotal thyroidectomy, total thyroidectomy is being performed increasingly for benign thyroid disorders. The recent literature shows comparable complication rates with total and subtotal thyroidectomy. The indications for total thyroidectomy for benign thyroid disorders are being evolved. Total thyroidectomy is a preferred option, as reoperation for recurrent goiter is associated with increased morbidity. The operation is indicated in all patients with toxic/ non toxic multinodular goiter and Grave's disease with ophthalmopathy. It is particularly relevant in endemic regions where patients present with a long standing, large nodular goiter with virtually no normal thyroid tissue. reoperation for recurrent goiter in such a setting often deed to with distressing complications.[3]

Total thyroidectomy can be done safely with a low rate of complications. The incidence of permanent recurrent laryngeal nerve (RLN) injury was 0.7% in our experience and should be less than 1%. The rate of permanent hyperparathyroidism was 2.2%, although other studies have shown that an incidence of less than 1% is achievable.2 Studies have been reported

Correspondence: **Dr. A.N. Sinha,** *C-3,3173, Vasant Kunj, New Delhi-70, (M) 9818266270, e-mail: aloknathsinha@yahoo.com*

with considerable higher complication rates and the complication rate appears to be higher when surgery is done by surgeons other than those with special endocrine surgical interest, expertise and experience.2 Several other studies have established that there is no significant difference in complication rates for total and subtotal thyroidectomy.

In pateints with ***benign multinodular goiter***, there is increasing recognition that total thyroidectomy is appropriate when there is significant nodular disease involving both lobes. When one lobe is relatively normal the case for total thyroidectomy is not as strong although in the longer term approx 12% of such patients develop recurrence and half of them require surgery. Subtotal thyroidectomy in a full-fledged MNG is a less satisfactory procedure with a long-term recurrence rate as high as 45% that is not influenced by thyroxine therapy.[2] Moreover there are increased risks of reoperative surgery when both sides have been previously operated. In another study the recurrence is 42% after 30 yrs follow up in a group of MNG patients.[5]

Three different *modalities* are commonly needed for treatment of ***hyperthyroidism*** i.e. *antithyroid drugs, radioiodine and surgery*. The treatment policy varies within and between different countries but surgery is worldwide less common and considered as second line of treatment. The first line is often antithyroid drugs or 131, both treatments carrying low complication risks. Surgical treatment is often performed in patients with large goiter or severe hyperthyroidism, for example with infiltrative ophthalmopathy. The relapse rate is high with antithyroid drugs — 40% to 60%. Patients with high initial total serum triodothyronine (T3) levels may have an increased risk for developing or worsening of ophthalmopathy when treated with 131 as compared to those treated by surgery. Treatment with prednisolone at the time of I treatment can decrease the risk for deterioration of the ophthalmopathy.[4]

Grave's disease is a controversial pathology with regard to the extent of appropriate surgery. Subtotal thyroidectomy has been the traditional operation for a century and has been performed for Grave's disease by surgeons who otherwise advocate total thyroidectomy for cancer and benign MNG. It has been well demonstrated in the literature that there is no reliable method of assessing the amount of thyroid to be preserved in individual cases treated by subtotal thyroidectomy. The thyroid remnants may hypertrophy in the future, causing pressure effects or suspicion of neoplasia if the remnant recurrence is nodular. There is an unavoidable risk of recurrent thyrotoxicosis which may require radioactive iodine treatment the patient may have wished to avoid when opting for surgical treatment initially and of hypothyroidism. Hypothyroidism may develop insidiously and remain unrecognized and untreated. It increases in frequency with time after surgery and is as high as 70% after 15 years. If it is accepted that the risks to RLN and parathyroid function are equivalent for subtotal and total thyroidectomy why not cure the disease with total thyroidectomy? It achieves predictable hypothyroidism which is controlled by immediate thyroxine replacement and monitoring.2

The role of total thyroidectomy specifically for patients with ophthalmopathy is an area of great interest. There is some evidence that thyrotropin receptor antibody levels tend to normalize after total thyroidectomy (Table I). Grave's ophthamopathy is a manifestation of the autoimmune disease and is not related to the level of thyroid hormones, so surgical treatment may be of more benefit than radioactive iodine therapy. There are anecdotal reports and some preliminary data indicating that total thyroidectomy, and not subtotal Thyroidectomy may be preferred for Grave's disease, particularly when there is ophthalmopathy.[2] (Figure 2)

Table1: *Laboratory Data After Surgery*

Group	N.	Tg. Detected	Tg Ab. (+)	TpoAb. (+)	Tr Ab. (+)
Total Thyroidectomy	50	1	0	8	2
Subtotal Thyroidectomy	50	21	21	32	6

Tg - Thyroglobulin; Tg Ab - Thyroglobulin antibody; TpoAb- Thyroid Peroxidase antibody; Tr.Ab - Thyrotropin Receptor Antibody.

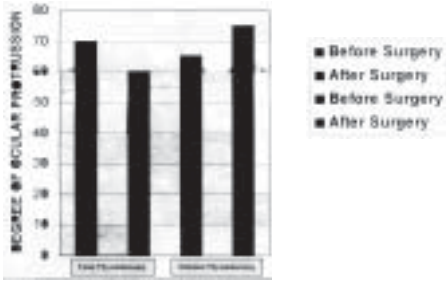


Fig. 2 changes of ocular protrusion after total and subtotal thyroidectomy in patients with thyroid associated ophthalmopathy

The technique of safe hemithyroidectomy or total thyroidectomy involves "apsuIar dissection". This was described in essence by Halsted and redefined more recently by Norman Thomson, Thomas Reeve. In the technique, the main inferior thyroid artery is not ligated, the parathyroid glands and their blood supply are preserved. The RLN is constantly sought during the dissection but is not always seen; in fact, it is ideally left undisturbed underneath a layer of fascia as it lies on the oesophagus or in the tracheooesophageal groove. It is not necessary to visualize and dissect the nerve in all cases but it would be advisable in situations where the nerve is at greater than usual risk of injury such as during reoperative surgery, large nodular goiters (especially, if they extend retrosternally) and cancers with local invasion or lymphnode metastases.2

Total thyroidectomy is a relatively safe operation with a low complication rate when performed by surgeons trained in "Capsular dissection" method. The lowest complication rate is appropriate for benign unilateral thyroid disease. There appears to be no significant difference in the rate of complications (haemorrhage, nerve injury, hypoparathroidism) for total and subtotal thyroidectomy.2

We have little enthusiasm fobtotal thyrodec because residual thyroid treatment may cause future recurrence, which is difficult to treat. It is preferred to thoroughly excise the pathology and eliminate the possibility of future recurrence and the need for reoperative surgery by performing total thyroidectomy for most patients (non toxic or toxic) involving both lobes or Grave's disease. Evidence suggests that the incidence of unsuspected malignancy with multinodular goiter is 63% and of these patients one — third presented with Grave' disease. Total thyroidectomy offers a complete initial treatment of these cases, taking care of multicentricity and future transformation into poorly differentiated carcinoma and avoiding the need for completion thyroidcectomy.3

Surgery thus has a definite role in the management of patients with hyperthyroidism, although other therapeutic modalities are available. As clinicians caring for patients, valid and reliable evidence may be obtained about the efficacy and safety of each of treatment options so that the information can be used during the decision-making process. Patients' value and preference must also be considered. Endocrine surgeons will continue to offer their expertise to both endocrinologists and patients to assist in optimizing care of thyrotoxic patients.

REFERENCES

1. ***Thomuch Oliver, Machens Andreas, Sekulla Carsten et a!.*** *Multivariate analysis of Risk Factors for Postoperative complications in Benign Goiter Surgery: Prospective Multicenter study in Germany. World Journal Surg 2000; 24:1335-41.*
2. ***Gough I R, Wilkinson David.*** *Total Thyroidectomy for Management of Thyroid Disease, World Journal Surg 2000; 24:962-65.*
3. ***Mishra A, Agarwal Amit, Agarwal Gaurav, Mishra S K.*** *Total Thyroidectomy for Benign Thyroid disorders in an Endocrine region. World Journal Surg 2001; 24:307-10.*
4. ***Petra Wegra-Kjellman, Jan Zedemin, Leif Tallstedt, Frank Traisk, Goran Lundel! and Goran Wallin.*** *Surgical Treatment of Hyperthyroidism: A ten year experience. Thyroid 2001; 11(2):187-92.*
5. ***Rojdmark J, Jarhult J: High Long*** *— term recurrence rate after subtotal thyroidectomy for nodular goiter. Eur. J. Surg. 1995; 161:725-27.*

AVIAN INFLUENZA: THE POTENTIAL PANDEMIC DANGER

Govind Chandra Sahoo

Department of Otolarryngology, Rajah Muthiah Medical College & Hospital,Annamalai Nagar
Annamalai University, Chidambaram (T.N.) – 608 002. India

Abstract: *Bird flu or Avian influenza is spreading like wild fire from Cambodia to Croatia and Romania to Russia including India in 2006, engulfing more than seventeen countries. The genetics of the H5N1 virus plays an important role in the virulence and the impending threat of the influenza pandemic for which more than 150 million chickens have died or culled in Hong Kong only. Recent deaths in migrating birds also have been reported from China, Russia and Mongolia including 70 deaths in human beings.*

The first epidemic of avian influenza was reported from Italy more than 100 years ago. But in recent year there has been regular outbreak of influenza which was first detected in 1996 in a domestic goose from the Guangdong province of China followed by the first infection of H5N1 virus both in poultry and humans in the 1997 epidemic of Hong Kong[1]. There have been regular outbreaks, since 1996 to 2006 in Indonesia, Thailand, Vietnam, Turkey, Laos, Malaysia, Ukraine, Iraq, Egypt, Nigeria, India and European countries etc.,[2] Tigers and leopards of a zoo also died who had fed on fresh bird carcasses infected by H5N1. The first great influence pandemic of last century in 1918-19 affected more than 25% of the total population world wide with an estimated 40-50 million death within a single year without any warning and causing unprecedented illness with maximum of death In the young and healthy persons in the age group of 15 to 35 years. The subsequent pandemics in 1957 and 1968 including Spanish flu spreading rapidly also killed millions of people. The influenza viruses are highly resilient, which can survive in contaminated manure for at least three months and one gram of H5N1 manure can contain enough viral particles to infect one million birds. The virus can survive potentially for indefinite period in frozen materials[3,4]. Out of the types of influenza virus(A,B,C), the avian influenza virus is the subtype of Type A influenza virus. Type A influenza virus is subtyped on the basis of surface of Glycoproteins, hemagglutin (HA) and neuraminidase (NA). There are 16 known HA subtypes and 9 known NA subtypes of influenza viruses which can recombined in permutation and combination to create novel combination of influenza[5]. The Eight separate gene segment composing the influenza A virus has the capacity of re-assortment of these gene segments to develop a novel virus leading to pandemics due to the lack of immunity. This major antigenic change is known as antigenic shift in contrast to the typical seasonal influenza virus exhibiting frequent point mutation, leading to more gradual shifts in their genome, which is known as antigenic drift, the reason for which new influenza vaccines must be prepared every year[6,7]. The influenza viruses are RNA viruses, lacking effective genetic proof-reading and minor mutations can cause genetic drift with a grave concern of increasing possibility of a human pandemic by H5N1 virus.

The main role of transmission of avian influenza on humans is via inhalation of respiratory droplets from coughing and sneezing. As the humans are very rarely exposed to avian influenza virus, so there is very minimal immunity in the general population which could become an epidemic or pandemic very rapidly. Of the three pre-requisites to start an influenza pandemic VIZ..

(a) Emergence of novel susceptible virus
(b) Ability of the new virus to replicate and cause disease in humans.
(c) New virus transmitting efficiently from human to human.

The first two of the above prerequisites have already been met by the current H5N1 outbreaks in Asian birds. All the conditions for a pandemic will be fulfilled if the last criteria of human to human transmission is met by the H5N1 virus, the potential candidate for the next pandemic. Although no one can predict with certainty the timing of pandemic, but there is a concern that it is imminent and there is a great possibility that it would occur sooner than latter beginning from Asia. Other than the morbidity and mortality the direct economic loss in the current H5N1 outbreaks to the affected countries were to the tune of 8 to 10 billion US dollar. It is estimated that the next pandemic may cause more than one billion case, with 2-7 million deaths resulting on sever strain on the health and other essential services with massive social, political and economic disruption. A modest pandemic lasting over one year may result in a loss of 150 to 200 billion dollars on GDP which is equivalent to 3% of GDP of Asia. The outbreaks of SARS in early 2003 spanning 30 countries in six continents which is the first new disease of this millennium on the global health stage, caused an estimated loss of 30 to 150 billion dollars to the affected countries with a significant mortality rate of around 11%. Individuals at high risk for developing influenza complications like primary viral pneumonia, multi organ failure etc., are usually children, old people, pregnant women, immuno compromised patients etc.,

Important measures for the pandemic preparation as per the guide lines of the center for disease control and prevention are based on surveillance for avian influenza infection control of the poultry and animals including healthy poultry and infection control on medical settings. The drugs which can be used both for the prophylaxis and therapeutic are Zanomivir (Relanza) and Oseltamivir (Tamiflu). At present there is no specific vaccine to prevent avian influenza in human beings, although clinical trials have started. The WHO influenza surveillance network and collaborating centers have developed recombinant H5N1 prototype vaccine strain according to the requirement of major national and international pharmaceutical licensing agencies. However, incase of a pandemic, the vaccine strain for production of the vaccine for commercial availabilities will take 3-6 months and it is highly unlikely that any developing country will have access to this vaccine. WHO is planning to stock pile 1 to 3

Correspondence: **Govind Chandra Sahoo,** B-4. R.S.A.Housing Complex,Annamalai Nagar – 608 002,Chidambaram, Tamilnadu., India

million courses of anti avian influenza drugs which could be used as a pre-emptive measures anywhere in the world for the impending pandemic.

There is global concern about the spread of avian influenza and the possibility of pandemic for which international organizations like WHO, FAO and international organization for animal health are exchanging information, advising national governments and assisting in resource mobilization. The formulation of a national pandemic preparedness is fundamental to harness the national resources and activate the systems in health and veterinary sectors to respond rapidly to the threat of pandemic. The concern of environmentalist about the want on killing of birds and animals though normally justified but past experience shows that prompt culling of birds is the only effective way to control the epidemic before it is pandemic. The role of WHO is very significant to avoid an avian influenza pandemic if the guide lines are followed strictly. WHO can play a major role on averting a pandemic by providing technical support develop guidelines, assist outbreak investigation, provide diagnostic reagents, stock pile anti-virals, develop vaccine strain and support research [8]. The present scenario in India is very vulnerable and precarious in the event of a pandemic as the number of immunocompromised people are growing due to HIV / AIDS [9]. With a growing number of immunocompromised people in addition to diabetes, malnourishment, geriatric and pediatric age group, an infectious disease like avian influenza will have devastating effect and growing chance of achieving its pandeminion vision is very much possible.

The lack of ready and repeatable transmissibility of the avian influenza between human is one of the only factors preventing a potential H5N1 pandemic in human beings. However, there is a high possibility of further mutation in the H5N1 virus and or intermixing with human influenza virus, which may remove this last hurdle of crossing the species barrier, thereby fulfilling all the condition for a H5N1 virus, the potential candidate for the next pandemic[10].

REFERENCES

1. *World Health Organization; H5N1 avian influenza http://www.who.int/entity/csr/disease/avian_influenza/timeline_3_03.pdf.*
2. *Centre for disease control and prevention. Avian influenza: current situation. http://www.cdc.gov/flu/avian/outbreaks/current.html*
3. *U.S. Department of labor occupational safety and Health Administration. Guidance for protecting workers against avian flu http://www.osha.gov/dsg/guidance/avian_flu.html.*
4. *Bio-security for the Birds, Avian influenza (AI). http://www.usda.gov/agency/oc/design/test/bird biosecurity/hpai.html*
5. *Centre for Disease control and prevention. Key facts about avian influenza (Bird Flu) and Avian influenza A (H5N1) virus. http://www.cde.gov/flu/avian/gen_info/facts.html*
6. *Centre for Disease control and prevention. Transmission of influenza A viruses between animals and people. http://www.cdc.gov/flu/avian/gen_info/transmission.html.*
7. *Centres for Diseases control and prevention. Prevention and control of influenza: Recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 2005; 54:(RR-8).*
8. *World Health Organization Guidelines on Avian influenza. http://www.who.int/csr/diseases/avian_influenza/en/*
9. *Avian influenza: Preparing for a pandemic. A cleon, S.Galwankar, JAPI, Vol.54, 2006.*
10. *Centre for Disease control and prevention. Question and answers about avian influenza (bird flu) and avian influenza A(H5N1) virus available online at: http://www.cdc.gov/flu/avian/gen-info/qa.html.*

The **IDEAL** choice in
ESBL producing pathogens

The First BID Nitrofurantoin

NIFTRAN™ 100mg

The USFDA approved NDDS Nitrofurantoin

AUA Annual Meeting, May 2008

Nitrofurantoin is an inexpensive, effective and acceptable treatment for uncomplicated urinary tract infections and should now be **considered first line treatment**

The **susceptibility of ESBL** producers to imepenem, **nitrofurantoin** and amikacin was found to be 100%, **89%** and 86% respectively.

Dosage

Acute Cystitis : 1 capsule bid

Long-term suppressive therapy : 1 capsule at bed time

RANBAXY UROLOGY SPECIALITY

BRIEF COMMUNICATION

JIMSA April-June 2009 Vol. 22 No. 2

EFFICACY OF DILTIAZEM IN THE TREATMENT OF CHRONIC ANAL FISSURE: AN OVERVIEW

B. Medhi, A. Prakash, L. Kaman

Departments of Clinical Pharmacology & General Surgery,
Postgraduate Institute of Medical education and Research, Chandigarh 160012, India.

Abstract: *Anal fissure is a common medical problem which effect both sexes equally. Until recently the gold standard treatment for chronic anal fissure was surgical lateral internal spincterotomy but recently chemical spinteractomy has become popular in the surgical practice. In the treatment of chronic anal fissure Ca^{2+} channel blockers play an important role which was proved to be efficacious in various clinical trials. Mechanism involved in these drugs supposed to be due to vasodilatation at the fissure site and hence it increases local blood circulation leads to enhance healing. Studies showed that healing occurs with infrequent headache and hypotension, with diltiazem treatment compared to glyceryl trinitrate therapy.*

INTRODUCTION

Anal fissure (AF) is traumatically induced longitudinal tear in the squamous epithelium of anal canal. It was originally described by Recamier in 1829. Studies suggested that one in five person developed anal fissure during there lifetime and occurred in all most all age group. Most commonly identified AF in clinical setup is posterior about 90% where as anterior it is 10% and less than 1% in both position[1]. Anal fissure is mainly characterized by the severe pain during and after defecation and bright red rectal bleeding which may last for an hour. Acute anal fissure (AAF) resolves spontaneously or with conservative treatment in 10 to 14 days but complete tear to may takes 6 – 8 weeks. The chronic and to assure (CAF) of more than 6 weeks. Needs surgical intervention. When fissure becomes large, deep and chronic they are called anal ulcers.[3] When a fissure is located in atypical locations or multiple and fail to heal following treatment, other conditions such as inflammatory bowel disease, neoplasm, leukemia, syphilis, tuberculosis, HIV has to be ruled out.[2,3,4]

TREATMENT STRATEGIES

In the treatment of AF, aim of treatment is to ***avoid constipation***, soften stool and reduce anal sphincter spasm. Bulk is added to diet and stool softener is prescribed. Increased fiber in the diet softens and bulks the stool. Regarding ***pharmacotherapy*** different Ca^{2+} channel blockers, vasodilators and cholinomimetics were tried.

ROLE OF DILTIAZEM

A calcium dependent mechanism is required to maintain internal sphincter muscle tone. ***Calcium channel blocker*** [CCB's]causes relaxation of gastrointestinal smooth muscle[1,2] ***oral diltiazem*** has been shown to reduce the resting anal pressure.[3] Carapeti EA et al, explored the possible use of calcium channel blockers and cholinomimetic to lower anal sphincter pressure. Both oral diltiazem and topical gel containing varying concentration was tested on healthy adult volunteers in order to determine dose response characteristics of these agents. They found that single dose of 60mg diltiazem lowered maximum resting pressure [MRP] by a mean of 21%. Once daily diltiazem produced clinically insignificant effect but twice daily regimen reduced anal pressure by mean of 17%. Study used 2% diltiazem gel produced a maximal 28% reduction, the effect lasting 3-5 hrs, this study also reported potential low side effect alternative to topical nitrates for the treatment of anal fissure.[14] Carapeti EA et al, conducted study on patients with chronic anal fissure. Patients were treated with 2% diltiazem gel 3 times daily for 8 weeks. Fissure healed in 67% of patients. There was no significant difference in maximum resting sphincter pressure between responders and non-responders. There was significant decrease in pain score after treatment with diltiazem (p=0.002). MRP was significantly lowered [p=0.001]. No headache or side effects were reported.[5] Jonas M et al, assessed the effectiveness of oral and topical diltiazem in healing chronic anal fissure. MRP fell by 15% and 23% in oral and topical group. Fissure healing was 38% in oral and 65% in topical treated group by 8 weeks. Oral diltiazem caused side effects such as rash, headache, nausea, vomiting, decreased smell and taste, while topically treated groups showed no side effects.[6] Knight JS et al, studied 2% topical diltiazem in 71 patients. 75% experienced healing in 2-3 months and in further 2 months 88% experienced healing. Minor side effects were perianal dermatitis, 1 patient had headache, and 6 of 7 patients with recurrent fissure were treated successfully by repeat chemical sphincterotomy.[7] Jonas M et al, did a study the efficacy of diltiazem for fissures that failed to heal with glyceryl trinitrate [GTN]. There were 39 patients with chronic anal fissure received GTN therapy of which 27 patients completed course and 12 patients discontinued. Out of 27 patients who completed GTN course and presented with resistant chronic anal fissure, 44% were healed with topical diltiazem while 56% of the patients the fissure persisted and had to undergo surgical sphincterotomy. In his trial 12 patients discontinued GTN therapy and 58% were healed with topical diltiazem.[8] Dasgupta D et al, also evaluated the treatment of patients with chronic anal fissure with diltiazem gel. Investigators found that fissure healed in 48% including 75% of the patients who previously failed to heal with GTN therapy. There were no recurrence and no adverse effects[9]. Griffin et al, also studied the role of topical diltiazem in the treatment of chronic anal fissure that have failed with GTN therapy. He concluded that topical 2% diltiazem is an effective and safe treatment for patients who have failed topical 0.2% GTN therapy. Sphincterotomy can be avoided in 70% of patients with chronic anal

Correspondence: Dr. Bikash Medhi, *Department of Clinical Pharmacology, Ph: (o)91-172- 2755250,(M) 919815409652 E-mail: drbikashus@yahoo.com*

Table: *Reported efficacy of Diltiazem in chronic anal tissue in different series*

Author (Year)	Patients (Duration)	% healing	Remarks
Carapeti EA et al[14] (1999)	30	Oral -21% Topical-28% Placebo Bethanicol	Effective
Carapeti EA et al[5] (2000)	30	67%	Comparable .
Jonas M et al[6] (2001)	50	52% (topical diltiazem)	Effectious than control
Knight JS et al[7] (2001)	71	83%	Effective
Jonas M et al[8] (2002)	39	48%	Effective in GTN failure patients
Dasgupta et al[9] (2001)	23	48%	Effective
Griffin et al[10] (2002)	47	48%	Effective
Kocher HM et al[11] (2002)	60	41%	Comparable
Bleleki et al[12] (2002)	43	86%	Comparable
Nash GF et al[13] (2006)	112	41%	Safe for long term use

fissure.[10]

Kocher HM et al, conducted a randomized control trial assessing adverse effects of GTN and diltiazem in treatment of chronic anal fissure. More headache occurred with GTN therapy than diltiazem [p=0.01]. There was no significant difference in healing and symptomatic improvement rates between the two groups.[11] Other study also showed similar result like Bleleki et al, conducted prospective randomized trial of diltiazem and GTN therapy in treatment of chronic anal fissure and this study concluded that diltiazem and GTN therapy are equally effective in healing anal fissure. But diltiazem reported in few adverse effects.[12] Study evaluated long-term outcome of diltiazem in chronic anal fissure. Total one hundred and twelve patients were treated for 6-week with 2% diltiazem cream for twice-daily topical application. The success rate and satisfaction of topical diltiazem were each over two thirds. Nearly 80% of patients reported no adverse effects, and it seems that those complaints attributed to diltiazem rarely led to reduced compliance. After completion of diltiazem therapy for fissure, 59% of patients required further treatment (medical and/or surgical) over the average 2-year period of follow-up[13].

In conclusion several trials have already conducted to proved safety and efficacy of topical diltiazem in chronic anal fissure. So diltiazem may emerged as main therapeutic agent in the management of chronic anal fissure in near future but surgery should be preferred to the patients with relapse or in patients not responding to diltiazem therapy.

REFERENCES

1. *Kodner IJ, Fry RD, Fleshman JW, Birnbaum EH and Read TE. Colon rectum and anus.In: Schwartz's Principles of Surgery. 7th edi, Mc Graw Hill Health Proffessions division-USA. 1999. pp 1265.*
2. *Nepomuceno OR, O'Grady JF, Eisenberg SW, Bacon HE. Tuberculosis of the anal canal: report of a case. Dis Colon Rectum 1971; 14(4):313-316.*
3. *Chapel TA, Prasad P, Chapel J, Lekas N. Extragenital syphilitic chancres. J Am Acad Dermatol 1985; 13(4):582-584.*
4. *Sweeney JL, Ritchie JK, Nicholls RJ. Anal fissure in Crohn's disease. Br J Surg 1988; 75(1):56-57.*
5. *EA Carapeti, M A Kamm, B K Evans, R K S Philips. Topical diltiazem and bethanechol decrease anal sphincter pressure and heal anal fissures without side effects. Dis Colon Rectum 2000; 43:1359-1362.*
6. *Jonas M, Neal KR, Abercrombie JF, Scholefield JH. A randomized trial of oral vs. topical diltiazem for chronic anal fissure. Dis Colon Rectum 2001; 44:1074-1078.*
7. *Knight JS, M Birks, R Farouk. Topical Diltiazem ointment in treatment of chronic anal fissure. Br J Surg 2001; 88:553-556.*
8. *Jonas M, W Speake, John H Scholefield. Diltiazem heals Glyceryl tri-nitrate resistant chronic anal fissures. Dis Colon Rectum 2002; 45:1091-1095.*
9. *Dasgupta R, Franklin I, Pitt J, Dowson P D. Successful treatment of chronic anal fissure with diltiazem gel. Colorectal Diseases 2002; 4:20-22.*
10. *Griffin N, Acheson AG, Jones M and Scholefield J H. The role of topical diltiazem in the treatment of chronic anal fissures that have failed glyceryl trinitrate therapy. Colorectal Diseases 2002; 4:430-435.*
11. *Kocher H M, Steward M, A, Leather AJM, Cullen PT. Randomized clinical trial assessing the side effects of glyceryl trinitrate and diltiazem in the treatment of chronic anal fissure. British Journal of Surgery 2002:89:413-417.*
12. *Bleleki K, Kolodzlejczak M. A prospective randomized trial of diltiazem and glyceril trinitrate ointment in the treatment of chronic anal fissure. Colorectal Diseases2003; 5:256-257.*
13. *Nash GF, Kapoor K, Saeb-Parsy K, Kunanadam T, Dawson PM. The long-term results of diltiazem treatment for anal fissure. Int J Clin Pract 2006; 60(11):1411-1413.*
14. *EA Carapeti, M A Kamm,B K Evans, R K S Philips. Topical diltiazem and bethanechol decrease anal sphincter pressure without side effects. Gut 1999; 45:719-722.*

LITERATURE REVIEW

Intensive Blood Glucose Control and Vascular Outcomes in Patients with Type 2 Diabetes

The ADVANCE Collaborative Group

Background *In patients with type 2 diabetes, the effects of intensive glucose control on vascular outcomes remain uncertain.*

Methods *We randomly assigned 11,140 patients with type 2 diabetes to undergo either standard glucose control or intensive glucose control, defined as the use of gliclazide (modified release) plus other drugs as required to achieve a glycated hemoglobin value of 6.5% or less. Primary end points were composites of major macrovascular events (death from cardiovascular causes, nonfatal myocardial infarction, or nonfatal stroke) and major microvascular events (new or worsening nephropathy or retinopathy), assessed both jointly and separately.* Results *After a median of 5 years of follow-up, the mean glycated hemoglobin level was lower in the intensive-control group (6.5%) than in the standard-control group (7.3%). Intensive control reduced the incidence of combined major macrovascular and microvascular events (18.1%, vs. 20.0% with standard control; hazard ratio, 0.90; 95% confidence interval [CI], 0.82 to 0.98; P=0.01), as well as that of major microvascular events (9.4% vs. 10.9%; hazard ratio, 0.86; 95% CI, 0.77 to 0.97; P=0.01), primarily because of a reduction in the incidence of nephropathy (4.1% vs. 5.2%; hazard ratio, 0.79; 95% CI, 0.66 to 0.93; P=0.006), with no significant effect on retinopathy (P=0.50). There were no significant effects of the type of glucose control on major macrovascular events (hazard ratio with intensive control, 0.94; 95% CI, 0.84 to 1.06; P=0.32), death from cardiovascular causes (hazard ratio with intensive control, 0.88; 95% CI, 0.74 to 1.04; P=0.12), or death from any cause (hazard ratio with intensive control, 0.93; 95% CI, 0.83 to 1.06; P=0.28). Severe hypoglycemia, although uncommon, was more common in the intensive-control group (2.7%, vs. 1.5% in the standard-control group; hazard ratio, 1.86; 95% CI, 1.42 to 2.40; P<0.001).* Conclusions *A strategy of intensive glucose control, involving gliclazide (modified release) and other drugs as required, that lowered the glycated hemoglobin value to 6.5% yielded a 10% relative reduction in the combined outcome of major macrovascular and microvascular events, primarily as a consequence of a 21% relative reduction in nephropathy. (ClinicalTrials.gov number, NCT00145925 [ClinicalTrials.gov] .)*

IMMUNO-SUPPRESSIVE THERAPY IN GLOMERULONEPHRITIS

P.J. Mahanta, S.C. Tiwari

Department of Nephrology AIIMS, Ansari Nagar, New Delhi-110029. India.

INTRODUCTION

Self tolerance means a state of absence of immune responsiveness to a self antigen. Self tolerance occurs due to two phenomena. *First* central tolerance is the process of the deletion of self reactive lymphocyte during maturation in bone marrow and thymus. *Second,* peripheral deletion, is the process of sequestration of self reactive T. cells that have escaped deletion in the thymus.

Auto-immune disease is believed to results from the bypass of at least one of the self tolerance mechanisms. The interaction between immunologic, genetic, environmental and even microbial factors play role in the initiation of this hazardous autoimmune response. Classsically, the immune system has been divided into two classes, humoral immunity, mediated by soluble antibody and cellular immunity, mediated by lymphocyte. Immune complex mediated glomerulonephritis is a classis humkoral response, whereas alteration in cellular response may responsible for the pathogenesis of minimal change disease. But there is evidence that in some diseases like ANCA associated glomerulonephitis, both d humoral and cell mediated immune response are involved.

Immuno-supressive medications, which are used for the treatment of various glomerulonephritis, may act by inhibiting or modifying different steps of immune response. It is important to remember that all immuno-suppressive medications are associated with serious adverse effect effect like, increased risk of infection and neoplasia. Therefore, in treating individual patient with glomerulonephritis, the risk versus benefits of the immunosuppressive medications must be weighed.

IMMUNOSUPPESSIVE AGENTS

Glucocorticoids; These have been used for may years as both immunosuppressive and anti-inflammatory agents. They suppress both cell mediated immunity, by inhibiting genes that code for important cytokines like Interleukin 1 through 6 and 8 and Interferon a, and humoral immunity, by diminishing B cell clonal expansion and antibody synthesis.

Currently, they are the most commonly used and are perhaps the most effective immunosuppressive medications for may only of the glomerular diseases either along or in combination with other drugs the cyclophophamide, azathioprine ormycophenolate. They are effective in minimal change disease(MCD), focal segmental glomerulosclerosis (FSGS), membranous nephropathy (MGN), induction as well as maintenance therapy of lupusnephritis (LN) and rapidly progressive glomeulonephritis (RPGN)

As these agents are associated with many adverse effects, close monitoring is crucial. These adverse effects include an increase susceptibility to infection, impaired glucose tolerance, sodium retention, hypertension, osteoporosis, cataract, cushigoid appearance etc.

Azathiopine; has been used as an immunosuppressive agent since 1960. It is pro-drug, which is converted in the body to its active metabolite-6 mercaptopuine, which they act by inhibiting the formation of phosphoribosy 1 pyrophosphate, an intermediate in purine metabolism. The drug is now commonly used to treat lupus nephritis, lg A nephropathy, and severe proliferative glomerulonephritis such as ANCA associated glomerulonehritis as maintenance therapy, after induction with steroids and cyclophosphamide. It is used at the dose of 2 mg/kg/day. Azathioprine has generalble adverse effects. It causes acute myelo-suppression resulting in leucopenia, megaloblastic anemia, and thrombocytopenia, due to the incorporation of azathiopine derived 6-thiogunanine nucleotides into DNA. Other side effect includes nausea, vomiting, diarrhea and hepatotoxicity. Moreover, concomitant therapy with allopurino9l, which inhibits its metabolism, potentiates toxicity. Although previously believed to be safe in borne to pregnancy, recently few reports of leucopenia and thrombocytopenia in neonates mothers azathioprine during pregenancy.

Cyclophosphamide is a prodrug it is converted in the liver to the active metabolite 4-hydroxy cyclophosphamide and phosphoramide mustard. Then these bind to DNA and inhibit cell proliferation and results in dose dependent neutopenia and lymphopenia, with reduction of both B cell and CD4+ and CD8+ T cell.

Cyclophosphamide has been used to treat autoimmune glomerular diseases, both as an intravenous pulse and an oral form. For many years, it has been line D therapy for proliferative lupus nephritis and it remains the drug of choice for most patients with severe crescentic rapidly progressive glomerulonephritis, whether related to anti GBM antibody jor ANCA associated disease. It is also used to treat steroid resistant minimal change disease, FSSGS, Ig A nephropathy and membranous nephoroathy. As it is associated with significant adverse effects. The newer immunosuppressive like mycophenolate, is now becoming a popular alternative therapy. As induction thereapy, it is used as monthly intravenous pulse of 0.5 to 1.0 g/m2 or daily oral cyclophosphanide 2 mg/kg/for about 6 months.

Cyclophosphamids causes dose dependant bone marrow suppression, and may results in anemia, leucopenia or thrombocytopenia. The effect on leukocyte is most pronounced, the aim should be to keep the TLC above 3000/cmm and cumulative dose should not exceed more than 200 mg/kg. Infections are especially common in neutropenic patient, but major bacterial and fungal infections can occur even without neutropenia, especially in the presence of concomitant steroid therapy

Small amount of cyclophosphamide metabolite may convert to acrolein, which is toxic tobladder epithelium, leading to hemorrhagic cystitis. It is usually are with intravenous route and aggressive hydration may prevent this complication. It can leads to infertility in both male and females, and it should not be given to pregnant women, unless absolutely indicated, because of treatogenicity.

Other side effects are nausea, hair loss, mouth sores, hyponaturemia due to increased released of antidiuretic hormones and long terms risk of malignancy. Because of these major toxicities, a strategy that should be strongly considered is induction therapy with cyclophophemide, and then maintenance therapy with a less toxic drug like mycophenolate or azathioprine.

Correspondence: Prof. S. C. Tiwari, *Fax: 91-11-26588641,* ***e-mail:*** *sctiwari@hotmail.com*

Chlorambucil is also a nitrogen mustard alkylating agent. Its mechanism of action is similar to cyclophophamide. It has been used to treat steroid resistant minimal change disease, FSGS, and membransousnephropathy, as a part of alternating monthly regime with steroid. The commonly used dose is 0.12 mg/kg/day. Major adverse effects include bone marrow suppression, CNS toxicity hepato toxicity, infertility, strong teratogenicity and delayed malignancy.

Cyclosporin is a peptide derived from the soil fungusTolypocladium inflatum . In 1970, cyclosporine was introduced as an anti-rejection agent for organ transplantation. Cyclosporin is a calcineurin inhibitor that suppresses the immune response by down-regulating the transcription of various cytokine genes, mainly interleukin-2 (IL-2), which serves as the major activation factorfor T-cells in numerous immunological processes. A further effect of IL-2 inhibition is a reduction in B-cells activation and subsequent antibody production, IL-2 level increase during proteinuria, and normalize during remission in adults with the INS and in children with MCD or FSGS. This may be a part of a generalized disorder of cellular immunity that results in nephritic syndrome, rather than being causal of proteinuria. Cyclosporin may also have an anti-proteinuric action through its effect on glomerular permeability.

Cyclosporin has significant nephrotoxicity. Studies have demonstrated that at least in the acute situation these changes are functional and are promptly reversed following dose reduction or cessation of therapy. A current recommendation is the reduction of cyclosporine dose if serum creatinine increases by -30% above the patient's baseline value (even if this increase is within the normal range).

The clinical efficacy and safety of once a day and pre-prandial administration of cyclosporine in patients with idiopathic nephrotic syndrome is currently being investigated. Cyclosporin is an effective treatment for minimal change disease, FAGS, and membranous nephropathy. It is also effective in adjunct to steroid in class V (membranous) lupus nephritis. Recent Grade A evidence supports its use after a 12-week course of cytotoxic therapy, that has failed in steroid dependant or ssteroid resistant MCD and as a first-line option in MGN and FSGS either as monotherapy or in combination with corticosteroids. Cyclosporin therapy provides a sustained remission of proteinuria in a significant proportion of patients. The side-effect profile of cyclosporine is well known and predictable. Adherence to dosing guidelines plus regular patient monitoring will minimize the risk of nephrotoxicity.

In children, cyclosporine should be started at 100mg/m2/day and may be increased to 150 mg/m2/day. In adult, the dose is 2mg/kg/day and gradually increased at 2-week intervals until remission is achieved. The maximum dose is 5 mg/kg/day.

Treatment target include complete or partial remission of proteinuria, preservation of stable GFR (+-20% of pretreatment level), and a C0 cyclosporin level of 80-175 ng/ml. In the steroid-resistant or dependent cases following complete remission of proteinuria, cyclosporine should be continued for 1-4 years and then tapered gradually to lowest effective does.

Children continuing on long-term cyclosporine therapy should be regularly monitored, including a renal biopsy every 2-3 years to cheek for histological evidence of nephrotoxicity. Adults continuing on long-term cyclosporine therapy should also be regularly monitored, including a repeat renal biopsy at 12-24 months, especiallyifserum creatinine is -30% above the baseline level, and/or if the maintenance dose of cyclosporine require is -3.5mg/kg/day. In those, who are non –responsive to corticosteroids, cyclosporine should be given for at least 6 months before declaring it as treatment failure. Repeat biopsies should be carried out in a patient of FSGS with rapidly declining renal function, a marked disparity between an increasing serum creatinine and decreasing proteinuria and in patients receiving cyclosporine therapy beyond 2-3 years. The presence of increasing fibrosis and tubular interstitial disease should lead to a careful review of the case. It is often difficult to differentiate between drug toxicity and the natural history of the FSGS disease and hence the decision between lowering or discontinuing medication and/or adding adjunctive therapy remains a balance between the patient's clinical course/response or the observed histologic changes. Renal function (GFR) serves as a guide more than renal histology.

Tacrolimus is another calcineurin inhibitor, which binds to an immunophilin, FK binding protein -12. Then this complex interacts and inhibits calcineuin, resulting in actions similar to cyclosporine, Like cyclosporine, it is also nephrotoxic, and has similar toxicity profile. Recently tacolimus is also tried in few RCT as a monotherapy in membranous nephropathy, with promising results. The acrolimus target trough was 5 jto 8 ng/ml in that study.

Mycophenolate mofetil (MMF) , a prodrug of mycophenolic acid, is an immuno-suppressive agent that blocks purine biosynthesis by inhibiting the enzyme inosine monophosphate dehydrogenase (IMPDH). MMF inhibits T-and B-lymphocyte proliferation, induces apoptosis of activated T-lymphocytes, reduces synthesis of antibodies and may decrease the migration of inflammatory cells into glomeruli after antibody deposition. It also prevents glomerular injury, prevents progressive renal scarring and fibrosis by inhibiting mesangial cell proliferation. MMF does not induce nephrotoxicity and is relatively well tolerated.

MMF has been shown to be effective in several induction and maintenance studies of severe lupus nephritis. It is now being studied in FSGS, lg A nephropathy, membranous nephropathy, steroid resistant nephritic syndrome and as maintenance therapy for ANCA associated glomerulonphritis.

Most common adverse effects or MMF are gastrointestinal upset including nausea, abdominal pain and diarrhea, which can be reduced by dividing the total dose. Other adverse effects are leucopenia, which responds to dose reduction and increased risk of infection. Available data suggest that MMF is better than AZA in kidney transplantation and a good, but not necessarily a better alternative to AZA in lupus nephritis.

As an induction therapy in class IV lupus nephritis, most of the studies have used MMF at the dose of 2.0 – 3.0 g/day for 6 months or more, along with prednisone 1 mg/kg/day. As a maintenance therapy, the dose of MMF is 1.0 -2.0 g/day, along with alternate day prednisone (0.25 mg/day). For therapy in FSGS, Ig A nephropathy, membranous nephropathy, steroid resistant nephritic syndrome and as maintenance therapy for ANCA associated glomerulonaphritis, MMF has been used at the dose of 1.0 -2.0 g/day, along with or after remission achieved by steroid, and with highest tolerated dose of ACEI or ARB, for a duration of 12 months or more. The target CO-MPA blood level was between 1.5 and 4.5 mg/ml.

Sirolimus, is ja macrolide antibiotic, and like tacrolimus binds to immunophilin FK binding protein 12. Then this complex binds to mTOR, there by inhibit cell cycle, through blockage of GI to S phase transition, resulting in inhibition of growth of haematopoitic and lymphoid cells (T cells). It is approved for use in organ transplantation by FDA in 1999. As it has antiproliferative and immunosuppressive action, it has been tried in few trials of steroid resistant FSGS withvariable results.

Unlike calcineurin inhibitors, it is not nephrotoxic, reported adverse effects are impaired wound healing, hyperlipidaemia, reversible

cytopensias, and inceased susceptibility to infection.

Rituximab a monoclonal antibody (mAb) against the CD20 antigen present on B clles, was approved by the FDA in 1997 for the treatment of relapsed or refractory non-Hodgkin's lymphoma and more recently for patients with RA. Because the CD20 antigen is not expressed on hematopoietic stem cells, normal plasma cells, or other normal tissues, the use of this mAb allows for selective depletion of B cells.

In nephrology, it is being actively evaluated as an immuno-modulator in lupus nephritis, Wegener's granulomatosis, ANCA associated rapaidly progressive glmerulonephritis, membranous nephropathy, few cases of steroid resistant minimal change disease and FSGS. Recommended dose is four weekly doses of 375mg/m2 or 2 doses 1000 mg given two weeks apart.

Reported adverse effects are allergic reactions, hypotension, reactivation of viral infections like HBV, JC virus and progressive multi focal leukoencehalopathy.

Mizoribine is an immunosuppressive agent and has been used in Japan for the treatment of lupus nephrities, glomerulonephritis, transplant rejection and rheumatoid arthritis. Its active form mizoribine -5-P, selectively inhibits inosine monophosphate and guanosine monophosphatase synthetase, resulting in blockage of guanosine synthesis, which leads to inhibition of activation and proliferation of T cell.

It has reported to be safer than other conventional immunosuppressive medications and reported adverse effects are hyperuricaemia with use of high dose and rhabdomyolysis with concomitant use with statins.

Intravenous immunoglobulin has variety of immunomidulatory action, including acceleration of catabolism of Ig G. They have been tried in diseases like lupus nephritis, henoch-schonlein purpura, and number of glomerulonephritis, with variable results.

As they possess a number of side effects including acute renal failure, especially seen with sucrose based IV IG, they should be cautiously used until their precise role has been established.

CONCLUSION:

The primary glomerular diseases are a type of immuno – inflammation having various types of immune reactions (acute and chronic BSA mode). The purpose of using immuno-inflammatory therapy is to decrease the degree of proteinuria and to decrease the ongoing immuno-inflammation. The choice of drug should be such that it decreses the degree of drug should be such that itdecrease proteinuria and immuno-inflammation, as well as has minimal possible and reversible adverse effects. The Hence the physician and the nephrologists should use the appropriate drug and doses, and should carefully monitor the foradverse effect profile.

RECOMMENDED READING

1. *Cattran et al Cyclosporin in idiopathic glomerular disease associated with the nephritic syndrome: Workshop recommendations,. Kidney International (2007) 72,1429-1447.*
2. *Praga M, Barrio, V, Fernandez-Juarez, G Luno J. Tarcolimus monotherapy in membranous nephropathy; a randomized controlled trial. Kidney Int 2007; 71: 924-930.*
3. *Sepe et al,Mycophenolate mofetil in primary glomerulopathies Kidney International (2008) 73, 154-162.*
4. *Rituximab treatment of idiopathic membranous nephropathy, Fervenza et al, Kindney International (2008) 73,117-125*
5. *Sepe et al, Inferferon-B Reduces Proteinuria in Experimental Glomerulonephritis; J Am Soc Nephrol 18;2875-2884, 2007.*
6. *Brenner and Rector's – The Kidney, 8th edition.*
7. *Diseases of the kidney & urinary tract, R.W. Schrier, 8th edition.*
8. *Therapy in Nephrology and Hypertension, C.S. Wilcox, 3rd edition.*

Conference News

World Congress on Clinical, Preventive and Geriatric Cardiology (WCCPGC 2009)

October 2nd to 4th, 2009

At Swaminarayan Akshardham, N.H. 24, Akshardham Setu, Near Noida, New Delhi- 92, India

Organisers: WHA, AACIO, CSI DB, APSC, CAPSC, SA, IMSA, ISCU, IAE, DRDO, CHC, EI, WAMS, WASS, WAMBM & BSC

Host: Swaminarayan Akshardham, New Delhi, 110092

Partners: Media Partners: Times Foundation, Nai Duniya, Express Healthcare, Corporate Partner: ASSOCHAM, PSUs Partner: SCOPE,

Electronic Media Partner: NDTV, Knowledge Partner: PriceWaterhouse Coopers (PWC), Health Magazine; Life positive; Heal India

H.K. Chopra	***S. K. Parashar***	***Navin C. Nanda***	***Anil Kumar***	***Rakesh Gupta***	***Satish Kr. Gupta***	***Rajeev Gupta***
Chairman	*Chairman,*	*International*	*Chairman*	*Chairman*	*Secretary*	***D. Prabhakaran***
Organizing	*Scientific*	*President*	*Reception*	*Coordination*	*General*	*Convenors*

Her Excellency Mrs. Pratibha Patil, Hon.ble President of India has been requested to be the Chief Guest.

Faculty: National and International of Repute

SCIENTIFIC HIGHLIGHTS (WCCPGC 2009)

Recent advances in clinical cardiology, preventive cardiology, hypertension, congestive heart failure, dyslipidemia, coronary artery disease, medical angioplasty, coronary imaging, cardiodiabetes, metabolic syndrome, echocardiography, electrophysiology and cardiac arrhythmia, interventional cardiology, cardiovascular surgery and cardiac anesthesia etc.

"Kindly confirm your participation"."Registration and local hospitality is free" on "first come first serve Basis up to Sept. 1st, 2009"

For Registration:

Dr. H.K. Chopra,
Organising Chairman,WCCPGC-2009
C/o IMSA World HQ,
2nd Floor National Medical Library, Ansari Nagar, New Delhi – 110029
E-mail: drhkchopra@yahoo.com
Fax: 91-11-26444242, M: +91-9811090204
Ph: 91-11-26444242, 25658826, 26656990

Ms. Surbhi Bhuddi
Manager, WCCPGC-2009
CHC, No.5A/11041, 3rd Floor, W.E.A, Karol Bagh, New Delhi 110005 - India
E-mail:- surbhi@cardiopulseheartcare.com
Fax: 91-11-25815540, M: +91-9212596645
Ph: +91-11-25815539 Extn. 33
All India Toll Free No; 1800 11 33 22

Website : www.wccpgc.com

SUMMATIVE INDICATIONS OF INITIATION OF DIALYSIS THERAPY FOR THE NEW CENTURY

P.J. Mahanta, S.C. Tiwari

Department of Nephrology AIIMS, Ansari Nagar, New Delhi-110029. India.

Once a patient has reachsed stage 4 chronickidney disease (CDD) (estimated glomerular filtration rate (eGFR) of -30 ml/minute/1.732), he should be under a nephrologists's care, and the risk factors associated with cardiovascular disease and progression of chronic kidney disease should be optimally managed. Survival of end stage renel disease (ESRD) patients on dialysis depends to a large extent on their condition at the time time dialysis was first initiated. So it is essential to optimize control of blood pressure, anemia, calcium/ phosphorus level, and nutrition during the predialysis period, as well as timely vaccination against HBV and creation of an arterio-venous fistula.

When GFR falls below 10ml/min/1.73m2, the patient develops uraemic syndrome, which consists of symptoms and signs that result from toxic effects of elevated levels of nitrogenous and other wastes in the blood. They include nausea, vomiting soon after awakening, progressive loss of appetite, weakness uraemic encephalopathy, uremic pericarditis or pleurisy, severe metabolic acidosis, hyperkalaemia not controlled with dietary measure or medical therapy. Conventionally, at this stage, haemodialysis is initiated. Indications for urgent dialysis are life threatening htyperkalaemia (associated with ECG changes; usually K =6.5mmol/1/2), acidosis, fluid overload, uraemic pericarditis with or without pericardial temponade.

But in a practical scenario some patients, especiially those with co-morbidities, are more susceptible to various complications of uraemia and may require earlier initiation of dialysis even if eGFR is much more above usual level of initiation of chronic dialysis, i.e. 10 ml/ minute/1.73m2.

The rationale for the approach of early initiation of dialysis is that ultimate survival on dialysis depends greatly on nutritional status, serum albumin, cardiorvascular status, fluid and electrolyte balance, acidosis, and immunological status at the time dialysis is initiated. Patients started early on dialysis (at higher eGFR levels) have higher serum albumin levels. Furthermore, spontaneous protein intake begins to fall early in chronic renal insufficiency (when eGFR is still above 25 ml/1.73m2). CKD state of relative immuno-deficiency; and it progressively leads to various infections like pneumonia, tuberculosis, even to opportunistic infections. With progression of CKD, normal homeostasis and metabolism of many vitamins and micronutrients get impaired, there by predispose to malnutrition and deficiency syndromes. Moreover various studies have documented that risk of cardio-vascular morbidity and mortality significantly increases with progressive worsening of renal functions. Calcium-phosphorus-PTH metabolism also progressively get deranged with deterioration of renal function, and besides worsening of renal bone disease, they are also implicated in cardiovascular morbidity and mortality. Metabolic acidosis, which gradually worsens with deterioration of renal functions, leads to disturbance in protein metabolism, aggravates renal bone disease, affects phagocytic functions, alters cellular metabolisms, and cause weakness and fatigue. Several studies have claimed that early initiation of dialysis results in decrease in mortality and also hospitalization rates 3-7. Therefore, indications of initiation of initiation of dialysis may be revised and classified as **major indications** and **minor or summative indications,** where atient with stage 4 CKD with any of the minor indications may be benefitedfrom early initiation of dialysis. But presence of 3 or more minor indications should be considered as a major indications and dialysis should be initiated.

Major indications or criteria	Summative/minor indication or criteria
1. Uraemic encephalopathy	Any level of GFR or stage 4 CKD with;
2. Metabolic acidosis	
3. Fluid overload or pulmonary edema	1. Severe malnutrition
4. Hyperkalaemia	2. Severe hypoalbuminaemia
5. Udraemic pericarditis	3. EPO resistant anaemia
6. Uraemic G.I. symptoms-nausea, Vomiting, hic-cough, anorexia with	4. Repeated infections
	5. Hypertiension difficult to control Conventional medications
7. Yraenucbeyropathy – foot drop, wrist Drop, tremor, asterixis, multifocal Myoclonus	6. Metabolic bone dispite adequate medical therapy
8. Uraemic coagulopathy EPO	7. Chronic fatigue, despite adequate nutritional supports including
	8. Marked cognitive impairment
	9. Marked sexual dysfunction
	10. Children, with failure to thrive
	11. Advanced cardiac disease – intolerant to Even mild or moderate fluid overload.
	12. Associated co-morbidities like diabetes
	13. Insomnia, or severe pruritus

INDICATIONS FOR DIALYSIS IN ARF;

There are no absolute levels at which dialysis is always needed. The risk is that a patent with moderate hyperkalaemia, acidosis, and mild pulmonary edema may become acutely worse or develop an arrhythmia, and then be un-resuscitable, and dialysis should be initiated early. For patients with multi-organ failure, sepsis syndrome, and postoperative renal failure, sustained low efficiency dialysis (SLED) or continuous renal replacement therapy (CRRT) should be begun early to minimize the deleterious effects of uraemia on myocardial function, bleeding, and wound healing. Poor nutrition and hence low muscle mass may mask severe renal failure by limiting the degree of uraemia. Some patients require dialysis for acidosis or fluid overload despite reasonable renal function. Diabetic patients often require earlier initiation of therapy. Other indications for acute dialyisis include poisoning (lithium.methanol, and ethylene glycol), hypothermia, hyperuricaemia, and metabolic,alkalosis-2,6.

RECOMMENDED READING

1. *Handbook of Dialysis, 4th Edition, John t. Daugirdas*
2. *Oxford Handbook of Dialysis, 2nd Edition*
3. *Brenner and Rector's The Kidney, 8th edition.*
4. *Diseases of the kidney & urinary tract, R.W. Schrier, 8th edition.*
5. *Therapy in Nephrology and Hypertension, C.S. Wilcox, 3rd edition*
6. *Intermitted versus continuous renal replacement therapy for acute kidney injury patients admitted to the intensive care unit: results of a randomized clinical trial – Robert L. Lins et al, Nephrol Dial Tranplant (2009) 24: 512-518*
7. *Short daily and nocturnal haemodialysis, new therapy for new century, MV Rocco, Soudi J Kidney Dis Transplant, 2009;20(1)1-11.*

Correspondence: Prof. S. C. Tiwari, *Fax: 91-11-26588641,* ***e-mail:*** *sctiwari@hotmail.com*

INNOVATIVE PROCEDURES IN OBSTETRICS/GYNECOLOGY

PROCEDURE - I

EVALUATION OF SURGICAL TREATMENT OF CERVICAL INCOMPETENCE : INTRODUCTION OF A SIMPLE STITCH & COMPARISON WITH OTHER TECHNIQUES

R. J. Sreenivasan, Venu Gopal

Department of Obst. & Gyne. , Karnool Medical College & Govt General Hospital, Karnool(Andhra Pradesh) India.

Abstract: *Cervical Incompetence is a definite entity. Many patients complain of mid-trimster abortion, Careful evaluation and timely intervention bestows faith and confidence in the patient; ecstacy and joy, on the face of the mother with her fond baby, is to be seen to believe and the happiness is shared by the obstetrician too. A Prospecitive study of 100 cases of cervical cerclage procedures of 4 different types was done to compare the success rate. The types of cerclage procedures done were Mc. Donald's, Wurm's modified shirodkar's and Sreenivasan's stich. A simple cervical stitch to treat cervical incompetence, is being presented.*

INTRODUCTION

Cervical incompetence (CI) is a deficiency of structure and function resulting in an inability of cervix to retain a developing pregnancy within the uterus. It causes repeated mid trimester foetal wastage and also pre-term labour. The authors have refrained from dealing with the incidence, anatomy of the cervix, etiologicalo factrs and clinical signs, symptoms and aids to diagnosis of cervical incompetence as the literature is well documented.

MATERIAL METHODS

One hundred (100) cases of cervical incompetence were subjected to four different cercleage procedures as depicted diagrammetically

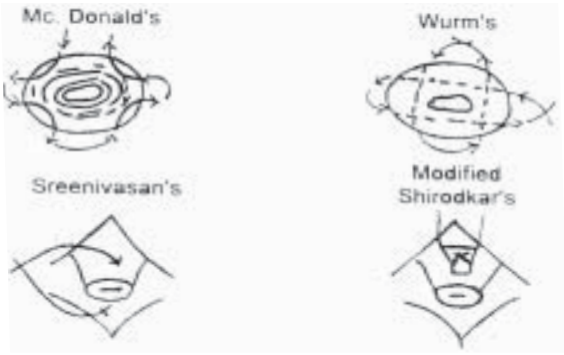


Fig.: Type of cerclages

(fig.)

Procedure: **Sreenivasan's stich** was performed under simple pre-medication using 100 mg diazepam I.V + 50 mg of atropine. After pre-medication patient was kept in lithotomy position. Vulva and vagina were cleaned with betadine lotion and draped with sterile towels. Cusco speculum was then introduced and cervix exposed. Both lips of cervicx were held with Allis forceps. Black silk (1.0) on a round bodied needle was passed antero-posteriously at the level of the isthmus and the knot placed anteriorly and tightened with Hegar's No.8 dilator in-situ. A finger was introduced into the cervical os to make sure the internal os is closed.

Prophylactic stitch:- In 14 cases of recurrent mid-trimster abortions during the past decade 1996-2006, author applied the stich prophylactically when the patient was not pregnant and all of them conceived naturally and carried the pregnancy to term. Routine antenatal checkup was carried out., and were without apprehension of fear of mid-termination. The patients were given tocolytics and aliylestranol and advised to apply vaginal povidon ointment daily.

Post operative period:- Immediately after the cerclage patients were confined to bed, uterine contractions were prevented with isoxsuprine hycrochloride 40 mg. IV. dripped slowly. Amplicillin 500 mg bd was given for 5 days. An injection of depot progerterone 500 mg. I.M. was also given. Micronised progerterone 100 mg. daily was given for 4 weeks. Patients were discharged on the same day after giving few hours of bed rest. They are advised to apply metrogyl and povidon ointment vaginally once daily to prevent local infection.

RESULTS & DISCUSSION

Cerclage Procedures

Four types of Cervical cerclage were applied to 100 patients of cervical incompetence (table 1). The diagnosis of CI was entirely based on history of repeated mid-trimister fetal losses and preterm labour. 84% of the cerclage procedures were done before 20 weeks of gestation. Cerclage was repeated in 20% of patients. The commonest age group of patient was 20 to 25 years.

Table1 : *Types Of Cerclages:*

S.No	Types of cerclages	No. of cases	%
1.	Mc. Donald's	48	48%
2.	Wurm's	09	9%
3.	Modified Shirodkar's	03	3%
4.	Srinivasan's	40	40%

Patients after **Srinivasan's stitch** were discharged from the hospital on the same day and in other types of cerclage procedures patients

Correspondence: R. J. Sreenivasan*, Medical Superintendent & Head, Department of Obst. & Gyne. , Karnool Medical College & Govt General Hospital, Karnool (Andhra Pradesh) India.*

were hospitalized for a perod of 3 to 7 days. All the patients were under the medication of lsoxsuprine hydrochloride and 17 hydroxy progesterone after the procedure. Black Silk (n 1.0) was the material used for the study and the placement of knot was anterior. The correct timing of cerclage appears to be ranging from 12-18 weeks. A failure rate of 37.5% was noted in cerclage procedures performed after 20 weeks when compared to 7.4% if procedure was done before 20 weeks.

Labour in all cases the stitch was removed at term; normal labour was allowed when no contra indication. Removal of the stitch is simple and in the event of their not attending our clinic clear instruction was given in the discharge summary to the local doctor about the removal of the stitch at term to enable normal delivery. In 3 cases, the stitch was dislodged and had fallen off without being detriment to pregnancy. In 6 cases where the patient delivered at home unattended the stitch simply got displaced without a tear and delivery proceeded normally.

The **end result of the prospective study**- i.e. the effect of cervical cerclage in cervical incompetence is best assessed by *foetal survival*. In comparison with other cerclage procedures the foetal survival rate in srinivasan's stitch is 87.5% where as in other procedures Mc. Donald's 89.59% wurm's 88.88% and modified shirodkar's 66.60%

Table 2 : *Infant salvage Ratio(ISR)*

	Infant survival						
	Before cerclage			After cerclage			
Types of cerclage	Preg-nancies	Survi-vors	%	Preg-nancies	Survi-vors	%	Infant Salvage Ratio
Mc. Donald'd	141	28	19.5	48	43	89.58	4.51
Wurm's	26	9	34.70	9	8	88.88	2.55
Modified Shirodhar's	5	1	20.10	3	2	66.6	3.33
Sreenivasan's	121	30	24.81	40	35	87.50	3.52

. (Table 2)

RECOMENDED READING

1) ***Shirodkar VN*** *– A new method of operative treatment of habitual abortions in the II Trimester of Pregnacy. Anti-Septic 1955. 52-299.*
2) ***Mc. Donald IA :*** *Suture of the cervix for Inevitable reisecarriage Obt. & Gynec. Of the Br. Evep – 1957: 64: 364-350.*
3) ***Hefner J.D., Patow WE, Ludwing T.M.*** *The Wurm Procedure, New Surgical Procedure for the correction of the incompetent cervix during pregnancy., Obst. & Gynec. 1961. 18 616 - 620.*
4) ***Mc. Donald IA:*** *CI. Obst. & Gyn. Cervical Cerclage, 1980, 7 461 -479.*
5) ***Lidegeard O.*** *Cervical Incoverpetercca and Cerclage in Denmark, 1980 – 1990 Acta Obst. & Gynec. Stand. 1994: 73: 35- B.*

PROCEDURE - II

SREENIVASAN'S SLING FOR NULLIPAROUS PROLAPSE

R. J. Sreenivasan, Dhunnanjaya Rao

Department of Obst. & Gyne. , Karnool Medical College & Govt General Hospital, Karnool(Andhra Pradesh) India.

INTRODUCTION

Prolapse of the uterus occurs in 20% of women over 40 years of age. Nulliparous prolapse occurs in 10% of women between 20 years and 30 years age. [1,2]

DEFINITION

Prolapse of the uterus is one form of uterine displacement i.e., downward, the others being lateral, posterior and inversion. ***Nulliparous Prolapse*** is defined as descent of cervix below the ischial spines and later outside introitus without descent of vaginal fornices, bladder of bowel.

ANATOMY OF PRO LAPSE UTERUS

Normally uterus is a pelvic organ – anteverted and anteflexed. It is kept in position by the following support:- ligaments; Fascia; Muscle; Intra abdominal pressure

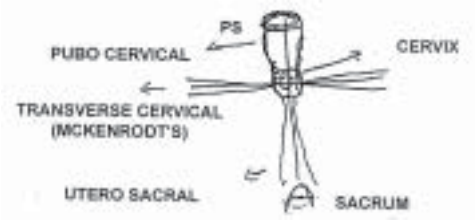
Ligaments:- Sign of the cross+ Three pair of Ligaments:- *Anterior* - Pubo cervical; *Lateral* - Transverse cervical (Mckenrodt's); *Posterior* - Utero sacral

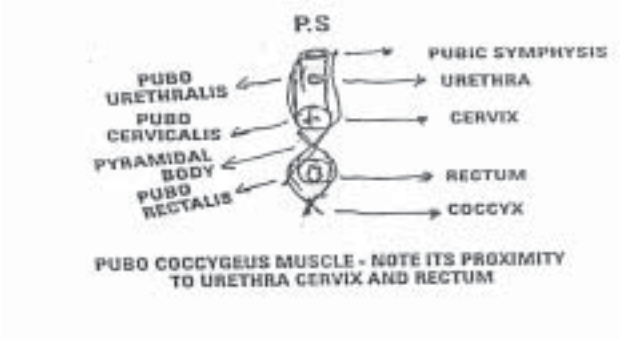
Fascia :- Endo pelvic Fascia – cushoning media consisting of smooth muscle, fat and connective tissue.

Muscle:- Pubo coccygeus – part of levator ani

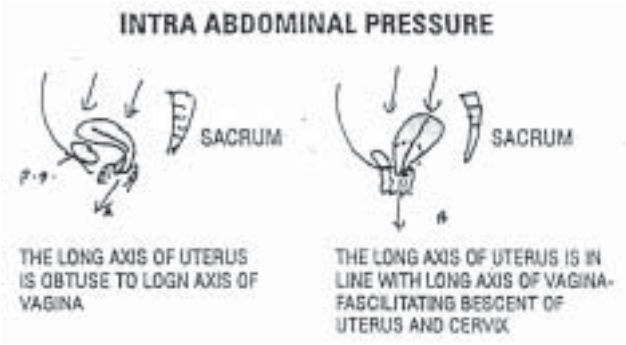
LIGAMENTARY SUPPORTS OF THE UTERUS (SIGN OF THE CROSS)

(Three pairs – Anterior, Lateral and Posterior)





Intra – Abdominal Pressure (IAP):- Any Increase in Intra Abdominal Pressure tends to antevert the uterus futher which impinges on P.S., and prevents descent (See Diagram)



ETIOLOGY OF PROLAPSE

1) Congenital
2) Traumatic – Obstetric Operative
3) Post meno Pausal
4) Chronic Bronchial Asthma
   Tuberculosis
   Constipation
5) Ovarian Tumours, Fibroid Uterus.

DIFFERENTIAL DIAGNOSIS

1) Cystocele, anterior vaginal cysts.
2) Cervical Polyp
3) Inversion uterus
4) Congenital elegation of cervix

SURGICAL TREATMENT

Aim of the Operation is: 1) Preservation of menstrual function.; 2) Preservation of child bearing function; 3) Preservation of sexual function; 4) Relief of symptoms – dragging pain, back ache, uneasiness on walking, dyspareunia; 5) To reposition the uterus in its normal place.

Various sling operations

1) Sacral Hysteropexy (Arthur and Sauage,1957)[3]
2) Posterior Abdominal Cervicopexy (Shirodkar, 1959)[4]
3) Abdominal Sling Operation (Purandare 1966, Chaudhuri 1983)[2,5]
4) Modified Manchester Operation (Currie 1952)
5) Vaginal Sling Operations (Shirodkar, 1959, Chaudhuri, 1979)[4,5]
6) Khanna's Sling Operation.

OPERATIVE PROCEDURE OF SRINIVASAN'S SLING

TECHNIQUE

Steps of Operation

1. Sub – umbilical midline incision.
2. Peritoneal cavity opened
3. General inspection of the pelvis
4. Both round ligaments held with straight artery forceps applied at its medial end
5. U.V. Fold opened – *Photo 2*.
6. Isthmus identified
7. A'Z' shaped stitch applied over the isthmus with No. '1' Black silk with the ends left long – *Photo 3*
8. The ends brought extra peritoneal lateral to the bladder and brought out on either side of medline – *Photo 4*
9. U.V. Fold sutured with 1 'O' chromic catgut *Photo 5*
10. Round ligaments plicate with silk.
11. General peritoneum sutured with 1 'O' chromic catgut.
12. The ends of the silk thread are brought in front of anterior rectus sheath lateral to rectus muscle. *Photo 6*
13. Rectus sheath sutured with No. '1' chromic catgut – *Photo 7*
14. Tension applied to both ends of silk thread till the cervix recedes above the ischial spines and the silk thread is anchored to rectus sheath – on either side of mid line – *Photo 8*
15. Cervix recede into its normal position – *Photo 9*
16. Skin sutured with interrupted sutures using thread.



Fig 1a: Nulli parous prolapse- showing CX outside introitus



Fig 1a: Nulli parous prolapse- showing CX outside introitus



Fig.2:Laparotomy showing uterus opening of utero- vesicle peritoneum



Fig.3: Showing isthmus- "Z" Stitch applied with long ends



Fig.4: Bringing the ends of the sutures extra peritoneally lateral to the rectus sheath 21/2 above public symphysis



Fig.5: Closure of Utero- Uesicle Peritoneum



Fig.6: Bringing the ends of the sutures extra peritoneally lateral to the rectus sheath 21/2 above public symphysis



Fig.7: Closure of rectus sheath



Fig.8: Anchoring of the silk thread on either side of midline in front of rectus sheath a make sure CX is drawn in



Fig.9: Showing CX well drawn up into normal position



Fig.10a : Showing pregnancy-Frontal



Fig.10a : Showing pregnancy-Lateral

MANAGEMENT OF LABOUR IN PREGANCY FOLLOWING SLING

When no contra indication normal vaginal delivery assisted with vacuum is preferred.

AUTHOR EXPERIENCE

Nulliparous prolapse results from laxity of pelvic floor (the urogenital diaphragm) due to congenital weakness. I observed that cervical isthmus could be vertically pulled upwards with No. 1 black braided silk and anchored in front of rectus sheath for repositioning the uterus in nulliparous prolapse. I applied this principle in the sling operation for cervicopexy with good results.

RESULTS

The author has performed 25 operations with good result. Pregnancy following Srinivasan's sling is 48 percent.

REFERENCES

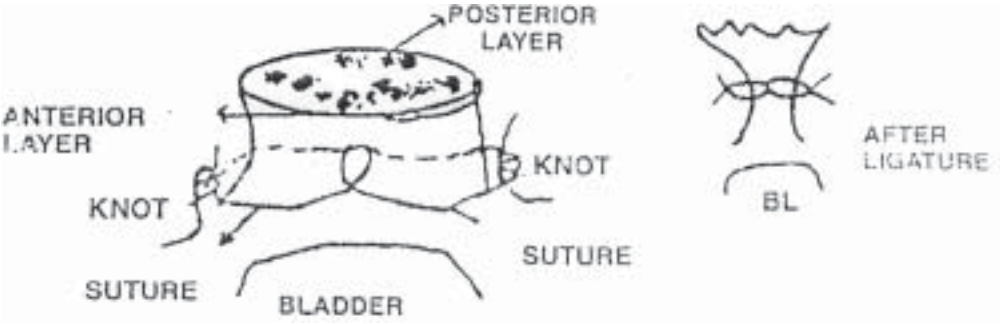
1. ***Anjaneyulu, R :*** *In Postgraduate Obsterics and Gynaecology (Eds. M.K. Krishna Menon, P.K. Devi and K. Bhaskar Rao), 1 stands. Orient Longmans ltd., New Delhi, 1980, p.249.*
2. ***Chaudhuri, S.K.*** *: J. Obstet. Gynaec. India. 32 : 817, 1938b.*
3. ***Arthure, H.G.E. and Savage, D.*** *: J. Obstet. Gyanaec, Brit, Emp. 64 : 355, 1957.*
4. ***Shirodkar, V.N.*** *: Contributions to Obstetrics and Gynaecology, E. & S. Livingstone Ltd., London, 1959.*
5. ***Chaudhuri, S.K.*** *: Int. J. Gynec. Obstet. 16 : 314, 1979.*
6. ***Howkins, J. and Hudson, C.N. (Eds.)*** *: Shaws Textbook of Operative Gynaecology, 5th Edn., Churchill Livingstone Edinburgh, 1983.*

PROCEDURE - III

A SIMPEL EMERGENCY LIFE – SAVING SURGICAL PROCEDURE FOR CONTROL OF PPH : A CASE REPORT

R. J. Sreenivasan, Dhunnanjaya Rao

Department of Obst. & Gyne. , Karnool Medical College & Govt General Hospital, Karnool(Andhra Pradesh) India.



CASE REPORT

31 years. G3L2 post-Cesar pregnancy was admitted with 7 MA and profuse bleeding PV of four hours duration. General condition - patient was pale, had tachycardia, B.P. 100/60mm HB 7 gm%, urine was clear, blood group B+ve.

Abdominal examination: Uterus 28 weeks, tense, foetal parts felt, cephalic foetal hearty absent.

Ultra sound Examination: single foetus, cephalic 28 weeks foetal heart absent. Placenta in lower segment covering internal OS, type II anterior.

Treatment: Compatible B+ve blood transfusion 2 units given. Laparotomy was done. Lower segment was vascular, was incised through placenta; she delivered a female dead foetus 1 kg. in weight. Placenta was adherent to the lower segment. Placenta previa and acreta removed piecemeal. There was profuse haemorrhage. Sub-total hysterectomy was done, bleeding was still uncontrollable. The anterior wall was very thin and papery. Blood was oozing. The anterior and posterior walls sutured as shown in the figure; haemostasis secured. Patient recovered uneventfully and was discharged on 8th day. This is a simple surgical procedure which can be life-saving in an emergency.

WOCKHARDT

Help your patients with anaemia

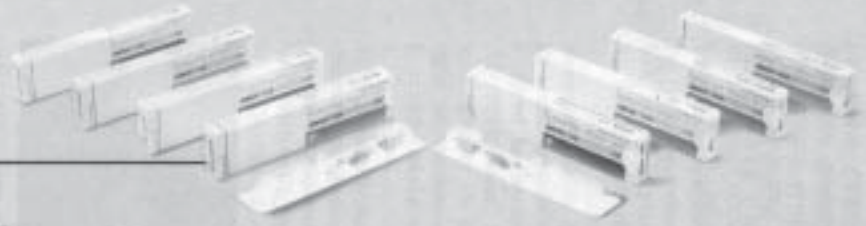
Move on with Life Energy

Human Recombinant Erythropoietin

WEPOX

Pre-filled Syringe

A True Regard For Life





Feroject

Iron Sucrose Injection

100 mg/5 ml ampoule

Unleashing energy... unrestrained

For the treatment of SHPT in dialysis patients

CeRACaL™

Cinacalcet 30 / 60 mg tablets

sensitizing CaSR. optimizing PTH

- A potent and selective type II calcimimetic agent
- Functions as allosteric activator of the CaSR
- Inhibits PTH secretion and synthesis
- Inhibits Parathyroid hyperplasia
- Lowers Ca x P and cardiovascular calcification

References

1. Chronic Kidney Disease: The Looming Threat. Brig AS Nanda* MIAPI 2008; 64 : 2-3
2. Chronic Kidney Disease: A Perspective, C Shyam, et al. JACM 2007; 8(2): 150-63
3. Rodriguez M. The calcium sensing receptor: a key factor in the pathogenesis of secondary hyperparathyroidism. Am J Physiol. 2005;288:F253-264.
4. Brancaccio D, et al. Vitamin D Receptor Activator Selectivity in the Treatment of Secondary Hyperparathyroidism. UnderStanding the Differences Among Therapies. Drugs. 2007; 67 (14): 1981-1998.
5. Torres PU. Cinacalcet HCl: A Novel Treatment forSecondary Hyperparathyroidism Caused by Chronic Kidney Disease. Journal of Renal Nutrition, Vol 16, No 3 (July), 2006: pp 253-258.
6. Drueke TB. Modulation and action of the calcium-sensing receptor. Nephrol Dial Transplant (2004) 19 (Suppl 5): v20–v26
7. Achieving NKF-KDOQITM bone metabolism and disease treatment goals with cinacalcet HCl- Kidney International, Vol. 67 (2005), pp. 760–771

Abridged Prescribing Information

COMPOSITION: CERACAL 30/60 mg: Each film coated tablet contains Cinacalcet hydrochloride equivalent to Cinacalcet 30/60 mg. DESCRIPTION: CERACAL (Cinacalcet) is a calcimimetic that increases the sensitivity of the calcium-sensing receptor to activation by extracellular calcium. PHARMACOKINETICS: Peak plasma concentrations achieved within 2-6 h after oral administration; higher plasma levels achieved with a fatty meal; Highly plasma protein bound (93-97%) ; Cinacalcet is metabolized by multiple enzymes, primarily CYP3A4, CYP2D6 and CYP1A2; Primarily excreted renally; Long terminal half-life (30-40 h); No significant pharmacokinetic differences in geriatric patients or in patients undergoing dialysis. INDICATION AND USAGE: Cinacalcet is indicated for the treatment of secondary hyperparathyroidism in patients with Chronic Kidney Disease on dialysis. Cinacalcet is indicated for the treatment of hypercalcemia in patients with parathyroid carcinoma. CONTRAINDICATIONS: Cinacalcet is contraindicated in patients with hypersensitivity to any component of this product. WARNINGS: Serum calcium levels should be closely monitored in patients receiving Cinacalcet, particularly in patients with a history of a seizure disorder. PRECAUTIONS: General: Hypocalcemia: Cinacalcet lowers serum calcium, and therefore patients should be carefully monitored for the occurrence of hypocalcemia. Potential manifestations of hypocalcemia include paresthesias, myalgias, cramping, tetany, and convulsions. Cinacalcet treatment should not be initiated if serum calcium is less than the lower limit of the normal range (8.4 mg/dL). Adynamic Bone Disease: A patient bone disease may develop if PTH levels are suppressed below 180 pg/mL. Hepatic Insufficiency: Patients with moderate and severe hepatic impairment should be monitored frequently with Cinacalcet. Information for Patients: It is recommended that Cinacalcet be taken with food or shortly after a meal. Tablets should be taken whole and should not be divided. Pregnancy - Category C: There are no adequate and well-controlled studies in pregnant women. Cinacalcet should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. Lactating Women: Studies in rats have shown that Cinacalcet is excreted in the milk. It is not known whether this drug is excreted in human milk. Considering these data in rats and because many drugs are excreted in human milk and because of the potential for clinically significant adverse reactions in infants from Cinacalcet, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the lactating woman. Pediatric Use: The safety and efficacy of Cinacalcet in pediatric patients have not been established. Geriatric Use: No differences in the safety and efficacy of Cinacalcet were observed in patients greater or less than 65 years of age. DRUG INTERACTIONS: An in vitro study indicates that Cinacalcet is a strong inhibitor of CYP2D6, but not of CYP1A2, CYP2C9, CYP2C19, and CYP3A4. In vitro induction studies indicate that Cinacalcet is not an inducer of CYP450 isozymes. Metoprolol: Cinacalcet AUC0-τ increased 2.3 and 2.2 times, respectively, when a single 90 mg Cinacalcet dose on Day 5 was administered to subjects treated with 200 mg metoprolol twice daily for 5 days compared to 90 mg Cinacalcet given alone. Calcium Carbonate: No significant pharmacokinetic interaction was observed when a single dose of 1500 mg calcium carbonate was coadministered with 300 mg Cinacalcet. Pantoprazole: No significant pharmacokinetic interaction was observed when Cinacalcet 90 mg was administered to subjects treated with 80 mg pantoprazole daily for 3 days. Simvastatin/HCTZ: No significant pharmacokinetic interaction was observed when 2400 mg simvastatin/HCTZ was coadministered with 90 mg Cinacalcet tablet. Omeprazole: The effect of Cinacalcet 100 mg/l on the pharmacokinetics of desipramine 250 mg/l has been studied in healthy subjects who were CYP2D6 extensive metabolizers. The AUC and Cmax of desipramine increased by 3.6 [(90 % CI: 3.468 ,3.96)] and 1.35 (1.52, 1.194,1.96) fold, respectively, in the presence of Cinacalcet. This indicates that Cinacalcet is a strong in vivo inhibitor of CYP2D6 and can increase the blood concentrations of drugs metabolized by CYP2D6. Amitriptyline: Concurrent administration of 25 mg or 100 mg Cinacalcet with 50 mg amitriptyline increased amitriptyline exposure and nortriptyline active metabolite exposure by approximately 20% in CYP2D6 extensive metabolizers. Warfarin: R- and S-warfarin pharmacokinetics and warfarin pharmacodynamic were not affected in subjects treated with warfarin 25 mg who received Cinacalcet 30 mg twice daily. The lack of effect of Cinacalcet on the pharmacokinetics of R- and S-warfarin and the absence of drug induction upon multiple dosing in patients indicates that Cinacalcet is not an inducer of CYP2C9 in humans. Midazolam: There were no significant differences in the plasma concentrations of midazolam, a CYP3A4 and CYP3A5 substrate, in subjects receiving 90 mg Cinacalcet once daily for 5 days and a single dose of 2 mg midazolam on Day 5 as compared to those of subjects receiving 2 mg midazolam alone. This suggests that Cinacalcet would not affect the pharmacokinetics of drugs predominately metabolized by CYP3A4 and CYP3A5. SIDE EFFECTS: Nausea/vomiting exacerbation in patients with Chronic Kidney Disease on dialysis: In clinical studies the most frequently reported events with Cinacalcet were nausea and vomiting. Other adverse effects observed were diarrhea, myalgia, hypertension, dizziness, rashes, asthenia, anorexia, chest pain [non cardiac] and access clotting. DOSE AND ADMINISTRATION: Dose titrate up to 300 mg once daily have been safely administered to patients on dialysis. Dose dosage of Cinacalcet may lead to hypocalcemia. In the event of overdosage, patients should be monitored for signs and symptoms of hypocalcemia and appropriate measures taken to correct serum calcium level. Since Cinacalcet is highly protein bound, hemodialysis is not anticipated to enhance the removal of Cinacalcet. DOSAGE AND ADMINISTRATION: Cinacalcet tablets should be taken whole and should not be divided. Cinacalcet should be taken with food or shortly after a meal. Dosage must be titrated to the individual patient. HOW SUPPLIED: CERACAL 30/60 are available in 10 tablets per blister.

Biocon

Biocon Limited
20th K.M. Hosur Road
Electronics City
Bangalore 560 100
T 91 80 2808 2808
F 91 80 2852 3423

www.biocon.com

Introducing...

In Anemia of CRF



Venofer®

Iron Sucrose

Global Leader in IV Iron Therapy

- Fast
- Safe
- Targeted

From the makers of

LG ESPOGEN™

LGLSI

Dedicated to Nephrology

LG Life Sciences India Pvt. Ltd. LG House, AB-3, Safdarjung Enclave, New Delhi - 110 029, INDIA
Tel. : +91-11-26714004, Fax : +91-11-26713716, E-mail : lglsi@lglsi.com www.lglsi.com