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A Multidisciplinary Medical Journal

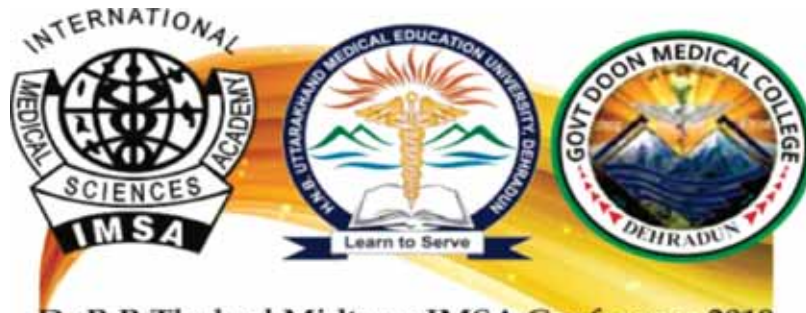
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Dehradun, Uttarakhand



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Aims and scope

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Association News

Dear Colleagues,

International Medical Sciences Academy (IMSA) is a global organization established as a registered society on 28th March 1981 with world headquarters at New Delhi. It is the only international body which encompasses all disciplines of medicine. It has regions in America, Australia, Europe, Africa, rest of Asia and India. There are 28 chapters world over. IMSA is run by Board of Trustees apart from other executive committees. IMSA is an associate member of Council for International Organizations of Medical Sciences (CIOMS). It has about 2850 Fellows & Members world over and the membership is expanding. Many Nobel Laureates are its fellows.

The main objectives of IMSA is to bring together national and international medical scientists, medical educationists, medical and public health administrators and research workers in medical and health sciences on a worldwide basis for advancement of health of all the people in the world. The academy also arranges courses, training programs, CME programs and Rural CME programs. IMSA publishes quarterly journal, JIMSA in which original articles, updates, symposia, special issues on topics of current interest are published.

Our Annual Conference - IMSACON 2019 will be held at Baskent University, Ankara, Turkey on November 6, 7 & 8, 2019. All are invited to be a part of this academic celebration.

Dr. R. R. Thukral Midterm IMSA Conference 2019 will be held at Govt. Doon Medical College, Dehradun, Uttarakhand, India on April 12 & 13, 2019. All are invited to be a part of this academic celebration.

Though IMSA has been in service of medical profession and has been encouraging development of medical sciences by bringing information technology into the profession thus improving the health of nations, yet we do not have our own building to work more effectively. Our organization is committed to the medical profession for promoting Continuing Medical Education and also holds educational programmes on topics of National and public health importance. We need to conduct more seminars, organize lectures by National and International experts and hold regular workshops and group discussions. For arranging such activities we are badly in need of our own building with adequate infrastructure and facilities like an Auditorium, projection room, library, committee rooms for interactive sessions etc. So far we have been operating from small rented space which can hardly accommodate our office.

Friends, we have been fortunate to get a piece of land about 500 sq.mtrs allotted to us by the Lt. Governor of Delhi for developing the IMSA World Head Quarters at Delhi. I am approaching all Fellows and Members to donate at least Rs. 5000/- each to meet the cost of the land as well as for construction of our own building. The donations are exempted from tax under 80G; the cheque may please be made in the name of "**IMSA - Building Fund**" payable at New Delhi, and sent to the Headquarters.

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Dr. K.Jagadeesan, President, IMSA,WHQ

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10.12.17

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Dehradun, Uttarakhand, India

April 12 & 13, 2019

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Vitamin - B₁₂ : Deficiency in India

Narinder Pal Singh, Anish Kumar Gupta

Department of Medicine,
Max Super Speciality Hospital , Vaishali

Vitamin B₁₂ (cobalamin) is an essential micronutrient needed for optimal health. Since the reporting of associations between homocysteine and chronic disease there has been renewed interest and increased worldwide concern about the consequences of vitamin B₁₂ deficiency on health. In developing countries such as India, B₁₂ deficiency is much more common, starting in early life and persisting across the life span and the prevalence of deficiency increasing with age. The reasons for these are related to diet and lifestyle that are greatly influenced by diverse religions, ethnic and socioeconomic heterogeneity of Indian population. There is paucity of true prevalence of vitamin B₁₂ deficiency in Indian population. The prevalence of subnormal vitamin B12 in elderly varies from 3 to 40.5% depending on the cut-off used for defining deficiency of the B12 level in serum[1]. Food that are high in B12 include: liver (26 to 58 µg/100g), beef and lamb (1 to 3 µg/100g), chicken (trace), eggs (1 to 2.5 µg/100g) and dairy foods (0.3 to 2.4 µg/100g). In Western countries people regularly consume meat or egg as source of protein. In addition, majority do consume raw vegetables and fresh fruits. Thus, consume regularly 5–30 mcg of cobalamin daily. As compared to a western diet, there is no vitamin B₁₂ in the average Indian diet as India has the maximum number of vegetarians in the world. Apart from dietary factors there are several others, which could potentially cause vitamin B₁₂ deficiency. They are related to interference in ileal uptake of vitamin B₁₂ due to diseases (subclinical, clinical or treated ileocecal tuberculosis, Crohn's Disease), hepatobiliary dysfunction or chronic pancreatitis, interference by bacterial overgrowth or parasitic infection, drug-nutrient interactions (Proton Pump Inhibitors, metformin, or other drugs such as cytotoxic

drugs, alcohol, colchicine, nitrous oxide anaesthesia and potassium chloride) as well as less common genetic defects [2].

Vitamin B₁₂ is a complex water soluble micronutrient, plays an important role in DNA synthesis and neurologic function. It serves as a cofactor for methionine synthesis by transfer of methyl group to homocysteine that is an atherogenic and potential endothelial toxin. This conversion of homocysteine to methionine forms demethylated tetrahydrofolate (THF) which is required for DNA synthesis. Further metabolism of methionine to S-Adenosyl methionine (SAM) is essential for myelin synthesis and neurotransmitter regulation as well as maintenance of neuronal integrity. Vitamin B12 absorption is complex and its bioavailability is dependent on an individual's GI absorption capacity, quantity and type of protein consumed [3]. Vitamin B₁₂ from foods appears to have different absorption rates with better absorption from chicken and beef as compared to eggs. Absorption of B₁₂ is both passive and active; Passive absorption is possible through buccal, duodenal and active absorption is from the ileum. It is assumed that 50% of dietary vitamin B₁₂ is absorbed. The majority of B₁₂ is stored in the liver; some B₁₂ is excreted in bile and undergoes enterohepatic circulation. Absorption, enterohepatic circulation and intracellular metabolism of vitamin B₁₂ have been demonstrated in figure 1.[4]

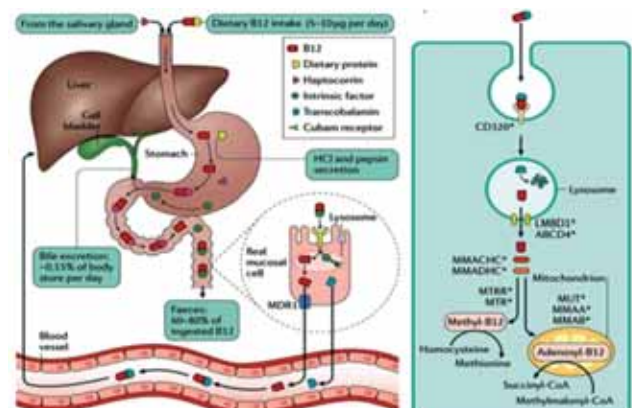


Figure 1: Absorption, enterohepatic circulation and intracellular metabolism of vitamin B₁₂ (adopted from Green R et al⁴)

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Vitamin B₁₂ deficiency is associated with hematologic (megaloblastic anemia, pancytopenia-leukopenia, thrombocytopenia), neurologic manifestations (paresthesias, peripheral neuropathy, and neural tube defect include spina bifida, anencephaly, and encephalocele), and psychiatric disorders (Irritability, personality change, mild memory impairment, depression, psychosis and cognitive decline) and nutritional risk factors for cardiovascular diseases include hypercholesterolaemia, hypertension and obesity and increase risk of myocardial infarction and stroke. In spite of being a much more common disorder, B₁₂ deficiency recognition is delayed or missed because the manifestations are diverse in nature, affecting all the systems and organs, and is often subclinical. Anemia in B₁₂ deficiency can be very severe and often mixed deficiency anemia of B₁₂, FA, iron. Vitamin B₁₂ has also been associated with the development of age related macular degeneration (AMD) that cause vision loss and risk of frailty (muscle wasting, diminished strength) which are leading causes of disability in the elderly. During pregnancy vitamin B₁₂ or folic acid deficiency may contribute to infertility, hyperemesis gravidarum, prematurity, recurrent fetal loss and neural tube defects. [2,5]

Traditionally, vitamin B₁₂ status is assessed by its concentrations in serum. Although low serum vitamin B₁₂ concentrations are a sensitive indicator of vitamin B₁₂ deficiency and high vitamin B₁₂ concentrations generally indicate sufficiency. Interpretation of the intermediate range of vitamin B₁₂ concentrations is unclear [6]. In certain situation assessment of vitamin B₁₂ concentration is not reliable. In autoimmune etiology, vitamin B₁₂ levels can be false normal. Serum methyl malonic acid could be used for early diagnosis, even in the absence of hematological abnormalities. However, it is also not reliable in renal failure. Homocysteine levels are elevated in both B₁₂ and FA deficiencies. However, homocysteine levels are also elevated in pyridoxine deficiency, CKD, alcoholism, smoking, hypothyroidism, steroid therapy [7-9]. Hence, there is no ideal test to define deficiency and therefore the clinical condition of the patients is of the most importance. Eliciting a proper dietary history is the most important step in diagnosis. Enquire about detailed drug history, history of GI surgeries and whether patients consume fresh fruits regularly or not and regarding adequacy of protein intake.

Management of vitamin B₁₂ deficiency includes replenish body stores with six IM injections of hydroxocobalamin (1000 mcg/dose) daily or at 3-7 day intervals. Maintenance is with 1000 mcg IM every month or at least once in 3 months. Folic acid and iron supplement can be

added if required. Treat the underlying disorder causing B₁₂ deficiency. The overall long-term preventive strategy for controlling B₁₂ deficiency is to promote consumption of foods rich with vitamin B₁₂. Since the natural dietary source of B₁₂ is animal origin foods, vegetarians will always have difficulty achieving this, unless they consume eggs and dairy products or foods fortified with the vitamin. Public health measures to improve vitamin B₁₂ deficiency depend on the socioeconomic factors, cultural practices, religious and the public health policies of the local, national and international regulatory authority.

We read with interest the article by *Meenakshi N et al* conducted a cross sectional study and demonstrated the association of clinical spectrum of vitamin B₁₂ deficiency including haematological, neurological and other complications with demographic data including age, gender, socio-economic. A small sample size has been a limitation for this study and study did not provide too much information on demographical association (race, religion, socio economic status) and pregnancy association with vitamin B₁₂.

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Spectrum of Presentation of Vitamin B₁₂ Deficiency in Adult Patients in a Tertiary Care Hospital

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ABSTRACT

- Background:** Severe Vitamin B12 deficiency has a fatal outcome. There are various manifestations of the disease but not many studies of the deficiency have been carried out in India. Most of these studies are laboratory based and not clinician based. There is a need to delve into the spectrum of presentations to enhance clinician suspicion towards the deficiency and supplement patients with Vitamin B12 to reduce the symptomatology.
- Objective:** **Primary** - To elicit the spectrum of manifestations of the Vitamin B12 deficiency in an adult population in a tertiary hospital.
Secondary - To correlate demographic data including age, gender, socio-economic data and to study hematological, neurological and other complications.
- Methods:** A descriptive cross sectional study from May, 2014 to May, 2015 in Max Super Specialty Hospital, Saket, New Delhi based on laboratory values of serum Vitamin B12 <145 pg/ml and borderline <180 pg/ml requested by physicians on their clinical suspicion of vitamin B12 deficiency in 100 patients.
- Results:** Vitamin B12 deficiency was found in age group more than 60 years of age (15%) with not much gender preference (males 52% and females 48%). In this study of Vitamin B12 deficient patients, 2% subjects were lactose intolerant. 77% took milk products however, 21% did not take any milk products; 14% of the people took alcohol, 8% were occasional drinkers and 2% quit drinking, 76% of the people did not drink at all; 9% of people who smoked, 3% had left smoking, 2% were tobacco chewers and 86% did not smoke. Common manifestations associated with the deficiency included altered bowel habits (27%), peripheral neuropathy (18%), cognitive impairment (1%), dizziness (13%), heaviness sensation (1%), lethargy (3%), irritability (26%), generalized weakness (8%) and oral ulcerations (1%). Comorbid conditions associated with Vitamin B12 deficiency was Hypertension (22%), Hypothyroidism (7%), Diabetes (13%), Autoimmune disorder (5%), Migraine (1%), Hepatitis (1%), Coronary artery disease (9%) including Atrial fibrillation (2%), Rheumatic heart disease (2%), Neuropsychiatric manifestations (3%) including Depression (2%), Forgetfulness (1%), Joint involvement (8%), Worm infestation (7%), Malaria (6%), and Dyslipidemia (1%).
- Conclusions:** The deficiency was associated with a varied presentation – haematological, neurological, gastrointestinal, rheumatological, neurological, cardiological, endocrine, respiratory disorders and infections. It was more prevalent in the elderly and vegetarians but no significant correlation with socio-economic, educational status or gender was found. 2% people who were Vitamin B12 deficient were lactose intolerant. Vitamin B12 levels were lesser in people who did not take milk products and who consumed alcohol and had quit alcohol consumption. Vitamin B12 levels were less in smokers and past smokers. A significant association of combined deficiency of Vitamin B12 with Vitamin D3 was elicited. A good correlation of cognitive impairment, generalized weakness and reduced stamina was found with the deficiency. Lower Vitamin B12 levels in diabetics who were on metformin. Small sample size has been a limitation and all manifestations of Vitamin B12 deficiency found in literature review could not be elicited.

Key words: Vitamin B12, Deficiency, Spectrum, Correlation

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Introduction

Vitamin B12 is a water-soluble vitamin, required for protein, phospholipid and neurotransmitter metabolism and also for synthesis of S-adenosyl methionine [1]. The methionine synthase is essential for purine and pyrimidine synthesis. Accumulation of Methylmalonyl CoA is responsible for neurological effects. The human body can store Vitamin B12 for upto five years and its biochemical assessment is done by serum concentration [2,3]. Mostly, the deficiency is subclinical and it might take years to be apparent. In 1849, Vitamin B12 deficiency was first described and had a fatal outcome [2]. The deficiency is due to decreased or limited intake of animal foods or malabsorption which is common in elderly and secondary to gastric achlorhydria [2]. By 1926, it was found that dietary supplements e.g. liver is high in Vitamin B12 and slows the disease process [4]. The US Institute of Medicine recommends daily intake of 2.4 mcg of Vitamin B12 in adults older than 18 years of age [4, 5]. It also recommends adults over 51 years of age to have fortified foods and other supplements as there is a higher chance of gastritis [4].

Material and Methods

The study was conducted at the Max Super Specialty Hospital, Saket, New Delhi on 100 patients from May, 2014-May, 2015. This descriptive cross-sectional study was conducted to correlate the clinical spectrum of the deficiency including hematological, neurological and other complications with demographic data including age, gender, socio-economic data. Case was defined as a person with signs and symptoms of Vitamin B12 deficiency with serum Vitamin B12 <145 pg/ml and borderline if levels 145-180 pg/ml. The patients included adults more than 18 years excluding those who refused to participate in the study; who had taken Vitamin B12 supplementation

(even one dose) in the past three months and with folate deficiency, from inpatient or outpatient department. Informed consent was taken and a detailed history, clinical examination and required investigations were done and incorporated in a structured format. Sample size was calculated at 95% confidence interval and 10 % precision taking prevalence of the deficiency as 35%. All statistical analysis were done using STATA 9.0 software.

Results

The results of our study are unique as the study correlates levels of vitamin B12 with clinical spectrum of the disease. There were 94% Indians, 2% Afghans, 1% British, 1% Iraqis, 1% Russian and 1% US American. Vitamin B12 deficiency was found to be more prevalent in Hindus compared to other religion (90% Hindus, 5% Muslims, 4% Christians). The deficiency was found in 77% people who took milk products and 21% who did not take milk at all. 2% of the people were lactose intolerant. But the mean value of serum Vitamin B12 levels was lesser in those who did not take milk at all (115.14+/-34.4 pg/ml) as compared to subjects who took milk (120.5+/-34.1 pg/ml) Table 1. The mean value of serum Vitamin B12 levels was lesser in subjects who took alcohol (97.94+/-38.1 pg/ml) to those who quit alcohol consumption (119+/-32.5 pg/ml) and in non-alcoholics (122.62+/-32.8 pg/ml). Table 2. The mean value of serum Vitamin B12 levels was lower in smokers and past smokers. Table 3. Vitamin B12 deficiency in this study was associated with altered bowel habits in 27%, irritability in 26%, peripheral neuropathy in 18%, dizziness in 13%, generalized weakness in 8%, lethargy in 3%, heaviness sensation in 1%, cognitive impairment in 1%, and oral ulcerations in 1% of the studied population with other comorbid conditions. Table 5.

Table 1: Relationship of S. Vitamin B12 Deficiency with Milk Products Intake (N=100)

Milk Products Intake	Mean	N	Std. Deviation	% of N
Lactose Intolerance	125.50	2	7.778	2.0%
No	115.14	21	34.404	21.0%
Yes	120.50	77	34.125	77.0%
Total	119.47	100	33.753	100.0%

Table 2: Relationship of S. Vitamin B12 Deficiency with Alcohol Intake (N=100)

ALCOHOL INTAKE	Mean	N	Std. Deviation	% of Total N
1. Yes				
2. No				
3. Occasional				
4. Quit Alcohol Consumption				
1.	97.94	14	38.174	14.0%
2.	122.62	76	32.838	76.0%
3.	127.38	8	24.389	8.0%
4.	119.00	2	32.527	2.0%
Total	119.47	100	33.753	100.0%

Table 3: Relationship of S. Vitamin B12 Deficiency with Smoking (N=100)

Smoker/Non-Smoker	Mean	N	Std. Deviation	% of Total N
1. Smoker				
2. Non Smoker				
3. Past Smoker				
4. Tobacco				
1.	113.33	9	30.000	9.0%
2.	120.46	86	35.020	86.0%
3.	108.00	3	15.875	3.0%
4.	122.00	2	2.828	2.0%
Total	119.47	100	33.753	100.0%

Table 4: Relationship of S. Vitamin B12 Deficiency with Vitamin D3 (N=50)

VITAMIN D3	Mean	N	Std. Deviation	% of Total N
< 30.00	119.82	44	32.632	88.0%
30.00+	109.50	6	25.509	12.0%
Total	118.58	50	31.817	100.0%

Table 5: Correlation of Vitamin B12 deficiency with diseases, signs and symptoms

Diseases, signs and symptoms	Mean	N	Std. Deviation	% of total N
Cognitive impairment	107.52	10	47.509	10%
Dizziness	110.69	13	21.857	13%
Generalised weakness, lethargy and reduced stamina	111.89	19	39.809	19%
Lethargy	98.00	3	70.150	3%
Peripheral neuropathy	120.12	18	39.850	18%
Generalised discomfort	108.63	8	18.094	8%
Oral ulceration	146.00	1		1%
Loss of weight	123.00	4	36.451	4%
Hyperpigmentation	173	1		1%
Hypertension	124.86	22	124.86+/-33.592	22%
Hypothyroidism	121.14	7	28.76	7%
Diabetes	115.31	13	36.529	13%

Vitamin D3 deficiency was found in 88% subjects who were Vitamin B12 deficient i.e. 44 of the 50 who were tested for Vitamin D3. This suggests a strong correlation with Vitamin D deficiency. The results are similar to those found in literature. Table 4.

Discussion

Vitamin B12 deficiency is one of the most common vitamin deficiencies. Aparicio-Ugarriza et al in 2014 published in Clinical, Chemistry and Laboratory Medicine review of the cut-off points for the diagnosis of Vitamin B12 deficiency in the general population (6). For this study, a cut off value of 145pg/ml was taken for Vitamin B12 deficiency. Broad ranges of cut-off points for Vitamin B12 and its biomarkers were identified however there was inconsistency even in the available literature. Therefore, it is essential to establish different reference cut-offs.

This is a unique study as the spectrum of the deficiency in India has not been published so far although there are laboratory based studies showing a high prevalence of the deficiency. There was not enough literature with Indian data to analyze the symptomatology. This study gives an overview of the prevalence of the disease in India in a tertiary care set up.

The study does not provide too much information regarding demographical association (race, religion, socio-economic status) as it is not a true representation of the total population of India. According to literature, Vitamin B12 deficiency is more common in elderly (7).

2% of people above 80 years of age and 15% above 60 were Vitamin B12 deficient in this study. Therefore, it is recommended that older people should have fortified foods and supplements to meet their needs and requirements(8,9). People who did not take milk products at all were found to have lower levels of Vitamin B12 and it was more common in vegetarians (10). 2% subjects were lactose intolerant, 77% took milk products and 21% did not take any milk products at all. 14% of the people took alcohol, 8% were occasional drinkers and 2% had left drinking and 76% of the people did not drink at all. 9% smoked, 3% had quit smoking, 2% were tobacco chewers and 86% did not smoke at all.

In literature, pregnant women were deficient in serum Vitamin B12 compared to non-pregnant females (11). Nothing significant was found in the child bearing age group as only one patient was enrolled in this study. A larger sample size or a study population would definitely give a better picture of the deficiency. The number of males (52%) and females (48%) with the deficiency was almost equal and there is no gender differentiation for the deficiency in this study.

An article published in Plos One of a large cross sectional survey on Vietnamese women studied overweight and micronutrient deficiency and found a positive correlation (12). Similar results were found in this study (BMI <23 in 30.4%, between 23-25 in 20.7% and >25 in 48.9%).

Review of literature supported the presentations of Vitamin B12 deficiency with the sensory and motor nerve function(13), atrophic gastritis (14), Diabetic Retinopathy(15), hormonal contraceptives and bone mineral density(16), Alcoholics (17) etc. Supportive

investigations such as methylmalonic acid and homocysteine to confirm the diagnosis of the etiology of the diseased deficiency state can diagnose the condition better.

A small sample size has been a limitation for this study. All the presentations found in the literature review could not be elicited in my study. Therefore, a larger sample size at multisite multi hospital would highlight various presentations of the deficiency. The study had more inpatients than outpatients for enrolment as it was easier to recruit admitted hospital patients. Mass screening of Vitamin B12 should be done to illicit the prevalence of Vitamin B12 deficiency including rural and urban populations. Food fortification programs in patients of cardiovascular disease (18), elderly and pregnant females should be introduced and incorporated to decrease the prevalence of the deficiency. Different studies quote different cut off values for Vitamin B12 deficiency therefore a standard value should be derived for the deficiency.

Conclusion

This descriptive cross sectional study is unique, describing the clinical spectrum of deficiency ranging from hematological, neurological, gastrointestinal, rheumatologic, neurological, cardiological, endocrine, respiratory disorders to association with infections in a tertiary care hospital. It was prevalent in the elderly. However, no significant correlation with socio-economic and educational status or gender was found in this study. The deficiency was associated in 2% lactose intolerant people and Vitamin B12 levels was lesser in people who did not consume milk products; in subjects who consumed alcohol, who had quit alcohol and in smokers and past smokers. There was a good correlation of cognitive impairment, generalized weakness and reduced stamina. Type 11 diabetics on Metformin showed lower Vitamin B12 levels. There was a good correlation of combined deficiency of Vitamin B12 and Vitamin D3. Mass screening of people with a larger sample size, multi-hospital, multi-center approach would give a better spectrum of manifestation including rural and urban population. Food fortification programs should be introduced and incorporated and a standard cut off value for serum vitamin B12 deficiency should be derived.

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Bleeding per Rectum in Children : North Indian Experience

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ABSTRACT

Background: Bleeding per rectum in children is a common cause of hospital attendance for the paediatric patients. We tried to look for the causes of the same in a tertiary care center on out patients basis so that proper care can be instituted.

Methods: We have enrolled 211 consecutive patients in our OPD from 1- 12 years of age and tabulated the cause . We divided the patients in two groups based on their stool. Proper history taking and examination done, some patients required special investigations like endoscopy and Meckel's scan.

Results: Anal fissure (64%) was the commonest cause of rectal bleeding in preschool children although polyp, intussusception and Meckels diverticulitis play a significant role.

Conclusions: Most of the causes can be diagnosed and treated on OPD basis with proper history taking and examination of the patients, thereby we can alleviate the distress to the family.

Key words: Bleeding per rectum, Paediatric rectal bleeding, Anal fissure.

Introduction

Per rectal bleeding is relatively less common problem in children though it is very much frightening and can cause a lot of anxiety to the parents. The majority patients of rectal bleeding are due to minor causes which needs little care but at times it may indicate significant pathology which needs prompt diagnosis and care[1]. Rectal bleeding is one of the most common presenting complaints in paediatric surgical superspeciality clinic though sometimes patient present in emergency clinic with a more severe problem which requires urgent treatment. the epidemiology of this problem is not well studied till date[2-6]. In infant and younger children anal

fissure is one of the commonest cause but in older children rectal polyp is the main culprit. A careful clinical history, examination specially digital rectal examination (DRE), stool test is essential to diagnose the cause. Special investigations like radiological investigation, endoscopy and

Meckel's scan sometimes needed in special circumstances to pin point the diagnosis. The objective of this study was to analyze the causes of rectal bleeding mostly in out-patient department (OPD) basis to provide proper care.

Materials and Methods

This is a prospective study of 211 paediatric patients who attended our paediatric surgical clinic from Jan 2015 to June 2016. Some of them were referred from other peripheral hospitals where paediatric surgical superspeciality was not available. All our patients were from infant to 12 years of age. We have excluded all neonates from this study as neonatal rectal bleeding requires different line of management. We have divided

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all patients in two groups on their stool and rectal bleeding basis.

Group A –normal appearing stool coated with bright red blood and constipation related.

Group B - normal appearing stool with blood mixed .

Different causes of bleeding are summarized in table 1 & 2 . Age related distribution are shown in table 3.

Table 1: Causes of Rectal Bleeding

Causes	Number
Anal fissure	135
Proctitis	4
Rectal Prolapse	20
External hemorrhoids	3

Table 2: Causes of Rectal Bleeding

Causes	Number
Polyp	20
Nodular Lymphoid hyperplasia	4
Meckel's Diverticulum	9
Intussusceptions	16

Table 3: Causes of rectal bleeding with number and age

cause	Number	age
Anal fissure	110	1-2 YEARS
	25	3-12 YEARS
Proctitis	8-12 Years	
Rectal Prolapse	1-5 Years	
External hemorrhoids	8-12 years	
Polyp	3-5 years	
Nodular Lymphoid hyperplasia	3-5 Years	
Meckels Diverticulum	3-12 Years	
Intussuception	1-2years	

We have enrolled 162 patients in group A and 49 patient's in group B

Each and every patient undergone proper history taking and DRE on OPD basis. DRE not done in all patient's with anal fissure. Few of our patients needed proctoscopy as well as sigmoidoscopy where we suspected rectal polyp or other pathology. Some of them needed ultrasonography of abdomen where strong suspicion of intussusceptions was there and Meckel's scan was advised in some appropriate patients. We did stool test for all patients as parasitic infection is very common in our part of world. Histopathological examination of excised rectal polyp was done .

Results

This study included 211 patients of whom 150(71%) were boys and 61(29%) girls with a male to female ratio of 2.5: 1. Age range of the patients was 1-12 years with a median age at diagnosis 2 years. The mean duration of bleeding was 2 months with a range of 1 week to 2 years. Maximum number of boys presented earlier than girls. In our study, anal fissure is the most common cause (64%) of rectal bleeding over all and most of the patients were in age group of 1-2 years. Boys were affected more with anal fissure and all patient's of anal fissure responded well with conservative therapy. Rectal polyp and rectal prolapse shared the same number , 20 each. In our series all but two were single rectal polyp and DRE was the initial method of diagnosis. (fig 1). The other two patient's had multiple polyp and endoscopic polypectomy were done by gastroenterologist and doing fine. All rectal prolapse patients had some form of bowel problem either diarrhoea or constipations with excessive straining. The age group of polyp patients was 3-5 years and age group of rectal prolapse patients was 1-5 years. A good number of patients (16) had intussusceptions all of them came with bleeding per rectum and subsequently required surgery. For intussusceptions patients the age group was 1-2 years. Nine patients presented with bleeding per rectum and later diagnosed as Meckel's diverticulitis. All of them showed ectopic gastric mucosa on histopathological examination of the resected specimen but all had Meckel's scan positive. Though less in number external haemorrhoid patients came late for treatment. Another two groups of patients namely proctitis and nodular lymphoid hyperplasia patients also contributed their number this present study. These patients were treated conservatively and all improve well in times.



Fig 1: Prolapsing rectal polyps with bleeding

Discussion

Per rectal bleeding is one of the most common complaints in paediatric surgical clinic. It causes a huge anxiety and psychological stress to the parents and family. But a significant number of cases the cause is minor, needs little care and supervision to cure the patients. Sometimes simple care is not sufficient when the cause demands expertise care and intervention. Although this problem generates a lot of curiosity, its epidemiology has not been properly evaluated and studied [2-6]. Gastrointestinal (GI) bleeding originating proximal to ligament of Treitz is termed upper GI bleed and if bleeding source is distal to Treitz ligament it is called Lower GI bleed [7]. In paediatric patients lower GI bleed mostly occurs from colon and rectum though Meckel's diverticulum is part of small bowel which contributes significantly to the cause of rectal bleeding in children. Till now the largest series of rectal bleeding in paediatric population showed that only 0.3% of patients contributed out of 40000 patients presented in a tertiary care center in US². In our series we tried to find out the causes and etiology of bleeding per rectum in north Indian paediatric population who are attending our OPD directly or referred to. In this study we found anal fissure is the leading cause (64%) over all and it affects mainly 1-3 years age group. This is the majority patients in group A where we have given stress on normal appearing stool coated with bright red blood associated with constipation. The cause of bleeding was erosion and tear of anal margins due to passage of hard stool [7]. All our patients were constipated without any red flag signs of constipation [8]. On further enquiry it has been found that due to lack of health education parents were practicing bad dietary habits for their children. There was significant lack of fiber in diet and most of them were consuming lots of fast food. So we treated the patient for fissure conservatively by giving Sitz bath, local application of anesthetic gel, stool softener and high fiber diet. But to prevent recurrence side by side we started health education for proper diet habit for their children. We saw that they are the most anxious patients and parents as anal fissure causes lots of discomfort and pain associated with bleeding per rectum. After proper treatment with our protocol they are the most satisfied patients group. In group A, rectal prolapse was another cause of concern and most of them are also associated with constipation. These patients also improved with conservative treatment. Another two groups, proctitis and external haemorrhoids also had significant improvement with treatment. In contrary to group A, group B patients presented with normal appearing stool with blood mixed and they were not associated with constipation. Group B was dominated by juvenile polyp (10%) followed by intussusceptions (5%)

and Meckel's diverticulitis (8%). The cause of bleeding in this group was erosion, ulceration and thinning of the mucosal surface of the gut. In India the incidence of juvenile polyp was (61%) as quoted by Poddar et al [9]. Among rectal polyp patients only two has multiple polyp, rest were single pedunculated polyp. All polyps were send for histopathological examination to confirm the diagnosis of juvenile polyp, no dysplastic changes were seen in our study. Other authors have found dysplastic changes from 0.3% to 11% [10,11]. Four patients did not showed up any obvious cause but on endoscopy they came out to be nodular lymphoid hyperplasia and they responded well with conservative therapy. All intussusceptions patients came to us with bleeding per rectum with typical features of colicky pain in a well nourished child. The diagnosis was confirmed by ultrasonography and proper surgical intervention done. In Meckel's group of patients the history of bleeding was for longtime and it was recurrent. There history was long and had taken treatment for several months. All of them had their Meckel's scan done and was positive for it. We operated them on routine basis and histopathology showed ectopic gastric mucosa. The idea of grouping them was that it was very much convenient to diagnose them on OPD basis. Stool routine test was positive for some patients and treated them accordingly. We also gave them a course of deworming drugs who were defaulters of school deworming programme and whose stool was positive for worm.

Conclusion

Paediatric bleeding per rectum is a common but distressing cause of concern to the parents and family of the affected patients. In infant and preschool children anal fissure is the forerunner among the causes. To give a composite care to the cause every patients need to be examined thoroughly and there is no substitute for proper history taking. Special investigations do play a role for the evaluation of the cause and health education to the parents cannot be neglected.

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Arthroscopic Management of Acromio-Clavicular Dislocations in High Demand Professionals.

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ABSTRACT

Background: Acromio-clavicular joint (ACJ) dislocations are the commonly encountered injuries by the orthopaedic surgeon but the debate about the ideal management of the ACJ injuries is unresolved.

Aim: To evaluate mid-term results of functional outcome of ACJ reconstruction using the arthroscopic technique in high demand professionals.

Methods: 25 patients with ACJ dislocations who underwent ACJ reconstruction surgery using arthroscopic tightrope techniques. 23 patients meeting the inclusion criteria were evaluated while 02 were lost to follow-up. The outcome was assessed clinically, radiologically, and with the Disabilities of the Arm, Shoulder and Hand (DASH) scores preoperatively and at the final follow-up

Results: The mean age of the patients was 31.56±5.98 years (range 20-48 years). All were male. The mean follow-up was 15.26 months (range 8 - 24 months). The mean coraco-clavicular distance preoperatively was 23.9 mm (range 17- 32mm) while at the final follow-up distance was 17.54mm (range 11-22) mm. 10 patients showed loss of reduction. DASH scores improved significantly from 45.36 ± 11.06 (range, 31.13-66.66) preoperatively to 7.26±9.12 (range, 0-29.15) at the final follow-up (p-value<0.001). 82% (19 out of 23) returned to the pre-injury level.

Conclusion: Arthroscopic management of the ACJ dislocation is associated with higher implant and technical failure because of inadequate stability.

Key words: acromioclavicular joint dislocations, shoulder arthroscopy.

Introduction

Acromioclavicular joint (ACJ) dislocation are the common injuries in the high demand professionals like athletes, military personnel, manual workers etc., and ACJ injuries represent 40% to 50% of the shoulder injuries in them [1-4].

Rockwood et al classified injuries of the ACJ into six types.

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Grade I and II injuries are the low grade injuries and non-operative treatment has a good functional outcome. Grade III- VI are the high grade injuries, which require surgical intervention especially in athletes and high demand professionals [5].

There are various techniques for the management of the high grade ACJ injuries described in the literature which use different mode of fixation methods like ACJ fixation with wires, coraco-clavicular fixation using screws and ACJ reconstruction and excision of distal part of clavicle along with coraco-clavicular ligament reconstruction, especially in arthritic joint [6-11]. However, there is no consensus as far as the best treatment modality for ACJ reconstruction is concerned [1,12,13].

With the advent of shoulder arthroscopic techniques, the trend is shifting towards mini-open or arthroscopic fixation of coraco-clavicular construct using loops, flip buttons, tendon autografts. However, results of the arthroscopic technique are variable[14-20]. There have been various studies about ACJ reconstruction using the Arthroscopic technique available worldwide. The present study has evaluated mid-term results of functional outcome of ACJ reconstruction using the arthroscopic technique in high demand professionals.

Material and Methods

In the present study, patients (military personnel) with ACJ dislocation, who met the inclusion criteria were evaluated prospectively. These patients were operated with Arthroscopic ACJ fixation at our centre between December 2014 and December 2015.

Inclusion criteria

1. Age 18-60 years
2. ACJ injuries Type III, IV and V

Exclusion criteria

1. ACJ injuries Rockwood Type I, II and VI
2. Compound ACJ Injuries.
3. Other associated injuries in the same limb, cerebral trauma.
4. Systemic disease affecting the scoring process and rehabilitation.
5. Prior pain/pathology in the shoulder joint.

Twenty-three cases were studied prospectively. Patients were classified on the basis of Rockwood classification [5], out of the 23 patients, 12 were grade III, 02 were grade IV while 09 grade V. The outcome was assessed clinically, radiologically, and with the Disabilities of the Arm, Shoulder and Hand (DASH) scores²¹ at the final follow-up.

Surgical Technique

All the patients were operated under general anesthesia in the lateral decubitus. A standard posterior viewing portal was established and diagnostic shoulder arthroscopy was performed. Antero-superior working portal was established and undersurface of coracoid exposed (figure 1a). A 2-cm incision was made over the lateral clavicle, perpendicular to the superficially palpable coracoid. The drill guide was inserted through the anterosuperior portal, and the guide tip was positioned under the coracoid base. The guidewire was passed from clavicle to the coracoid. Drill hole with 4.5 mm cannulated drill bit was drilled over the guide wire (figure 1b). A sufficient bone bridge of at least 4-mm was ensured around the reamed tunnel. The guide wire was then removed, and the drill was left in situ. A nitinol suture passing wire was passed through the drill (figure 1c) and taken out through the anterosuperior portal using an arthroscopic grasper. The drill was then removed leaving the wire in position. The two white traction sutures of Acromioclavicular Tightrope [Arthrex, Naples (FL), USA] button were passed through the wire loop, and the button was then flipped to pass through the drill hole. Once the tightrope button was seen under the coracoid, the trailing suture was used to flip it, and locking it under the bone (figure 1d). The clavicle was then reduced by the assistant. The reduction was assessed clinically with palpation of the acromioclavicular joint and once satisfactory reduction was achieved, the tightrope device was knotted securely over the clavicle. The clavicular wound was closed in layers and the portals were closed in standard fashion. Sutures were removed after 10-14 days.

Postoperative Rehabilitation

Postoperatively, the shoulder was protected in a sling for

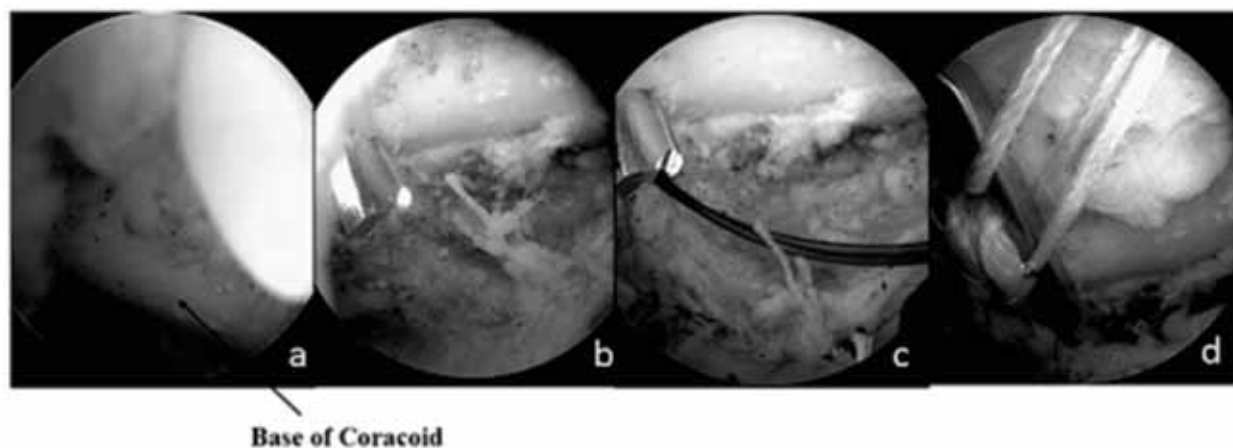


Figure 1(a-d): Arthroscopic view showing exposure of coracoid (1a), drilling of coracoid base (1b), wire passed through drill bit (1c), Tight rope at the base of coracoid (1d)

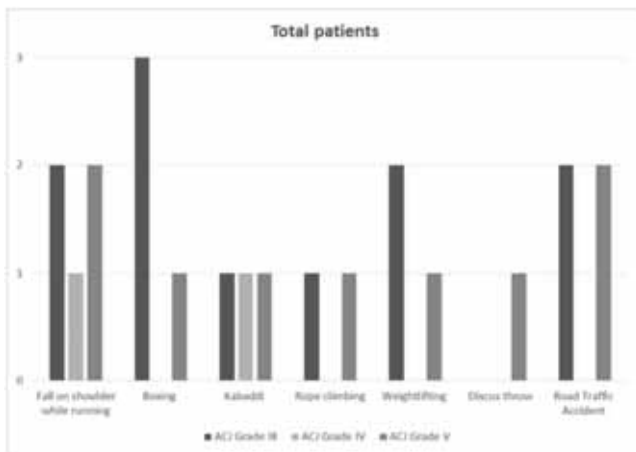


Figure 2: Mode of injuries in various ACJ grades

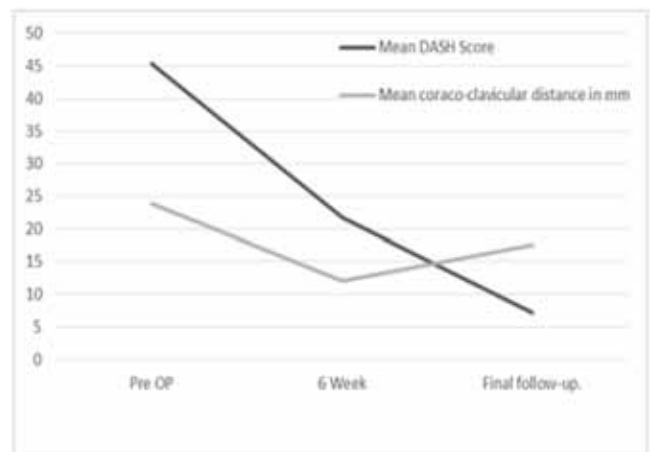


Figure 4: Mean DASH score and Coraco-Clavicular distance pre-operatively, at 06 weeks and at the final follow-up.

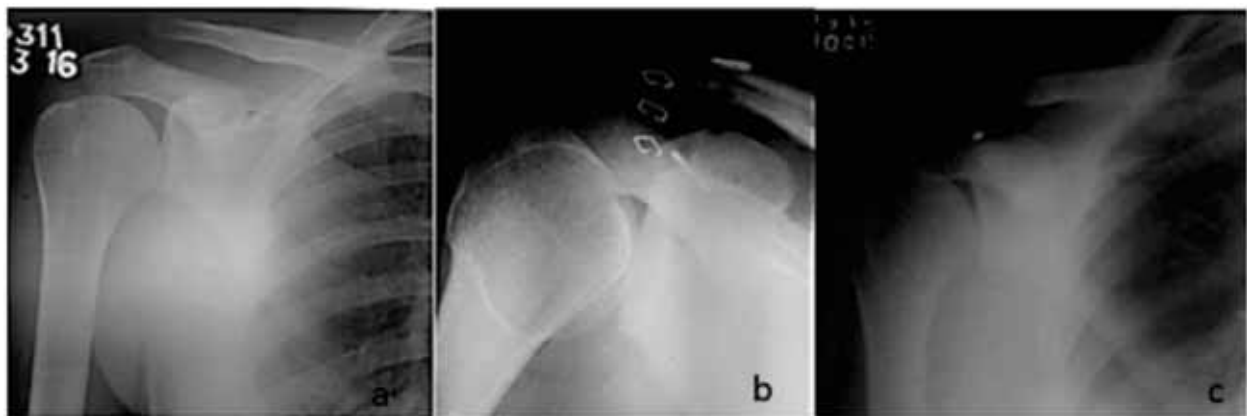


Figure 3 (a-c): Radiographs of ACJ: pre-operative (3a), post-operative (3b), implant failure at 6 months (3c)



Figure 5: Radiographs of ACJ: loss of reduction on 7th post-operative day

6 weeks. Gentle pendulum movements were started from 1st post-operative day. At 4 weeks, passive range of motion exercises of the shoulder were started, and active movements of the shoulder were advised after 06 weeks. Return to manual work or sporting activities was allowed after 16-20 weeks.

Ethical clearance and informed consent

Ethical clearance was obtained from the ethics committee of the hospital before the start of the study. Written informed consent was obtained from each patient before the conduct of the study.

RESULTS

25 patients with ACJ dislocation were operated between December 2014 and December 2015 at our centre using arthroscopic ACJ fixation, 02 were excluded from the study because of lost to follow-up. 23 patients were analyzed in this study. All were male. The mean age of the study population was 31.56 ± 5.98 years (range 20-48 years). The mean follow-up was 15.26 months (range 8 - 24 months). Right side was involved in 14 (60.87%) patients and left side was involved in 09 patients (39.13%). The dominant arm was affected in 16 cases and the non-dominant arm was affected in 7 cases. The mechanism of injury in different patients was as per figure 2. According to Rockwood classification, 12 were grade III, 02 were grade IV while 09 were grade V. The average time from initial trauma to surgery was 10.78 ± 4.60 days (range 5-19 days).

The Disabilities of the Arm, Shoulder and Hand (DASH) scores [21] was 45.36 ± 11.06 (range, 31.13-66.66) preoperatively and it improved significantly (p -value < 0.001) to 21.77 ± 14.59 (range, 0-49.17) and 7.26 ± 9.12 (range, 0-29.15) at the 6 weeks, and final follow-up evaluations respectively. Seven patients achieved DASH Score of 0.

The mean coraco-clavicular distance preoperatively was 23.9 mm (range 17- 32mm). Postoperative radiographs showed good reduction in the majority of the patients (22 out of 23)(figure 3 a,b). The coraco-clavicular distance, postoperative and at the final follow-up was 12.1 mm (range 09-14) mm and 17.54mm (range 11-22) mm respectively(figure 4).

At the final follow-up, radiological evaluation showed that 13 (56.52%) out of 23 patients had maintained. One patient demonstrated the failure of fixation in the immediate post-operative period, he also had intra-operative breakage of the drill bit (figure 5). Another failure was noticed at the time of 6 weeks follow-up, and eight patients had loss of reduction after 6 weeks(figure 3c). 06 loss of reduction were in grade III injuries patients, while 04 were grade V injuries patients.

04 out of 10 patients in whom the loss of reduction was present, were satisfied with the functional outcome and they were not willing for the revision surgery. 03 patients were not satisfied with the surgery but they were not willing for the second surgery. 03 patients were re-operated for failed ACJ reconstruction. All the re-operated patients were grade V injuries at the time of initial trauma. 02 patients were operated with hook plate while in one patient revision arthroscopic ACJ fixation was done. However majority of the patients (19 out of 23) were satisfied and returned to pre-injury level of activity at the final follow-up (Table 1).

There was no neurovascular deficit or infection postoperatively. At the time of final follow-up, four patients had tenderness at the ACJ. While four patients reported tenderness superior to the clavicular flip button which was not limiting the patient's day to day activities of living or their sporting and recreational activities. Five patients were bothered about the prominence of ACJ.

Table 1: Various grades of ACJ dislocation operated, loss of reduction, cause of failure and return to preinjury level at the final follow-up

Total Patients				
ACJ dislocation	Operated	Loss of reduction at the final follow-up	Cause of failure	Return to pre-injury level
Grade III	12	6	04 Suture breakage 02 coracoid side failure	11
Grade IV	2	0	-	2
Grade V	9	4	02 Technical failure 03 Suture breakage 01 clavicular erosion	6

Discussion

ACJ dislocations injuries are the common injuries around the shoulder joint. These injuries are much more common than reported, as certain patient with low grade injuries like grade I and II may not consult the physician. ACJ injuries are commonly seen in males in the third decade of life, especially in individuals who are involved in athletic and high demand activities [22-24]. In this series, all the patients were male who were high demand professionals as the majority of clientele visiting our centre are involved in strenuous physical activities and training.

The treatment of Grade I and II injuries is conservative in the form of a period of rest to the part, analgesics and then gradual return to pre- injury activities. While treatment of the grade III injuries is debatable, as various studies document advantages of conservative treatment [25] while other studies documenting advantages of the operative method in these injuries especially in high demand professionals [26-27]. The treatment for higher grade (IV –VI) injuries is surgical intervention. In the present study, all the patients of grade III injuries (n= 12) visiting our centre were operated, as all of them were high demand professionals and the nature of the job they were involved in.

There are various operative interventions described for the management of ACJ injuries. However, the gold standard technique for the management of injuries has not been defined. But none of these techniques has been found to be superior to other. Furthermore, these techniques have their own disadvantages. K-wires and tension band wiring commonly used for fixation of ACJ reconstruction. However, these techniques are associated with complications like increased incidence of ACJ arthritis, breakage or migration of the pins into the lung, the heart, and vessels [28-29]. The Hook Plate is commonly used for ACJ dislocation, requires a second surgery for the implant removal and can be associated with complications like fracture of the acromion, ACJ arthritis [8]. ACJ Stabilization with a coraco-clavicular construct like screw between clavicle and Coracoid [34], prevents movements between the clavicle and coracoid leading to complications like fatigue and failure of the implant, and early joint degeneration, while the transfer of coraco-acromial ligament to substitute the torn coraco-clavicular ligament as in weaver dunn procedure [33] is considered as a weak construct.

With the advancement of shoulder arthroscopic technique and knowledge about the shoulder pathologies, many newer arthroscopic or arthroscopically assisted techniques have evolved in the past decade. These

techniques are minimally invasive techniques and have advantages of providing better intra-articular visualization of the shoulder joint and its pathologies [14-19,30-32]. In the present case series, all of the patients were managed with arthroscopic ACJ fixation using tightrope.

The postoperative radiographs showed the good reduction in the majority of the patients (22 out of 23). But, at the final follow-up, 10 (43.48%) out of the 23 patients had loss of reduction. 8 out of these 10 patients had loss of reduction after 6-week follow-up. One patient demonstrated a failure of fixation in the immediate post-operative period (at 7th day) because of tunnel blowout from corocoid side probably due to the eccentric drilling of the tunnel, he also had breakage of the drill bit at the time of surgery. Another failure noticed at the 6 weeks follow-up, was also due to eccentric drilling of the tunnel. Therefore, 02 radiological failure which occurred before 06 weeks were the technical failure, while 08 delayed radiological failures which occurred after 06 weeks were implant failures. Thus, this arthroscopic technique of fixation requires a learning curve, good technical skill and precise instrumentations. Furthermore, we feel that tightrope construct though considered to have biomechanical properties similar to the native coraco-clavicular ligaments [35,36] but being of a non-biological and non-anatomical nature, this construct is a weak construct. It provides stability in superior- inferior plane and not in the anterior-posterior plane and the implant is likely to fail if the healing of the local tissue is delayed. Even the modifications of tightrope technique (use of double tightrope or a dogbone) are associated with complications like residual deformity or coracoid fracture [32-37].

Despite the high rate of failure 10 out of 23, The DASH scores improved significantly (p-value < 0.001) from preoperatively 45.36 ± 11.06 (range, 31.13-66.66) to 7.26 ± 9.12 (range, 0-29.15) at the final follow-up. These results are similar to the results of other studies which have also documented satisfactory functional outcome even though the radiological failure was significantly higher [17,35,36,38]. 19 out of the 23 patients were able to return to the pre-injury level at the final follow-up.

6 out of the 10 radiological failure were in grade III injury and 83% (5 out of 6) of these patients (grade III injuries) were able to return back to the pre-injury level of activity. Therefore, inspite of the high percentage of radiological failure in grade III injuries, the majority of the patients were able to return back to the pre-injury level of activities and they were not keen for the re-surgery for the cosmetic correction. However, 03 patients with grade V injuries at the time of initial trauma who had

radiological failure opted for the surgery. We feel that in such a scenario, the conservative treatment for the grade 3 injuries still remains a good option even in the high demand professionals

Conclusion

Arthroscopic management of the ACJ injuries using tightrope is a minimally invasive and safe technique but has higher implant and technical failure because of its non-biological and non-anatomical nature.

Conflict of interest:	All authors declare no COI
Ethics:	There is no ethical violation as it is based on voluntary anonymous interviews
Funding:	No external funding
Guarantor:	Dr Lt Col Ravindra Chauhan will act as guarantor of this article on behalf of all co-authors.

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Assessment of the Functional Outcome after Arthroscopic Mosaicplasty for Localized Osteochondral Defects in the Knee

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ABSTRACT

- Background:** Full-thickness chondral defects of weight-bearing articular surfaces of the knee are a difficult condition to treat. Our aim is to evaluate the mid- and long-term functional outcome of the treatment of osteochondral defects of the knee with autologous osteochondral transplantation with the OATS technique (Often referred to as Mosaicplasty).
- Methods:** Twenty four patients were included in this study. Twenty patients were male (Serving soldiers) and four were female with a mean age of 30.5 years (range: 21-46 years). The cause of the defect was trauma accounting for 75% , osteochondritis dessicans 16.66 % and AVN 8.33 % . The sizes of the graft used ranged from 6mm to 10mm with 33.33 % patients with size 7mm, 25% patients with 8mm. There was a preponderance towards the Right knee accounting for 75% of the total knees. The study revealed the most common site of involvement was medial femoral condyle accounting for 83.33% of all knees. Average length of stay ranged from 5 days to 10 days with 75% of patients who were discharged on the 05th postoperative day. Incidence of concomitant other injuries to the knee were 83.33%.
- Results:** Based on Tegner and Lysholm scoring scale at the end of 24 months 91.66% patients had excellent results with 8.33% patients with fair results. No correlation was found between patient age at operation, the size or site of the chondral lesion and the functional outcome.
- Conclusion:** We believe that autologous osteochondral grafting with the OATS technique is a safe and successful treatment option for focal osteochondral defects of the knee. It offers a very satisfactory functional outcome and does not compromise the patient's future options.
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Introduction

Articular cartilage is complex tissue that is able to withstand tremendous force over many cycle but does not

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have the ability to heal even after minor injury. Articular cartilage forms the load-bearing surfaces of all synovial joints. Its highly organized structure provides the biomechanical properties necessary for the tissue to withstand multiple forces created during movement. Following injury, articular cartilage has limited healing potential because the cells have minimal mitotic activity and the matrix lacks a vascular supply [1,2,3]. Advances in the treatment of cartilage defects are utilizing tissue engineering repair techniques in an attempt to regenerate and develop tissue with structural and mechanical properties similar to those of normal

cartilage. The main functions of articular cartilage in synovial joints are to provide a low-friction surface for motion and to resist tensile, shear, and compressive forces [4,5,6]. Articular cartilage varies in specific composition within the same joint and between different joints, but it consists of the same basic components and structure throughout all joints [7,8,9]. Grossly, articular cartilage appears as a smooth, homogeneous tissue approximately 2 to 5 mm thick [7]. When probed, healthy cartilage is firm and resists deformation. Diseased cartilage is soft, deforms when probed, and may contain visible surface disruptions. Articular cartilage consists of a sparse population of chondrocytes embedded within a highly hydrated extracellular matrix composed of collagen and proteoglycans. The composition of articular cartilage varies with depth from the surface, and it is divided into four structural zones. The matrix is also divided into three regions, and its composition varies with distance from the chondrocyte [5,8]. This precise arrangement of the tissue components provides specific mechanical properties for each zone [10]. Chondrocytes synthesize matrix components and regulate homeostasis of articular cartilage [9]. Articular cartilage has a limited capacity for natural healing owing to lack of blood supply, absence of chondrogenic progenitor cells, and decreased mitotic activity [11]. Cartilage injuries occur through a variety of mechanisms, including a single load of great magnitude or repetitive joint overloading of lesser magnitude [7]. Cartilage injuries have been divided into three categories based on depth of injury: (1) cell and matrix damage without visible surface changes; (2) cartilage disruption with visible fibrillations, fissures, flaps, or defects; and (3) visible cartilage and subchondral bone disruption [12].

Current surgical treatments for cartilage damage include arthroscopic débridement, microfracture, autologous chondrocyte implantation, and osteochondral transplantation [2,3]. Osteochondral autograft transplantation is the transfer of one or more cylindrical osteochondral autografts into the cartilage defect, providing a congruent hyaline cartilage covered surface. The autografts are harvested from the non-weight-bearing area of femoral articular surface like trochlea or the margin of the intercondylar notch. With a combination of different graft sizes, 90% to 100% of the defect can be filled [10]. This technique is limited by the amount of donor tissue available in the knee. Osteochondral autograft transplantation is best for small lesions (<2 cm²), but good clinical results have been reported with lesions between 2 to 4 cm² [11].

Materials and Methods

Study design

Assess the functional outcome after arthroscopic mosaicplasty for localized osteochondral defects in the knee. Twenty four cases of localised osteochondral defects were treated with arthroscopic mosaicplasty and were studied prospectively for functional outcome.

Place of study

Base Hospital Delhi Cantt

Study period

From Nov 2014 to Nov 2016.

Study population

All patients with localised osteochondral defects fulfilling the inclusion criteria of study.

Sample size

All patients of localised osteochondral defects fulfilling inclusion criteria were to be included in the study.

Inclusion criteria

1. Age: 15 to 55 years.
2. All those with localized osteochondral defects knee (MR proven or incidental detection during Arthroscopy).
3. The Patients should be skeletally mature with documented closure of growth plates.
4. The Patient who were symptomatic with difficulty in ambulation that have not been relieved by appropriate non-surgical therapies
5. The Patient who had focal, full thickness (grade III or IV) lesions on the weight bearing surface of the femoral condyle. These Patients were assessed for the quality of surrounding cartilage (minimal to absent degenerative changes in surrounding articular cartilage, Outer bridge grade II or less).
6. The Patient should be free from any systemic/localized infection.

Exclusion criteria

1. Age Less than 15 and more than 55 years.
2. Concomitant other medical illness such as malignancy, vascular insufficiency, local/systemic infections.
3. Global articular cartilage changes i.e. tri-compartmental changes.
4. Fixed flexion deformity of more than 15 degrees,

restricted range of motion of the knee.

5. Previous malunited fractures of the proximal tibia or distal femur with coronal plane deformity of more than 15-20 degrees.

Ethical clearance

Ethical clearance was obtained from institutional ethics committee of Base hospital before the start of the study. Written informed consent was obtained from each subject before the conduct of the study.

Conduct of the study

A prospective and observational study was conducted by identifying patients attending the Orthopedic OPD at Base Hospital, Delhi Cantt who gave consent for participation in the present study.

The Patients were thus admitted, and they underwent preoperative evaluations which included a detailed history taking and physical examination of the affected knee joint with regard to joint line tenderness, range of motion, laxity of the joint. A preoperative radiological survey in the form plain radiographs, MRI and preoperative investigational protocol for assessment of pre-anesthesia check was performed. Detailed history regarding co-morbidities was documented and patients with co-morbidities were duly excluded.

All the patients with localized osteochondral defects were treated with arthroscopic mosaicplasty in this study between November 2015 to Nov 2017 at Base Hospital, Delhi Cantt.

The cases were followed up for a minimum period of 1 year to 02 yrs and are still on follow up. All surgeries were performed by the orthopedic surgeons on the posted strength of Base Hospital Delhi Cantt.

Surgical technique

Surgical technique involved standard arthroscopic protocols. Preoperative antibiotics were given at the time of induction of anesthesia. Patient were prepared for surgery as per standard International norms. Anterolateral portal was made 1cm below the lower pole of patella and 1cm lateral to the patellar tendon.

A diagnostic arthroscopy was done starting from the suprapatellar pouch to the medial femoral compartment, intercandylar notch was scoped for assessing the integrity of ACL/PCL and rule out tears.

Superficial defects not going up to the subchondral bone were treated with abrasion chondroplasty and micro fracturing. Meniscal tears were dealt according to tear patterns and geography of tears with either repair/

excision and balancing.

The localized osteochondral defects more than 5 mm in size where the subchondral bone was exposed were treated with mosaicplasty. The graft was harvested arthroscopically from the non articulating part of the femoral condyles either medial/lateral (mostly lateral femoral condyles) using a harvester (we used the Mosaicplasty kit of Arthrex). Graft diameter of size equal to the defect measured was harvested. The usual sizes of the osteochondral defects was 6-10 mm in diameter. The larger defects required the insertion of 02 osteochondral plugs (in two cases) and sometimes even 03 osteochondral plugs (in one case only). The depth of the defect was approximately 15mm as measured by the harvester which has markings that are visible arthroscopically.

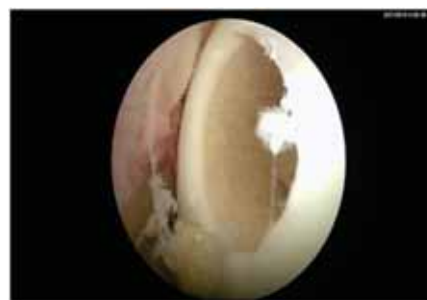


Figure 1: Depicts the recipient site after it has been prepared for grafting

Once the graft is harvested the recipient area is prepared by creating a trough by an equipment which is similar in shape to the harvester but is 1 mm less in diameter. The depth of the trough created is also 2 mm less than that of the graft which is done so that the implanted graft sits flush with the native articular surface. The graft is placed in the recipient trough with help of plastic cannula which is conical on the front for easy passage into the defect. Once implanted the graft is checked for stability using a probe.

Routine arthroscopic procedure of ACL/PCL reconstruction were carried out either prior to following

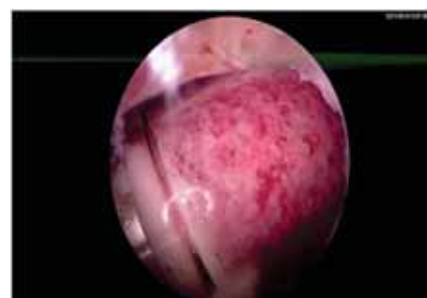


Figure 2: Shows the introduction of the osteochondral peg into the defect



Figure 3: Depicts the final placement of the graft.

a mosaicplasty. Before closure the extracted part of the diseased osteochondral bone was swapped into the donor area. Joint was lavaged and closed. Compressions dressing were done thereafter.

Rehabilitation Protocol and Followup

Post operatively these patients were not allowed to bear weight on the affected limb for 06-08 weeks although range of motion exercises and isometric quadriceps and hamstring exercises were initiated from day 1 of the procedure. Partial weight bearing at 06-08 weeks time was started and the patients were encouraged to bear full weight by 12 weeks from surgery.

Follow up of these patients was done according to **Tegner and Lysholm scoring scale**. Follow up was done at 03 months, 06 months, 01 year and 02 years from the time of surgery and results were documented and data was interpreted.

It was planned that the patient would be rescoped at 06 months (for a relook arthroscopy) for academically documenting the take up of the graft but consent for the above procedure was not given by most patients as it was purely an academic exercise which was explained to the patients. Hence the assessment of the procedure will mostly be by the knee pain score.

Results

All data from cases was collected and compiled. Data was studied in references of gender distribution, Age Distribution, Mode of Injury, Size of the defect (as assessed by the graft size used to treat the defect), Laterality, site of involvement, Average Hospital Stay, Type of defect (based on Outerbridge classification), incidence and types of intra-articular pathology, associated ligament injuries and meniscal pathologies and post operative complications were tabulated. Outcome measures were studied using **Tegner and Lysholm scoring scale** and the outcome was graded as excellent, good, fair and poor at 06, 12 and 24 months.

Gender distribution

In our study we observed a male preponderance with 83.33 percent out of a total of 24 patients.

Age Distribution

Most of the patients in our study were in the age group of 21- 46 yrs with an average age of 30.5 yrs.

Mode of Injury

In our study most of the osteochondral defects were sustained due to trauma in which road traffic accidents accounted for 50% and military training activities accounted for 25% of all injuries. 16.66 % of osteochondral defects were because of osteochondritis dessicans, and 8.33 % of osteochondral defects were because of AVN.

Size of the defect (as assessed by the graft size used to treat the defect)

The sizes of the graft used ranged from 6mm to 10mm with 8.33 % patients treated with a single osteochondral plug of size 6mm, 33.33 % patients with size 7mm, 25% patients with 8mm, 16.66 % with size 9mm, 8.33% with size 10mm and 8.33% with multiple pegs of different sizes respectively (one patient was treated with 02 pegs and 01 with 03 pegs of assorted sizes).

Side of involvement/ Laterality

In our study there was a preponderance towards the Right knee accounting for 75% of the total knees.

Site of Involvement

Our study revealed the most common site of involvement was medial femoral condyle accounting for 83.33% followed by the lateral condyle which accounted for 16.66% of all knees.

Average hospital stay

In our study the average length of stay ranged from 5 days to 10 days with 75% patients who were discharged on the 05th postoperative day and 25 % patients who were kept for a maximum of 10 days due to effusion and observation for ruling out infection of the operative wounds.

Incidence and types of intra-articular pathology, associated ligament injuries and meniscal pathologies

In our study 50% of patients had concomitant ACL tears, 33.33% patients had meniscal tears and 16.66% patients had isolated osteochondral injuries.

Final Assessment

Based on Tegner and Lysholm scoring scale at 06 months 66.66 % patients had excellent result, 25% patients had good result and 8.33 % patients had fair results respectively. At the end of 12 months 75% patients had excellent results with 16.66 % with good results and 8.33% patients with fair results. At the end of 24 months 91.66% patients had excellent results with 8.33% patients with fair results.

Discussion

Treatment recommendation for articular cartilage injury and arthritis includes nonoperative and operative management. Non operative treatments involves decreasing the load of joint by having the patients lose weight, alter activities and strengthen muscles across the joint may help to absorb some of the load. Orthoses or brace also are beneficial, as are analgesics and anti-inflammatory medications. Operative treatment is generally indicated if non operative treatment fails to relieve pain and mechanical symptoms. Treatment options includes visco-supplementation, marrow stimulation i.e. Intraarticular Injection of PRP, transplantation to fill the defect (Autologous Chondrocyte implantation) which is a cell based therapy and use of growth factor of Pharmacological agents. The choice of procedure is based primarily on size of the lesion and the activity demand of the patients

Osteochondral autograft plug transfer (OAT) involves the direct transplantation of osteochondral segments from less loaded regions of cartilage to areas with symptomatic focal defects [15]. Despite concerns of donor site morbidity and limited availability, this method has been demonstrated to reliably restore native hyaline cartilage architecture and the underlying subchondral bone.

Yamashita et al in 1985 described two patients who underwent graft harvest from the superomedial femoral trochlea in a region which "in extension was in contact with neither patella nor meniscus." Donor sites were filled with iliac crest bone graft, and all segments were fixed using orthogonal mini-cancellous screws [16]. Second-look arthrotomy for screw removal revealed macroscopically intact hyaline cartilage with mild irregularities at the interface between graft and native tissue. Slight surface contour irregularities were noted at both donor and graft sites, but with negligible clinical sequelae. Outerbridge et al in 1995 described the transfer of an osteochondral graft from the lateral facet of the patella to repair a large osteochondral defect in the ipsilateral femoral condyle in 10 patients. A manual press-fit technique was used for graft fixation [17].

Preoperative and postoperative function was assessed using the Cincinnati Knee Score; an average improvement from 43 points (range, 24 to 64) to 93 points (range, 79 to 100) was reported. All patients were satisfied with the procedure, and 70% were able to resume full, unrestricted activity. Second-look arthroscopy revealed solid graft fixation and intact surface hyaline cartilage.

Numerous treatment algorithms have been proposed for the management of articular cartilage lesions. Smaller lesions (<10 mm) are typically managed with simple débridement, citing the limited increase in biomechanical loading and rim stresses around the edges of the defect [18,19,20]. However, more recent studies have raised concerns about the durability of fibrous/fibrocartilaginous repair tissue [21,22]. Larger lesions (surface area >200 mm²) and those with defects extending into subchondral bone represent a more complex scenario [15]. However in our study we have used osteochondral bone plugs for osteochondral defects where subchondral bone was exposed even for lesions of size less than 10mm.

Fixation of a loose osteochondral fragment with a bone peg or with tissue adhesive was studied along with comparison of fresh-frozen allografts versus autogenous grafts harvested from the lateral femoral condyle [23]. The authors concluded that precise reconstruction of the articular surface was essential because failures were seen in cases where restoration of joint surface congruity was inadequate. The osteochondral plugs harvested are slightly larger than the defect allowing a press fit implant that will stay in place without the need for additional fixation [10,14]. Ivănescu A et al (Between January 2009 and June 2010) performed 55 transchondral drillings and 10 mosaicplasties on patients with articular cartilage defects of the knee. All patients were followed up at 6 months. Hughston clinical and radiological scales were used to evaluate the patients in the transchondral drilling group. Results: The Hughston Clinic score was 2 in 2 cases (3.6%), 3 in 5 cases (9.9%) and 4 in 48 cases (86.5%), giving over 95% of good results. The Hughston radiological score was 2 in one case (2%), 3 in 4 cases (7.3%) and 4 in 50 cases (90.7%). In another study by the author in 2010 evaluated if mosaicplasty is effective in returning elite athletes to participation in sports. The results of mosaicplasty were prospectively evaluated at 6 weeks, 3 months, 6 months, and yearly in 354 patients. Good to excellent results were found in 91% of femoral mosaicplasties, 86% of tibial, and 74% of patellofemoral; 92% of talar mosaicplasties had similar results. The investigators concluded that despite a higher rate of preoperative osteoarthritic changes in the athletic patients, clinical outcomes of mosaicplasty in this group

Table: Comparison of various recent studies with our study carried out at our centre

Authors	Number	Age, Yr	Follow-up	Scoring System	Subjective
Braun Arthritis Res Ther 2008	33 (23M, 10F)	15-59 (34.3)	46-98 mo (66.4)	Lysholm 12-79 (49) >40-100 (86)	27/33 return to sports 31/33 satisfied, would redo
Duany Arch Orthop Trauma Surg 2009	9 (5M, 4F)	18-74 (43.4)	11-120 mo (42.1)	KSS 39-75 (57.9) >43-100 (80.2)	88.9% survivorship (1 conversion to TKA)
Hangody Injury 2008	967			HSS, Cincinnati, Lysholm, ICRS	Femoral 92% excellent/good, tibial 87% excellent/good, troch/patella 74% excellent/good
Marcacci AJSM 2007	30 (22M, 8F)	17-46 (29.3)	7 yr	IKDC 7A, 16B, 4C, 3D at 7 yr; 11A, 12B, 4C, 3D at 2 yr; IKDC subjective 34.8 >71.7	2 yr: 22 return to sports same level, 4 return to sports lower level; 7 yr: 7 same level, 14 lower level
Tetta Eur J Radiol 2009	24 (17M, 7F)	29.9 ± 8.7	96-125 mo (113)	IKDC 31 >82; CSE/ICRS 15C, 9D >7A, 12B, 4C, 1D; Tegner 3 >6	25% return to sports at same preinjury level, 54.2% lower level, 20.8% no sports
Our study	24 (20M,4F)	30.5 (21-46)	06-24 Mo	Tegner and Lysholm scoring scale	91.66% patients had excellent results, 8.33% patients fair results.

demonstrated a success rate similar to that of less athletic patients.

The table given below gives a comparison of various recent studies with the study carried out at our centre with equivocal results.

Conclusion

To conclude the advantage of osteochondral grafting is the implantation of hyaline cartilage without the need for sutures or adhesive, while the disadvantage is the lack of lateral integration with the surrounding cartilage. This technique can be used for both small and medium sized lesions. The donor plugs can also be of variable size to allow for complete fill of the injured defect. The space between the hyaline graft plugs is filled with fibrocartilage and the donor holes are filled with cancellous bone that incorporates quickly which is then also covered by fibrocartilage [24]. It has been shown that mosaicplasty results in good to excellent clinical outcome scores in 92 % of patients depending on the location and size of the implanted tissue. Although a promising surgical procedure, there is potential for donor-site morbidity

(even though the grafts are taken from the non-weight bearing part of the knee)[25]. Further limitations include difficulty in matching the contour of the host cartilage and marginal cell death that can precipitate graft degeneration and failure [26].

Osteochondral grafting is a one stage procedure which can be done with ease and has promising results.

Conflict of interest:	All authors declare no COI
Ethics:	There is no ethical violation as it is based on voluntary anonymous interviews
Funding:	No external funding
Guarantor:	Prof. Ravindra Chauhan will act as guarantor of this article on behalf of all co-authors.

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Assessment of the Functional Outcome of Arthroscopic Assisted Reduction and Percutaneous/ Open Internal Fixation of Tibial Plateau Fractures.

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ABSTRACT

Background: Tibial plateau fractures are complex injuries which require anatomical reduction of bony injuries along with management of associated ligamentous and meniscus injuries. The outcome depends not only on personality of initial injury but on management of fracture and associated injuries also. ORIF is the gold standard for treatment of tibial plateau fractures, but arthroscopic assisted fixation provides a viable alternative for Schatzker type I- IV tibial plateau fractures, as evidenced in this study.

Methods: A prospective study was conducted for assessment of the functional outcome of arthroscopic assisted reduction and percutaneous /open internal fixation of tibial plateau fractures. Twenty four cases of arthroscopic assisted reduction and percutaneous /internal fixation of tibial plateau fractures were studied prospectively for functional outcome. Following diagnostic round of arthroscopy, reduction of fracture was done and fractures were fixed as per case merits. No more than 2 mm articular step off was accepted. Fixation methods included percutaneous 6.5 mm CCS, or ORIF with 6.5 mm PTCCS/proximal lateral tibial anatomical plate/medial buttress plating. Following this intra-articular pathologies were addressed. All peripheral reparable meniscal tears were repaired with inside out/outside in technique. Unreparable meniscal tears were excised or debrided. Cruciate ligaments tear were not repaired/reconstructed acutely. Collateral ligaments were repaired acutely. Functional outcomes were assessed based on American knee society score [2] (AKSS).

Results: The mean followup was 06 months. Final assessment based on AKSS done and objective score at 06 month turned out to have 75% patients with excellent result, however 88 % patients have excellent functional score at 06 months. Only 01 patient has poor objective score and none has poor functional score table.

Conclusion: Arthroscopic assisted fixation include direct visualization of articular surface for perfectness of reduction and single stage management of associated ligamentous and meniscal injuries. Arthroscopy allows for visualisation of accurate fracture reduction while obviating the need for extensive operative exposure. In some regards, arthroscopy narrows the gap between the extremes of open versus nonoperative management. The arthroscopy allows for evacuation of hemarthrosis and any fracture debris. Arthroscopy may offer the advantages of more rapid recovery, reduced pain, early full range-of-motion, improved fracture healing, and more complete and functional recovery.

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Introduction

Our life in the present time, driven by pace in industrialization, urbanization and mechanization, leads to an increased number of high energy traumatic incidents. Crowded cities, irregular traffic arrangements, and, fast moving vehicles are the most important contributory factors causing polytrauma with associated

bony injuries particularly comminuted fractures and significant soft tissue injury. Fractures of the tibial plateau are a common association amongst such injury patterns. The incidence of tibial plateau fractures is ever increasing due to exponential rise in motor vehicular accidents. Fractures of tibial plateau are serious injuries that frequently result in significant functional impairment. Today, the first challenge in the management of tibial plateau fractures is to decide between non-operative or surgical treatment [1]. Fractures that are stable and are minimally displaced may be amenable to cast immobilization. Other indications for non-operative treatment may include injuries to the peripheral (submeniscal) rim of the plateau and fractures in elderly, low-demand, or osteoporotic patient [2]. The emphasis in treating displaced articular fractures is an anatomical reconstruction of articular surface, restoration of the soft tissue envelope and rigid internal fixation to obtain a stable painless articulation with normal range of motion. Historically, conservative treatment mostly yielded poor results. Surgical intervention for this fracture is currently the mainstay of treatment. Open reduction and internal fixation of complex fracture patterns are often complicated by a compromised soft tissue envelope and suboptimal address of intra-articular pathology. Also, excessive dissection may impair vascularity of the fracture fragments and increase risk of infection [5]. Arthroscopically assisted percutaneous fixation, which was first recommended by Caspari [7] and Jennings [3] has gradually become popular since its initial use as a diagnostic tool. Arthroscopic assisted internal fixation may represent a viable alternative to open surgery and may reduce morbidity associated with fracture repair [8,9]. Arthroscopy is minimally invasive in comparison to ORIF [3]. The entire articular surface may be visualized without the extensive dissection required for traditional ORIF [3]. The arthroscope allows for evacuation of hemarthrosis and any fracture debris [10]. In addition, arthroscopic treatment of meniscal and ligamentous injuries is often superior to repair or reconstruction using larger, open incisions [8,4]. Arthroscopy may offer the advantages of more rapid recovery, reduced pain, early full range-of-motion, improved fracture healing, and more complete and functional recovery [1,3,4]. It is acknowledged that percutaneous lag or buttress screws, percutaneous plates, or even open buttress plating may be required in such cases, and arthroscopic assisted internal fixation is specifically defined as a surgery where anatomic reduction and rigid internal fixation is achieved without (a large or submeniscal) arthrotomy [7]. Recently there has been an interest in the arthroscopic assisted method which has been tried in Schatzker I - IV type of fractures by a different authors. The method is

still evolving and under evaluation. This requires a high degree of arthroscopic skill and familiarity with minimally invasive fracture fixation techniques. At our institution over 800 arthroscopic procedures are done annually. This study was conceived as our ever increasing use of the arthroscope, allows us to extend these skills to more and more complex problems.

Potential disadvantages of arthroscopic assisted internal fixation or arthroscopically assisted ORIF of tibial plateau fractures require consideration. The rate of complication related to arthroscopic surgery is reported to be between 1% and 8%. These are either intraoperative complications such as vascular or neurological injury, articular cartilage injury, and those due to broken instruments or are early postoperative complications as hemarthrosis, deep vein thrombosis, infection, compartment syndrome, and, loss of correction [6, 11].

Materials and Methods

Study design: A prospective study was conducted for assessment of the functional outcome of arthroscopic assisted reduction and percutaneous /open internal fixation of tibial plateau fractures. Twenty Four cases of arthroscopic assisted reduction and percutaneous / internal fixation of tibial plateau fractures were studied prospectively for functional outcome.

Place of study: Base Hospital Delhi Cantt

Study period: From NOV 2013 to NOV 2015.

Study population: All patients with tibial plateau fracture fulfilling the inclusion criteria of study.

Sample size: All patients of tibial plateau fracture fulfilling inclusion criteria were to be included in the study. Based on previous year records from the statistical department of this hospital average amounted to be around 12 per year.

Inclusion criteria:

1. Age < 60
2. Tibial Plateau Fractures Schatzker Type I, II, III and IV

Exclusion criteria:

1. Age > 60
2. Tibial Plateau Fractures Schatzker Type V and VI
3. Open fracture
4. Pathological fracture
5. Severe head injury (initial Glasgow coma scale score of < 8) and
6. Severe systemic illness (active cancer, chemotherapy, hemophilia, or a medical contraindication for surgery).

Ethical clearance: Ethical clearance was obtained from institutional ethics committee of the hospital before start of study. Written informed consent was obtained from each subject before the conduct of the study.

Conduct of the study

All cases on presentation to the emergency department were seen. The history was taken, followed by general and local examination of the patient to determine the mechanism of trauma, status of soft tissue envelope and neurovascular competence. The affected limb was initially immobilized in an above-knee POP slab with elevation, or put in calcaneal traction on a Bohler-Brown splint depending upon the condition of the soft tissues. Concerned specialists undertook appropriate management of the associated injuries. Intensive care was given to those patients who presented with shock and immediate resuscitative measures were taken as necessitated. All patients underwent radiological plain-film study in anteroposterior and lateral views as well as computerized tomography with 3 D reconstruction of the knee prior to surgical intervention.

Surgical technique: After detailed preoperative evaluation and imaging studies, like Radiographs, CT scan with 3-D reconstruction, the fracture geometry was carefully studied. The operative planning included the need for and marking the cortical window, necessity for bone graft/substitute and the necessary internal fixation method that was tailored for each case. The treatment method was based on the type of fracture, the amount of displacement and the amount of depression of the tibial plateau. The patients were taken for surgery at the earliest possible time depending on their medical condition, skin condition and the amount of swelling. All surgeries were done by arthroscopic assisted joint visualisation and under C-arm image intensifier control. We set the patient in supine position on fluoroscopic table with involved leg hanging from side of table whenever required. Prophylactic antibiotics were routinely administered with the induction of anaesthesia as per departmental protocols. Tourniquet was applied and confirmation of pre-operative antibiotics dose done. Surgical check list was confirmed before commencing the surgery.

The standard anterolateral port was used to drain the hemarthrosis which was followed by joint lavage to enable visualisation. An anteromedial port was established to further probe the joint for intra-articular pathology. We didn't use arthropump, instead we use gravity method for fear of compartment syndrome.

Following diagnostic round of arthroscopy, reduction of fracture was done and fractures were fixed as per case

merits. No more than 2 mm articular step off was accepted. Fixation methods included percutaneous 6.5 mm CCS, or ORIF with 6.5 mm PTCCS/proximal lateral tibial anatomical plate/medial buttress plating. Following this intra-articular pathologies were addressed. All peripheral reparable meniscal tears were repaired with inside out/outside in or all inside technique. Unreparable meniscal tears were excised or debrided. Cruciate ligaments tear were not repaired/reconstructed acutely. Collateral ligaments were repaired acutely.

Type 1 Fractures: Closed manipulation with help of ligamentotaxis and reduction clamps; joystick technique if needed. After confirmation of reduction with arthroscopy and fluoroscopy these were fixed with 6.5 mm PTCCS.



Use of a large forceps for fracture reduction and fixation



Arthroscopic view after reduction

Principle of fracture reduction via ligament traction: The traction exerted on the bone structures elevates the fragment.

Type 3 Fractures: Anterior cruciate ligament tibial guide was used to localize the center of the depressed articular surface and a cortical window was created to elevate these depressed fractures with the help of a tamp under direct arthroscopic visualization. The defect was filled with autograft/synthetic bone graft and elevated fracture fragments rafted with 6.5 mm PTCCS/proximal lateral tibial anatomical plate.

Type 2 Fractures: A combination of both of the above mentioned techniques was used for dealing with these injuries.

Type 4 Fractures: Closed manipulation with help of reduction clamps, joystick technique and percutaneous

pinning were usually attempted. Sometime such fractures required an ORIF using medial or posteromedial approach to reduce the fracture fragments. After confirmation of reduction with arthroscopy and fluoroscopy these were fixed with 6.5 mm PTCCS/ medial buttress or anti-glide plating.

Post operative protocol : Postoperative X-rays were usually done on the first post-op day and repeated at 6 weeks and 6 months or whenever felt necessary to assess progress until union.

Postoperative Rehabilitation

1. **FIRST WEEK:** Patients were encouraged to do isometric quadriceps and hamstrings exercises starting on the first postoperative day. ROM exercises of hip, ankle, foot were also part of the early rehabilitation program. CPM was used if the patient was not able to follow this protocol.
2. **SECOND WEEK – FOURTH WEEK:** Non-weight bearing ambulation with the help of walker or crutches was encouraged.
3. **SECOND MONTH:** The patient was allowed partial weight bearing ambulation as tolerated with crutches or a walking stick.
4. Full Weight bearing was only allowed after radiological consolidation of the fracture.

Functional Outcome: Functional outcomes were assessed based on American knee society score1 (AKSS). Radiological union was also correlated with results of functional outcome.

Results

All data from cases was collected and compiled. Data was studied in references of gender distribution, Age Distribution, Mode of Injury, Laterality, Average Hospital Stay, Type of fracture (based on Schatzker's classification), incidence and type of intra-articular pathology, Complications, Objective score based on AKSS at 6 months and functional score based on AKSS at 6 months.

In our study we observed a male preponderance with 87.5 percent male patient. Out of total 24 patients only 03 patients were females.

Most of the patients in our study were in the age group of 31- 40 yrs and constituted 58.33 % of the study group. The next common age range was <30 yrs with 25% patients. Most of the fractures in our study were sustained due to road traffic accidents (50%) and military training activities (21%). Other modes of injuries were fall from height, sport related activities and injury due to assault.

Side of involvement: in our study amounts to almost

equal with slight left preponderance.

Average hospital stay in this study was 5- 10 days for 63% patients. Only 02 patients were stayed more than 03 weeks who developed post op complications. According to Schatzker classification, type I fractures (42 %) and type II fracture (29%) constitutes maximum patients (71%) of this study. Out of 24 patients of this study, 10 patients (41%) had intra-articular pathology. Amongst them lateral meniscus tears were the commonest (25%) pathology. It was observed on association of different types of intra-articular pathology with fracture pattern, that type I and III Schatzker type fractures have only lateral meniscus injuries (3 and 1 respectively) type II have lateral meniscus tear and ACL tear and type IV have medial meniscus tear and ACL tear. Complications were not encountered frequently in this series. Only 02 cases had significant complication. One had deep vein thrombosis and other had compartment syndrome.

Final assessment based on AKSS done and objective score at 06 month turned out to have 75% patients with excellent result, however 88 % patients have excellent functional score at 06 months . Only 01 patient has poor objective score and none has poor functional score table.

Discussion

This study was carried out to assess the functional outcome of 24 cases of tibial plateau fractures managed with arthroscopic assisted fixation. The analysis of results were made in terms of age of patients, gender distribution, mode of injury, laterality of fracture, type of fracture, intra-articular pathologies, complications and functional outcome.

In our study 21 out of 24 patients were male (87.5%). This was partly because this was an army hospital. Its clientele has a male dominance and partly it may be attributed to more outdoor training activities. Albuquerque et al⁶⁸ also found a male dominance in their series with 70.3% male. However the study done by Schulak Gunn¹² shows no gender difference.

In our study the mean age of the patients was 35 and maximum patients were in age group of 30-40yrs (58.33%). The next group was 20-29yrs (25%). According to Albuquerque RP et al [13] the mean age was 44.5±14.4 with maximum patients belonging to age group 40-49yrs, and the next most frequent group was 30-39 yrs.

In our study the most common mode of injury was road traffic injuries (50%). The next most common etiological association was related to training activities which includes both sports and army related training (37.5%). Other modes of injuries were fall from height (02 cases)

and assault (01 case). Similar distribution of mode of injuries was observed in other studies [13,15] also. However in a study of Hung et al [16] 97% injuries were sustained during traffic accidents.

The left tibia was slightly more prone to get injured as observed in our study. 13 cases were left sided and 11 were right sided. Left side prominence is also observed in the study by Albuquerque et al [13]. This possibly is just an observation common to our study also.

In our study, the average hospital stay was 12 days with most patients being discharged within 2 weeks of injury. 2 patients were required to stay longer in hospital for treatment of related complications i.e. compartment syndrome and deep vein thrombosis. They stayed for 24 days and 22 days respectively.

Out of four types of Schatzker fractures included in our study, type I (42%) and type II (29%) were more common in comparison to type III (12%) and type IV (17%). In the study done by Albuquerque et al Type II (35.1%) was the commonest fracture. Mehmet Asik et al [17] also show dominance of type II fracture in their study.

One of the biggest advantages of arthroscopic assisted fixation is to diagnose and treat the associated intra-articular pathology. In our study 06(25%) lateral meniscus tears were observed. Out of them 04 were amenable to repair, being peripheral tears, and 02 were managed with partial meniscectomy as they were irreparable. Out of these 06 lateral meniscus tears, 03 were associated with type I fracture, 02 with Type II fracture and 01 with type III fracture. 02(8%) medial meniscus tear were also observed associated with type IV fracture, both were managed with meniscus repair. 02(8%) ACL tears were also observed which were left unattended as they were bony avulsion type tears. Total intra-articular lesions were 41% in our study.

The frequency of intra-articular lesion varies widely in various studies. Abdel-Hamid MZ et al [18] reported 71% soft tissue lesion associated with tibial plateau fractures out of which 57% were meniscal injuries, 25% ACL injuries, 5% PCL injuries 3% MCL and LCL each and 1% peroneal nerve injury. No significant association was noted between fracture type and incidence of meniscus, PCL, LCL, MCL, artery, or, nerve injury.

In a study by Vangness CT et al [74], intra-articular lesions were reported to be around 47%. 56 % intra-articular lesions were reported by Bennett WF [19] .

T. Scheerlinck et al [20] have found 53.8% incidence of intra-articular lesions in which the lateral meniscus was most commonly injured. On follow-up they observed that such injuries did not seem to play a major role in the final functional outcome of the patients reviewed. On the other

end of the spectrum only 2% intra-articular lesion were reported by Holzach et al [14,20].

With regards to complications, we observed 02 cases of loss of reduction, identified in the immediate post-op period. One of them was managed by repeat arthroscopic assisted fixation while another was tackled with ORIF+PLATING after a week. Post op they performed well. One case of deep vein thrombosis (4%) was also identified and managed successfully with anticoagulants for 03 months as per recent guidelines by ACP [21] initially with 60 mg BD subcutaneous LMWH for 14 days and subsequently with Tab Warfarin dosage adjusted to maintain INR at 2-3.

One case also developed a presumed compartment syndrome as evidenced by a tight and swollen calf towards the end of the fracture fixation (4%). He was managed with two incision fasciotomy at the conclusion of fracture fixation. The possible reason was thought to be a combination of initial injury and early surgery as this patient was operated on third day of injury. In retrospect it was felt that his soft tissue injury might have been incorrectly assessed following the trauma. Though this was an observation made in retrospect, Belanger M et al [22] in 1997 reported a case report of compartment syndrome of the leg after arthroscopic examination of a tibial plateau fracture. Chang YH et al [23] in 2000 observed 10.3% incidence of compartment syndrome in tibial plateau fractures in Taiwan. However in other studies compartment syndrome was not found to be a complication.

02 patients were also observed to have varus instability at 6 weeks follow-up, which was found to be stable at review after 06 months.

Final outcomes were made on the basis of American knee society score.

Objective score at 06 months showed that 18 patients (75%) scored excellent, 02 patients (8%) scored good, 02 patients (8%) scored fair and 01(4%) patient scored poor results.

Functional scores at 06 months indicated that 20 patients (83%) scored excellent, 01 patient (4%) scored good, 02 patients (8%) scored fair. These results are comparable to available studies in literature.

Itokazu and Matsunaga[24] in 1993 reported a series of 20 diverse cases, (7 type I, 9 type II, 1 type III, 2 type IV, and 1 Type V fracture), and observed that all patients scored good to excellent results based on Rasmussen criteria. Superior results were noted in type I and II fractures which commensurate with our study results.

Holzach et al [21] in 1994 reported a series of 16 patients.

01 patient was lost to follow-up and 14 patients out of 15 were rated excellent using the Davos knee scoring system.

Cassard et al[8] in 1999 treated 26 patients arthroscopically for tibial plateau fractures that included follow-up at 32.7 months for 19 cases. The mean Knee Society Score (KSS) score was 94.1 for pain and 94.7 for function. Two patients had early radiographic signs of osteoarthritis, and 2 had valgus deviation. There was no cases of late bony collapse in their study. The authors concluded that results of arthroscopic management were as good as or better than what might be expected from ORIF.

Ohdera et al[24] in 2003 performed a comparative study between ORIF and AAIF. Comparison of 19 patients treated with AAIF and 9 patients using ORIF showed no difference in duration of the operation, postoperative flexion, or clinical results. Faster, easier postoperative rehabilitation was noted in the group managed arthroscopically. In addition, anatomic reduction was achieved in 85% of patients treated arthroscopically versus only 55% treated by open surgery.

Conclusion

Tibial plateau fractures are complex injuries which require anatomical reduction of bony injuries along with

management of associated ligamentous and meniscus injuries.

The outcome depends not only on personality of initial injury but on management of fracture and associated injuries also.

ORIF is the gold standard for treatment of tibial plateau fractures, but arthroscopic assisted fixation provides a viable alternative for Schatzker type I- IV tibial plateau fractures, as evidenced in this study.

The advantages of arthroscopic assisted fixation include direct visualization of articular surface for perfectness of reduction and single stage management of associated ligamentous and meniscal injuries. Arthroscopy allows for visualisation of accurate fracture reduction while obviating the need for extensive operative exposure. In some regards, arthroscopy narrows the gap between the extremes of open versus nonoperative management. The arthroscopy allows for evacuation of hemarthrosis and any fracture debris. Arthroscopy may offer the advantages of more rapid recovery, reduced pain, early full range-of-motion, improved fracture healing, and more complete and functional recovery.

In our study the objective and functional scores showed excellent results for 75% and 83% respectively. In our study 41 % patients had intra-articular pathology,

	Cassard ⁸ 1999	Gill ¹⁰ 2001	Rossi ²⁵ 2008	Siegler ²⁶ 2009	t.Cristea ²⁷ 2010	Our study 2013
No of patients	26	25	57	28	262	24
Number of follow-up patients	26	25	46	21	184	23
Average age	42.3	45.2	48	43	51	35
Average follow-up (months)	32.7	24	60	59.5	60	06 months
Associated lesions (%)	-	-	39	32.1	63	41
Meniscal lesions (%)	30.8	36	28	7.1	53	33 Lateral 25 Medial 08
ACL Lesions (%)	11.5	32	11	3.6	10	8
Postoperative complications (%)	7.7	0	3.5	0	4.9	8
AKS average	94.1	-	93.2	85.2	93	-
AS functional average	94.7	-	94.8	91	95	94.5
Clinical Rasmussen average	-	27.5	28.2	25.5	-	-
Malalignment (%)	10.5	-	8.7	32.1	4.9	8

amongst which meniscal injuries were managed in same sitting.

Apart from usual complication of such fracture management, the compartment syndrome due to extravasation of arthroscopy fluid is most dreaded and serious complication which should be looked for after arthroscopic assisted fixation. emergent fasciotomy should be offered in cases of compartment syndrome.

Having enumerated all the advantages of arthroscopic assisted fixation, it requires a steep learning curve with infrastructure and facilities of arthroscopy. It requires the excellence in both arthroscopy and traumatology on surgeons' part to achieve better result with this minimally invasive technique.

In present surgical scenario of minimally invasive or key-hole surgeries with cutting edge technology, it is prudent to use the arthroscopy in fracture fixation to meet the expectations of patients and science. In the near future we should see more and more use of arthroscopy in various intrarticular fractures.

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Severe Jaundice in a patient with Acute Hepatitis - E Infection with Coexistent Glucose-6-Phosphate Dehydrogenase Deficiency

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ABSTRACT

Hepatitis E is prevalent in most developing countries, and common in any country with a hot climate. Hepatitis E virus is one of the common causes of acute viral hepatitis in India but usually manifests as a mild self-limiting illness. Viral hepatitis in the presence of glucose-6-phosphate dehydrogenase (G6PD) deficiency may be associated with complications such as severe anemia, hemolysis, renal failure, hepatic encephalopathy and even death. The incidence of G6PD deficiency in the general population of northern India is reported to be between 2.2% and 14%. The coexistence of viral hepatitis and G6PD deficiency has been reported to be associated with severe jaundice and other complications [1,2]. Hepatitis E infection with G6PD deficiency has been associated with more severe illness in only one previous report [3]. We report an additional case.

Key words:

Glucose-6-phosphate dehydrogenase, Hemolysis, Hepatitis E

Introduction

Hepatitis E is prevalent in most developing countries, and common in any country with a hot climate. Hepatitis E virus is one of the common causes of acute viral hepatitis in India but usually manifests as a mild self-limiting illness. Viral hepatitis in the presence of glucose-6-phosphate dehydrogenase (G6PD) deficiency may be associated with complications such as severe anemia, hemolysis, renal failure, hepatic encephalopathy and even death. The incidence of G6PD deficiency in the general population of northern India is reported to be between 2.2% and 14%. The coexistence of viral hepatitis and G6PD deficiency has been reported to be associated with severe jaundice and other complications [1,2]. Hepatitis E infection with G6PD deficiency has been associated with more severe illness in only one previous report [3]. We report an additional case.

Case Report

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18 years old male patient presented with mild to moderate fever without chills, upper abdominal pain, fatigue and loss of appetite for 4 days. He had noticed a yellow discoloration of the eyes and dark colored urine for one day. There was no history of alcohol consumption or previous history of liver disease. On the 5th day, he developed vomiting. For which he got admitted to the hospital. On examination, he was icteric. Abdominal examination revealed a soft, tender hepatomegaly, palpable 2 cms below the costal margin. There was no splenomegaly or lymphadenopathy and no other significant physical findings.

Next day of admission, he developed fever with chills and back pain along with vomiting. He became more deeply icteric within 24 hours. His Hemoglobin which was 13.3 dropped to 8.6 within 12 hours. Bilirubin risen to 14.4 to 41.5 over 12 hours and within 24 hours risen to 49.5 Leucocytes count risen to 12,790 to 22,740 over 12 hours and 46,460 within 48 hours. There was no evidence of hepatic encephalopathy. Since there was acute drop in hemoglobin, patient was having severe weakness and breathless, one packed cell transfusion was given.

Laboratory investigations revealed Prothrombin time was 14 s (control: 12 s) INR 1.16. Immunoglobulin IgM anti-hepatitis A virus, hepatitis B surface antigen, IgM anti-hepatitis B core and anti-hepatitis C virus were negative, while IgM anti-hepatitis E virus (HEV) was positive. A diagnosis of HEV hepatitis was made.

His laboratory investigations are as follow:

Date	14/7 (morn)	14/7 (evening)	15/7	18/7	19/7	22/7	25/7	27/7
Hb	13.3	8.6	8.3	7.1	8.0	8.9	9.8	10.2
WBC	12790	22740	43380	46460	10930	7090	7380	6700
T.BIL	14.4	41.5	49.5	46	45	15.5	14.0	13.4
D.BIL	6.9	26.5	28.5	27.5	25	7.5	8.0	6.8
SGOT	2570	7030	8030	1140	1909	570	159	129
SGPT	4420	6700	7109	5407	2740	233	335	240

The peripheral blood smear showed polychromasia, anisopoikilocytosis, Microcytosis, plenty of Burr Cells, Few Bite cells and few spherocytes and reticulocytosis (reticulocyte count was 12%). Urine was positive for hemoglobinuria. Osmotic fragility test – hemolysis starts at 0.6% and completed at 0.3% of NS (reported as normal). LDH 1385 (N 225- 450 IU/L)

Estimation of G6PD level: More than 1 hours (N: 30- 60 minutes)

Coomb's Test: Direct as well as Indirect — Negative

The patient was managed conservatively, including avoiding all hepatotoxic, nephrotoxic and oxidant drugs, and maintaining an adequate urine output. Patient improved over period of three weeks.

Discussion

Hepatitis E virus (HEV) is an icosahedral, nonenveloped single stranded RNA virus that is approximately 27 to 34 nm in diameter (4). It has been classified as the single member of the genus hepevirus in the family Hepeviridae.

Mild hemolysis associated with decreased red blood cell survival may be commonly seen with viral hepatitis, but is seldom of clinical significance (5). However, when viral hepatitis occurs in G6PD-deficient patients, hemolysis may be severe (6).

The patient described in this case had severe intravascular hemolysis as evidenced by a rapid fall in hemoglobin, sudden rise in bilirubin, and hemoglobinuria. Profound hemolysis in G6PD-deficient individuals is usually

precipitated by exposure to selected drugs. However, as in this case, viral hepatitis may precipitate massive hemolysis even without the intake of such drugs. The mechanism of hemolysis is thought to occur through decreased levels of reduced glutathione in red blood cells (7). Reduced glutathione levels could result from the accumulation of oxidants due to hepatic dysfunction and lead to increased hemolysis in the presence of G6PD deficiency. Despite the high levels of bilirubin in these patients, the prognosis is mainly related to the severity of hepatic injury (8). Acute renal insufficiency, though uncommon in uncomplicated acute viral hepatitis, can occur as a fatal complication of severe intravascular hemolysis in these patients (6). Excess hematin and bilirubin may result in the obstruction of renal tubules, leading to acute renal insufficiency with increased morbidity. Renal failure may be non-oliguric, therefore, kidney function should be assessed by regularly monitoring blood chemistry, and urinary sodium and osmolarity. Measures to prevent renal failure include maintaining good hydration and adequate urine output, and avoiding nephrotoxic drugs(9).

HEV infection is transmitted through the feco-oral route but, unlike other enteric agents, does not generally spread from infected persons to their close contacts (10).

In patients with acute viral hepatitis and unexplained anemia with very high serum bilirubin levels, intravascular hemolysis should be considered and investigated.

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Large and Rare Adrenal Oncocytoma

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ABSTRACT

Adrenal oncocytoma is a very rare tumor, with just about a hundred reported in literature. The fact that they are mostly non-functioning and benign leads them to attain a large size or they are discovered incidentally on imaging. We describe a case of a non-functioning giant adrenal oncocytoma in a 30-year-old lady that was managed successfully. This is probably the largest case of adrenal oncocytoma reported till now.

Key words:

Oncocytoma, non-functioning, gross examination

Introduction

Adrenal oncocytoma is a very rare differential of adrenal incidentaloma with other possibilities being cortical adenoma, pheochromocytoma, adrenocortical carcinoma, granulomas, adrenal cysts, myelolipoma, ganglioneuroma and metastatic [1,2]. Oncocytoma or the neoplasms composed entirely of oncocytes are well described in the kidney, thyroid, salivary glands and other sites like the pituitary, parathyroid, lacrimal gland, respiratory tract and choroid plexus. Oncocytic adrenal tumors have only a few reported cases [3].

Case Report

A 30-year-old lady presented to the out patient department with complaints of post prandial heaviness for the last 6 months. She had no co-morbidities and her personal, family and past history was unremarkable. Her general physical examination was normal and abdominal examination revealed an 8 x 8 cm smooth mass with ill-defined borders in the right hypochondrium. A computed tomography scan showed a large 20 x 15 x 7 cm well demarcated enhancing mass between the right kidney and liver with multiple internal septae and few hypodense areas suggestive of a right adrenal mass.

Her baseline tests including haemogram, renal function, and liver function were normal. Her urinary

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metanephrines, 24 hour urinary VMA, and serum cortisol were all within normal limits. The patient underwent an open right adrenalectomy, which was uneventful.

On gross examination, the specimen weighed 3200 grams, and measured 23.5 x 17.5 x 10 cm. Microscopically the tumor was encapsulated and composed of oncocytic cells arranged in sheets, nests, trabeculae and pseudoalveolar pattern. The cells were polygonal with round to oval nuclei and abundant eosinophilic granular cytoplasm. The central area of the tumour showed necrosis and there was no capsular and vascular invasion. The periphery showed a normal adrenal gland. The tumour cells were negative for Vimentin, S-100 and Chromogranin A. The postoperative period of the patient was uneventful.



Figure 1 : Cut section of the oncocytoma showing central necrosis

Discussion

Oncocytic neoplasms arising in the adrenal glands are extremely rare, and are usually discovered to be nonfunctional and mostly benign tumors. However,

recent data indicates that about 20% of adrenal oncocytoma's demonstrate some elements of malignancy and about 10-20% of them appear to be functioning secreting cortisol and adrenal androgens. Most of the tumors have arisen in the age group of 27 to 72 years with a greater majority present in females. The size varies between 3 to 17 cm in all reported cases, making this the largest reported case in literature [4,5].

On gross examination they are a large, rounded, encapsulated a well-circumscribed mass. They are usually brown or mahogany on cut section and may display areas of necrosis and hemorrhage. The microscopic appearance includes cells arranged in solid, trabecular, tubular or papillary patterns and the cells are highly eosinophilic and granular. On electron microscope observation, the cells contain abundant mitochondria. The immunophenotypic profile is difficult to evaluate, as it was not studied in more than half of the cases. They are generally negative for S-100 and Chromogranin and immunoreactivity being variable for vimentin [4,6].

The CT and MRI findings are non-specific and there is no characteristic imaging of adrenal oncocytoma, however it is possible to differentiate the lesion from adrenal adenoma based on fat concentration.

The approach to an adrenal mass depends on its size and function. The surgical management has traditionally been open surgical approach, however these days a laparoscopic approach carries less morbidity, quicker recovery and a shorter hospital stay. The contraindications include very large size, vascular invasion, diffusion to surrounding structures and presence of lymphadenopathy [6].

Conclusion

Adrenal oncocytoma is one of the histological subtypes of incidentally detected adrenal masses. It is usually a large, benign, nonfunctional adrenal tumor and it usually presents as an incidental, large adrenal mass. The CT and MRI findings cannot be used to differentiate benign and malignant oncocytic neoplasms and only microscopic criteria are able to identify precise histology characterization and clinical behavior, so adrenalectomy is the mainstay of therapy and laparoscopy is now the most popular approach.

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A Rare Case of Chronic Myeloid Leukemia with Massive Splenomegaly with Splenic Infarct

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ABSTRACT

Chronic myeloid leukemia (CML) is a common clinical entity associated with splenomegaly but CML with massive splenomegaly with splenic infarct is a very rare presentation. Splenic infarct occurs as a result arterial and venous compromise. CML is a common hematological malignancy with a wide variety of presentation with classical long term history. The median age at diagnosis of CML is 65 years. We present here a case report of 45 year old male having CML with massive splenomegaly with splenic infarcts for which splenectomy was done .

Key words:

Chronic myeloid leukemia, splenomegaly, splenic infarct, splenectomy

Introduction

Chronic myeloid leukemia (CML) is a common hematological malignancy with a wide variety of presentation. Massive splenomegaly is not uncommon but massive splenomegaly with splenic infarct is very rare presentation of CML. Splenic infarction is itself a rare clinical entity which has gained increasing clinical attention in the recent years [1]. Massive splenomegaly is usually defined as a spleen extending well into the left lower quadrant or pelvis or which has crossed the midline of the abdomen. Massive spleens weigh at least 500 to 1000 gram. The most common disorders associated with splenomegaly were hematologic, infectious, hepatic, congestive, and inflammatory [2, 3]. The median age at diagnosis of CML is 65 years.

CML can be divided into 3 phases: chronic, accelerated, or blast phase. Massive splenomegaly with splenic infarction can be caused by hematological disorders like sickle cell anaemia, polycythemia, myelofibrosis, leukemia or embolic disorders due to atrial fibrillation, infective endocarditis, prosthetic mitral valve, paradoxical emboli from right heart, HIV associated mycobacterial infection, chronic infections and some connective tissue disorders. Splenic infarction occurs either due to a sudden occlusion

of splenic blood supply due to big embolism or due to gradual infarction of splenic parenchyma by hematological cells [4]. Ultrasonography is the initial investigation modality but contrast enhanced computerized tomography (CECT) is the investigation of choice [5].

Case Report

A 45 yrs old gentleman a known case of diabetes mellitus from 10 years on OHA and Hypertensive, was diagnosed to have CML in December 2012 in view of echymotic patches over whole body. His total leukocyte count was more than three lakh with platelet count more than 21 lakhs at the time of diagnosis. He was started cytoreduction with tab hydroxurea and Imatinib. He showed good response to continuous therapy but in month of July 2015 he developed pain in left hypochondrium and was evaluated to have hepatosplenomegaly with leukocytosis (34,000). Gradually his TLC increased more than one lakh and started having difficulty in walking and backache. MRI LS spine done showed heterogeneously enhanced mass lesion at L3 level causing severe canal stenosis with thecal sac indentation. His BCR-ABL ASSAY: 46.65% and bone marrow biopsy showed myeloid hyperplasia with M:E ratio 10:1. In view of his worsening symptom Imatinib resistant mutation analysis was done, which was positive. He received palliative EBRT to L5-S3 region and was started on Nilotinib. He developed Anemia with Pancytopenia and Nilotinib was stopped for the same. He again started on Dastinib in December 2015. His symptoms gradually worsen with diffuse abdominal pain, decrease appetite, difficulty in walking and orthopnea. On examination, massive hepatosplenomegaly

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with liver span of 23 cm and splenic span of 25 cm found. Blood parameter showed anemia with pancytopenia. Opensplenectomy was performed to relieve the symptoms and for good outcome Figure 1. Intraoperative multiple splenic infarcts were seen which was confirmed on histopathologic examination Figure 2.



Figure 1: Intra-operative view of splenomegaly



Figure 2 : Showing enlarged spleen with splenic infarcts.

Discussion

The most common disorders associated with splenomegaly are hematologic, infectious, hepatic, congestive, and inflammatory [2,3]. The hallmark of CML is the presence of a balanced translocation between the long arms of chromosomes 9 and 22 (also known as the Philadelphia chromosome) [6]. The disease is diagnosed during the chronic phase in over 80% of cases [7].

CML can be divided into 3 phases: chronic, accelerated, or blast phase, patients may be asymptomatic at the time of diagnosis [7]. When present, symptoms may include fatigue, night sweats, weight loss, left upper quadrant abdominal discomfort, early satiety, or anorexia.

Leukocytosis is a common feature of the disease with a white blood cell (WBC) count as high as $1000 \times 10^9/L$. Tyrosine kinase inhibitors with activity against the BCR-ABL fusion product, such as imatinib, dasatinib, or nilotinib, are the treatment of choice for CML presenting in the chronic phase [6]. Splenic infarct is rare in CML. Splenic infarction occurs as a result of arterial and venous compromise. It is a rare clinical entity. The clinical

presentation of infarction have wide variation. They may be discovered incidentally on radiological, intra-operative or post mortem studies or can even present as haemorrhagic shock as a result of subcapsular haematoma [8]. Splenic infarct can be diagnosed by radiological imaging tests like diagnostic USG imaging, CECT, gadolinium enhanced MRI, splenic scintigraphy using radio labeled colloids.

Complications of splenic infarction include abscess formation, haemorrhage, pancreatic fistula, gastric fistula, splenic fistula, splenic rupture and pseudocyst formation. Medical management of splenic infarction is only symptomatic treatment for pain relief by using NSAIDs or narcotic analgesics. Splenectomy is reserved for cases of splenic infarct with massive splenomegaly in order to improve the outcome and to relieve the symptoms.

Conclusion

CML have a wide variation in its presentation. Splenomegaly is not uncommon but massive splenomegaly with splenic infarct is very rare. Therefore it is important to evaluate all symptoms and parameters so that the underlying major diseases will not be missed.

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Brainstem Encephalitis

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ABSTRACT Brainstem Encephalitis is a rare disease and though it may follow various infections, *inflammatory and non infectious conditions* account for most cases.

Key words: Brainstem Encephalitis

Introduction

Acute demyelinating encephalomyelitis is an immune-mediated disorder localized to the central nervous system which can be localized (clinically isolated syndrome or CIS) or disseminated (acute disseminated encephalomyelitis, or ADEM). When localized to the brainstem, it is known as Bickerstaff's Brainstem Encephalitis (BBE). It is characterized by the acute onset of signs of brainstem involvement, days to weeks after a variety of viral and bacterial infections or vaccinations.

Incidence

The incidence of BBE is higher in Japan compared to the Western nations. The precise incidence and prevalence of BBE in the United States and other Western nations is currently unknown, which can be attributed to the rarity of the disease and confusion and overlap with other Anti-Gq1b antibody syndromes. A recent nationwide survey of patients in Japan with BBE estimated that the annual incidence of BBE is approximately 0.078 per 100,000 individuals. Prevalence is unknown.

Etiology

The exact etiology remains unknown though it is postulated that it has an autoimmune origin. It often follows a preceding illness and its association with herpes simplex, enteroviruses, cytomegalovirus, mycoplasma pneumoniae, listeriosis and C. jejuni has been reported. The Anti-Gq1b antibody may have a role in the pathophysiology of BBE and MFS (Miller Fisher Syndrome); however the absolute underlying

pathogenesis has yet to be discovered. Anti-Gq1b antibody is the one that interacts with the peripheral nerve ganglioside, Gq1b (predominantly expressed at neuromuscular junctions, sensory nerves, and proximal segments of cranial nerves like oculomotor, trochlear and abducens nerves 3, 4, 6 respectively). Binding at these sites by the Anti-Gq1b antibody can be a potential explanation for the presenting symptoms and signs such as ophthalmoplegia and ptosis.

We here report a case of BBE in a girl, who presented with multiple cranial nerve palsies, severe ataxia and incoordination.

Case Report

13 year old girl admitted to Max Superspeciality Hospital with complaints of headache off and on since 1 month (right hemi headache) worsened since 4 days; vomiting since 3 days, 5-6 times/day, non bilious non projectile; altered sensorium; diplopia; slurred speech since 2-3 days; paraesthesias all over the body, right > left since 2 days. The child was not able to walk since 1 day. No history of contact with Koch's; or trauma; or drug intake.

Examination revealed a well built child with normal higher functions; GCS of 11/15 (E3V3M5); dysarthria; diplopia on leftward gaze with left eye ptosis (cranial nerves 3, 4, 6); left 7th nerve palsy; rest cranial nerves normal (good cough and gag reflex). Motor examination revealed normal tone; power > 3/5; DTRs not elicitable; plantars down going. No signs of meningeal irritation were present. She was grossly ataxic; with grossly impaired coordination; pupils were normal size and reaction, no papilloedema. Systemic examination was normal. Her initial blood investigations showed a normal sepsis profile and normal renal functions. MRI brain with contrast was done which revealed T2/FLAIR hyperintensity involving right middle cerebellar peduncle and the adjacent pons and right cerebellar hemisphere.

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No post contrast enhancement is seen in the region of altered signal intensity; probability of focal demyelination/infective process. Lumbar puncture was done which was suggestive of viral meningoencephalitis (TLC = 85 cells; N05L95; sugar = 59/81 mg%; protein = 39mg%; CSF for bacterial meningitis negative; viral studies for enteroviruses and DNA viruses were negative). Nerve conduction velocities in all 4 limbs were done which was normal ruling out GBS. She was started on IV fluids/Inj. Ceftriaxone/Inj. Acyclovir/Inj. Ampicillin/Azithromycin/Gabapentin (for paraesthesias). She showed a gradual stabilization over the next 48 hours. A repeat MRI brain was done after 72 hours which showed a mild increase in the hyperintensity in the previously involved areas though clinically she showed improvement. Repeat CSF study was done on day 5 of admission which was the same as previous one (TLC = 85 cells, N10L90, sugar 60/108 mg%, protein = 44 mg %). Her speech became much clearer; right cranial nerve 6 paresis persisting and all other improved. The coordination also was better but still ataxic. Though no investigation on CSF was positive her medication was continued in view of clinical improvement to the combination. She was investigated for autoimmune diseases but was normal. Her serology for herpes simplex, mycoplasma pneumonia, and antiganglioside antibodies specifically GQ1b was negative. Antibodies to immune markers NMDA receptors and VGKC were negative. CSF for oligoclonal bands was negative. She was given IV acyclovir/ Ampicillin/Ceftriaxone for 7 days and discharged.

Discussion

Brainstem encephalitis is a medical condition that causes swelling and inflammation of the brainstem. BBE is a type of Clinically Isolated Syndrome (CIS) in the consensus definitions proposed by the International Pediatric Multiple Sclerosis Study Group. It is the only CIS that can be associated with encephalopathy.

Bickerstaff's brainstem encephalitis (BBE) is a rare post-infectious neurological disease characterized by the association of external ophthalmoplegia, ataxia, lower limb areflexia, extensor plantar response and disturbance of consciousness (drowsiness, stupor or coma).

Symptomatology of brainstem encephalitis includes visual disturbances, vertigo, and an overall lack of coordination of which ataxia and vertigo are often the first noticeable symptoms. Patients usually present with onset of diplopia and gait disturbance following upper respiratory or gastrointestinal infections. The external ophthalmoplegia is progressive (within 4 weeks of onset) and relatively symmetrical. Flaccid symmetrical

quadriparesis may also be observed in over 50% of the patients, along with deep or superficial sensory impairment, facial weakness, bulbar palsy, internal ophthalmoplegia, oscillopsia (bouncing vision), blepharoptosis and nystagmus. In the acute phase of disease, BBE may be so severe that there is complete ophthalmoplegia, facial diplegia and full paralysis of arms and legs, resembling 'brain-death'. Respiratory problems may develop and can become severe enough that the patient might require ventilatory support. Nausea, vomiting, and persistent headaches may occur due to the abnormal vision. The lesions are located mainly in the brainstem; hence the presentation is related to brainstem dysfunctions.

Diagnosis is based on the clinical findings, patient history, cerebrospinal fluid (CSF) analysis (revealing raised protein levels), detection of anti-GQ1b IgG antibodies (not present in all patients), MRI studies (revealing high-intensity abnormalities in the posterior fossa, white matter or thalami) and neurophysiological examinations (electroencephalogram and electromyography indicative of central nervous system and predominantly axonal involvement).

Treatment

Effective management and treatment of BBE and other variants of Anti-Gq1b syndrome requires prompt recognition and diagnosis. While the majority of patients with BBE and MFS achieve nearly complete recovery, a number of reported cases of recurrence exist. *Surprisingly, despite alarming presenting symptoms, spontaneous recovery is frequently seen both BBE and MFS.* The majority of documented cases of BBE have shown patients to regain baseline functional status within 6 months of diagnosis. Because of frequent spontaneous recovery and rare occurrence, today there is a lack of generalized consensus on the role of specific treatments for BBE. A recent Cochrane review was not able to give any specific recommendations for the treatment of both BBE and MFS because of an apparent lack of randomized trials evaluating treatment in such clinical settings. A few case reports have shown **plasmapheresis** may hasten recovery in patients presenting with high serum titres of Anti-Gq1b antibody and with severe complications such as coma. There has been one reported case of a plasmapheresis and immunoglobulin therapy resistant BBE with eventual resolution with **Rituximab** treatment. Rituximab may have a role in the treatment of BBE based on the effect of anti-CD20 on mice models in countering the immune and complement mediated attack on the pre-synaptic terminal by the Anti-Gq1b antibody. The potential role of plasmapheresis and Rituximab in the treatment of BBE highlights that Anti-Gq1b antibodies

Comparison of Bickerstaff Brainstem Encephalitis; Miller Fisher Syndrome and Guillain Barre Syndrome

	BBE	MFS	GBS
Epidemiology	South east Asia	South east Asia	Worldwide
Diagnostic Criteria	Symmetrical ophthalmoplegia	Symmetrical ophthalmoplegia	Areflexia
	Ataxia	Ataxia	Acute ascending symmetrical limb motor weakness
	Altered sensorium	Areflexia	Dysautonomia
Lab Findings	Anti Gq1b Antibody	Anti Gq1b Antibody	Anti Gq1b Antibody*
	CSF pleocytosis	CSF pleocytosis	CSF pleocytosis
	CSF Albumino-cytological dissociation	CSF Albumino-cytological dissociation	CSF Albumino-cytological dissociation
MRI Findings	High T2 signal with little if any enhancement in brainstem and basal ganglia	Typically MRI Brain findings are unremarkable given the peripheral predilection.	Anterior nerve root thickening and enhancement surrounding the medullary cone extending along the cauda equina

* Can be seen in GBS, however less common than in BBE and MFS

may be a player in the pathogenesis of BBE. Whether BBE is a distinct disease entity or a variant of FS and GBS is still to be clarified. Many authors even consider FS and BBE as the same disease. BBE has a monophasic remitting course, with 2/3rd of patients becoming symptom-free at six months and around **16% patients requiring ventilation**. Deaths have been reported in BBE. Thus *patients with BBE need careful observation and in cases that worsen or have respiratory involvement, immunomodulatory therapy to prevent residual deficit and death needs to be started.* **There are no randomized controlled trials of immunomodulatory therapy in FS or related disorders (BBE) on which to base practice.** Until recently, corticosteroids were the only widely used treatment for acute demyelinating encephalitis. However, there is disagreement regarding their efficacy and there have not been any prospective studies or treatment guidelines. Several reports suggested that *plasmapheresis and IVIG have a beneficial effect on patients with BBE.* Moreover, combined therapy of IVIG and high-dose methylprednisolone should be more efficacious therapy. No advantage of any particular treatment can be inferred from the data available. Controlled clinical trials are needed to test this proposal. Management is based on immunotherapy with intravenous immunoglobulin

(IVIG) or plasma exchange.

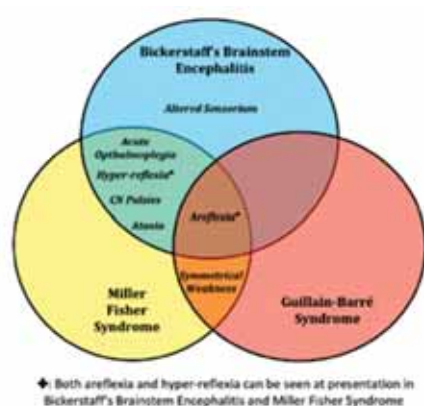
The CSF screen for infections was negative in our patient and markers for vasculitis were normal. The presence of extensor plantar response inspite of areflexia indicated that weakness is caused by brainstem lesion rather than polyradiculoneuropathy. NCV studies were normal though it may suggest peripheral motor axonal degeneration overlapping axonal variant of GBS.

Thus, **the presence of cranial neuropathy (diplopia, dysarthria), disturbed state of consciousness, ataxia, areflexia and dramatic MRI signal abnormalities in brainstem indicated a diagnosis of BBE.** Our patient was managed conservatively and observed over one week. Patient improved partially with dysarthria subsided and ataxia improved; however diplopia and mild facial paresis persisted. Repeat MRI and CSF studies after 7 days showed no progression and patient is under close follow up.

CONCLUSION

Bickerstaff's Brainstem Encephalitis is a rare neurological condition that many general physicians and even neurologists will not encounter during their lifetime. This interesting condition has characteristic signs and

symptoms including altered sensorium that should be kept in mind in the setting of new onset ataxia. Anti Gq1b antibody seropositivity has also suggested that BBE is most likely part of a spectrum of diseases under the umbrella known as Anti-Gq1b antibody syndrome. The similarities between BBE, MFS, and GBS may often cause confusion in reaching a diagnosis especially in the setting of clinical overlap. More epidemiological studies of the incidence and prevalence of BBE should be performed in Western nations. There is also a need for randomized controlled trials to evaluate if certain therapeutic agents or procedures may hasten recovery for patients with BBE.



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“Spectrum of Sleep Disturbances in Parkinson’s Disease”

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ABSTRACT Sleep disturbances are one of the most common non motor symptoms in patients with Parkinson’s disease. It affects the quality of life of PD patients adversely. Sleep disorders in PD are diverse and have complex pathophysiology, however it can be diagnosed clinically by most of the neurologist if diligently asked for. Treatment of the sleep disturbances in PD may result in improved motor functions and better quality of life. Here we review the various sleep disorders, its pathophysiology and treatment.

Keywords: Parkinson’s disease, Sleep disorders, Excessive daytime sleepiness, Quality of life

Introduction

Parkinson’s disease (PD) is a chronic, progressive, disabling neurodegenerative disorder with a prevalence of approximately 360 per 100,000 and an incidence of 18 per 100,000 per year. As the average age of the population increases, the prevalence of PD can be expected to rise. The motor symptoms in PD result from degeneration of dopaminergic neurons in substantia nigra. The pathological process in PD is even more extensive to involve serotonergic, noradrenergic and cholinergic systems responsible for development of nonmotor symptoms (NMSs) of PD. NMSs such as depression, anxiety, psychosis, sleep disturbances, cognitive impairment and autonomic dysfunctions are commonly seen in PD and they adversely affect the quality of life. One large series has reported that almost every patient with PD exhibit at least one nonmotor symptom.¹ Sleep disorders are among the most common non-motor symptoms in Parkinson’s disease. The overall prevalence of sleep disturbances in PD varies from 68%-98%.² Sleep disturbances in PD is multifactorial and have negative impact on quality of life (QoL) of PD patients. Clinicians and PD patients may not recognize the sleep disturbances and its relation with PD so the prevalence

may be underestimated. However it can be diagnosed clinically with the help of questionnaires and very occasionally polysomnography is required. Treatment options are diverse and depend upon the diagnosis of sleep abnormality which is present in patient.

Parkinson’s Disease and sleep disturbances:

The whole gamut of sleep disorders may occur in PD. Sleep in PD patients may be disturbed because of the neuronal degeneration of sleep regulating areas in brain, motor (difficulty in turning inside bed) and non-motor symptoms such as autonomic dysfunction, hallucinations, depression and due to effects of drugs used for treatment of PD. The various sleep abnormalities in PD are as below:

1. *Insomnia*

Nocturnal symptoms with sleep disturbances are well known in PD.^{3,4} Frequent night-time awakenings and sleep dis-ruptions are the most common sleep problems in PD. In addition, periodic leg movements in sleep, fragmentary nocturnal myoclonus, sleep apnea, REM sleep behavioral disorders, and parasomnias may all disrupt sleep in PD. Reversal of sleep rhythm with sun downing also is common in PD. Upto 80% of patients with PD complain of sleep fragmentation, insomnia, and nocturia. A nationwide German study has recently included 1449 PD patients and reported sleep disturbances in 49% of patients.⁵ Another study (PRIAMO study) done on 1072 PD patients showed frequent occurrence of nonmotorsymptoms fatigue being commonest (58%) followed by insomnia, (37%), urgency and nocturia (35%), RBD (30%), EDS (21%), and restless legs syndrome (RLS) (15%).¹

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Insomnia can be due to problem with onset or maintenance of sleep. Impaired sleep maintenance is one of the major factor for poor nighttime sleep quality in PD. Sleep benefit has been observed in patients with PD even prior to start of treatment denoting the importance of maintenance of sleep quality in PD. A good nighttime sleep reduces the severity of daytime parkinsonism, and many patients describe better mobility the morning after a restful night. Polysomnographic studies have demonstrated increased sleep latency and frequent awakenings, with as much as 30-40% of the night spent awake in patients with PD. Drug induced hallucinations also disrupt night time sleep in patients with PD. Apart from dopaminergic drugs, depression, anxiety, and autonomic dysfunction also contribute to poor sleep quality in PD. On the other hand insufficient dopaminergic treatment causing nocturnal akinesia, nocturia, muscle cramps, and various other nonmotor symptoms may cause sleep disruption. There are various scales to measure the subjective sleep quality and they are validated for use in PD.

2. Excessive Daytime Somnolence

Prevalence of Excessive daytime sleepiness (EDS) varies in different studies from 15.5 to 74% of PD patients.⁶ EDS is considered to be a risk factor for development of PD and it may be present even at premotor stage of disease. The prevalence of EDS increases as the disease progresses and its prevalence varies according to the assessment method. Excessive daytime somnolence may occur due to fragmentation of nighttime sleep and sleep apnoea but it is thought to be integral part of disease itself. PD patients with EDS do not correlate with variables such as disease severity, total sleep time or sleep stage percentages, but rather found to be related to primary impairments of waking arousal and REM-sleep expression. In PD, sleepiness is associated with cognitive decline and hallucinations suggesting EDS to be a poor prognostic marker. Although infrequent but sudden, unforeseen sleep is also reported in PD known as sleep attacks. Significant number of PD patients may be unaware of their sleepiness despite being involved in accidents. Dopaminergic drugs used to treat PD also contribute to excessive daytime sleepiness. Dopamine agonists have been found to be associated with EDS but total dose of dopamine is reported to be the main predictor of EDS. A multicenter study reported correlation between ESS and levodopa equivalent dose irrespective of drug class selected.⁷ EDS can be assessed subjectively by using the Epworth sleepiness scale (ESS)⁸ and is recommended by the Movement Disorders Society, for the evaluation of EDS in patients with PD. ESS >10 is indicative of pathological sleepiness. In a recent review on polysomnographic studies in PD

has shown lack of objective sleepiness by multiple sleep latency test despite significant subjective sleepiness in patients.⁹

3. Circadian Rhythm Sleep Disorders

Circadian rhythm disturbances are also present in PD mainly imparted by the degenerative process. There may be alteration in levels of hormones especially melatonin, changes in heart rate, blood pressure due to the process of neurodegeneration in hypothalamus, sleep regulating centers in brain stem, autonomic dysfunction and levodopa therapy. PD patients with depression have been found to exhibit circadian rhythm abnormalities more compared to patients without depression. Suprachiasmatic nucleus is intact in PD but presence of circadian rhythm abnormalities indicates involvement of hypothalamus and brainstem.

4. Rapid Eye Movement Sleep Behaviour Disorder

Rapid eye movement sleep behaviour disorder (RBD) is present in 25-50% of patients with PD. RBD is characterized by a loss of muscle atonia during REM sleep that results in dream-enacting behaviour, and sometimes even leads to injury to the individual or bed partner. It is more common in males and its association with neurodegenerative diseases especially synucleinopathies has been found. Locus coeruleus, pedunculopontine nucleus and cholinergic nuclei are pathologically involved in RBD. Early manifestations of PD preceding even the onset of typical motor symptoms, impaired visual and olfactory discrimination have also been observed in idiopathic RBD patients. In the survey of their PD patients, Scaglione and colleagues (2005) found that only 33% had RBD. Of these, PD preceded RBD in 73%, an average of 8 years before onset of RBD. However not all patients with RBD develop PD but early detection of RBD can be used as a preclinical marker and a basis for intervention. PD patients with RBD are non tremor type, have more tendency to fall and less responsive to dopaminergic medications. American Academy of Sleep Medicine (AASM) 2001, proposed criteria for RBD includes intermittent loss of REM sleep electromyographic atonia and by the appearance of motor activity associated with dream enacting behaviour. The motor activity involves kicking, punching severe enough to hurt the bed partner along with emotional outbursts.

The gold standard for diagnosis remains a polysomnography with audiovisual recording. Video-PSG is more specific which also shows increased muscle activity during REM sleep.

5. Restless Legs Syndrome (RLS)

RLS is an urge to move the legs due to uncomfortable sensation in legs during period of inactivity or rest. RLS is commonly observed along with motor symptoms, and sleep disturbances of PD. In 80% of RLS patients have periodic limb movement during sleep. Response to dopaminergic therapy indicates similar pathophysiology of PD and RLS. The pathophysiology of RLS is not known but a central dopaminergic dysfunction has been proposed based on responsiveness to dopaminergic therapy and decreased dopamine D2 receptor binding observed in the RLS patients by SPECT.

Diagnostic criteria for RLS

Required criteria

- Uncomfortable and unpleasant sensations in the extremities (prickling, stinging, itching, 'like crawling ants,' sometimes described as pain), with an urge to move
- The sensations begin or worsen during inactivity
- The sensations and/or urge to move are partially or totally relieved by movement
- The sensations and/or urge to move display a circadian pattern: worse in the evening or night compared than the early morning; or only occurring in the evening or night

Supportive features of RLS

- Positive family history
- Clear beneficial response to dopaminergics
- Presence of periodic limb movements during sleep

6. Periodic Limb Movement Disorder

Periodic limb movement disorder often co-occurs with RLS but it is a distinct disorder. PLM is more common in PD compared to control and it is thought to be a factor causing nocturnal sleep disturbances and excessive daytime sleepiness.

7. Sleep Apnoea Syndrome (SAS)

Studies have reported 20-60% incidence of SAS in PD, attributing it to nocturnal akinesia or hypokinesia of respiratory and pharyngeal muscles. However, polysomnographic studies did not find difference in apnoea-hypopnea index (AHI) in patients with PD and control. The relation between PD and SAS is still not clear.

Pathophysiology:

Sleep dysfunction in PD is multifactorial, primarily due to neurodegenerative process impairing the

thalamocortical arousal and affecting sleep regulating centres in the brainstem. Other factors responsible are nocturnal hypo or akinesia, nocturia, bizarre dreams, comorbid anxiety and depression and side effects of the pharmacological treatment. Finally, there can be overlapping with independent age-related sleep disturbances.

It has been hypothesized that mesocorticolimbic dopaminergic system modulates sleep and wake cycle.¹⁰ Impairment in this pathway leads to fragmentation of night sleep and excessive daytime somnolence. Dopamine maintains sleep homeostasis and its role is independent from nigrostriatal dopaminergic system. It has been postulated that degeneration of mesocorticolimbic dopaminergic system is imparting sleep disturbances in PD. Apart from mesocorticolimbic system, degeneration of serotonergic, cholinergic, and noradrenergic system also play an important role in sleep disruption in PD. These neurons form a flip-flop switch model of sleep-wake regulation. This model was proposed by Saper, which includes an ascending arousal system regulated by noradrenergic locus coeruleus, glutaminergic parabrachial nucleus, serotonergic dorsal raphe, dopaminergic ventral periaqueductal gray matter and histaminergic tuberomammillary nucleus and a sleep inducing system controlled by ventrolateral preoptic nucleus. The axons of ascending arousal system run through hypothalamus where they contact lateral hypothalamic orexin neurons and maintain thalamocortical arousal. During wake state arousal system inhibits preoptic sleep promoting cell groups. There is a complex relationship between hypothalamic hypocretin system and dopaminergic system in basal ganglia and it functions as an external regulator of the flip-flop switch promoting wakefulness. PD patients have fewer dopaminergic neurons in cell groups of arousal system than controls.¹¹ Other foci of arousal such as noradrenergic and cholinergic nuclei are also depleted in PD. It has been hypothesized that dopaminergic dysfunction and neuronal degeneration can destabilize this switch and its regulators, promoting rapid transitions to sleep intruding on wakefulness and also disrupting nocturnal sleep. Hallucinations also have a disruptive effect on sleep, as they show a clear nocturnal preponderance and mostly visual.

However, sleep disturbances themselves, in particular, REM sleep abnormalities, represent the strongest predictor for the occurrence of hallucinations. Interestingly, hallucinations in PD may reflect intrusions of dream content into wakefulness, possibly due to degeneration of brainstem areas specifically involved in REM sleep regulation. This hypothesis is supported by increased nighttime RBD and abnormal REM sleep during

daytime napping in PD patients with hallucinations. EDS is very common symptom in PD. Gjerstad et al, & Boddy et al, 2007 proposed that excessive daytime sleepiness (EDS) is frequent in Parkinson's disease and its presence has been associated with longer disease duration and dementia.^{12, 13} EDS is currently part of the proposed criteria for Parkinson's disease related dementia as a supporting feature. EDS is due to multilayered causes primarily due to degeneration of sleep regulating centres, fatigue caused by the motor disability, night time awakenings due to akinesia, nocturia, hallucinations and RBD, dopaminergic treatment especially dopamine agonists. The presence of daytime rapid eye movement (REM) sleep intrusions associated with visual hallucinations and sleep onset REM periods in the multiple sleep latency test (MSLT) in some PD as seen in narcolepsy suggests that both disorders may share similar pathology. Like in narcolepsy, role of orexin/hypocretin system has also been postulated in development of EDS in PD and it has been hypothesized that degeneration of hypocretin neurons in hypothalamus is causing excessive daytime somnolence in patients with PD. However, cerebrospinal fluid (CSF) levels of hypocretin-1, which are typically low in narcolepsy have been normal in various studies in Parkinson's disease.¹⁴ In addition to hypocretin system, noradrenergic, cholinergic, and serotonergic system in pedunclopontine, raphe nucleus and locus coeruleus respectively causes disruption of sleep structure and EDS. Other comorbid conditions such as depression and anxiety also have negative impact on sleep quality. In polysomnography recordings destructuring of sleep has been observed in PD mainly the duration of NREM 2, 3

and REM.

Dopaminergic therapy and sleep

Chronic medication with levodopa may be one of the important factor resulting in poor night time sleep quality and excessive daytime somnolence. Levodopa induces sleep at lower doses but disruption of sleep and wakefulness at higher dose. However, because of alleviating effect of levodopa on motor symptoms may lead to improvement in sleep. Therefore the relationship between dopaminergic therapy and sleep is more complex. Studies have reported that dopaminergic drugs result in better sleep architecture due to improvement in nocturnal akinesia, nocturia and some of the dopa responsive nonmotor symptoms. However, worsening of nighttime sleep quality and daytime somnolence has also been reported in patients taking dopaminergic therapy. Some of the drugs such as ropinirole has beneficial effect on sleep quality while amantadine, selegiline may result into sleep disruption. One recent study has reported subjective sleep impairment in patients taking high doses of levodopa but there was no correlation with objective polysomnographic findings. Excessive daytime sleepiness rather improves after dopaminergic therapy due to improvement in motor symptoms.¹⁵

Management of sleep problems

Sleep disturbances in PD are multifactorial. Treatment requires detection of responsible factors and their management. There are various drugs also which help in improving sleep quality in PD patients. (table no. 1)

Table 1: Etiology and management of sleep disturbances in PD

SLEEP DISORDER	ETIOLOGY	MANAGEMENT
Nocturnal sleep disturbances	<ol style="list-style-type: none"> 1. Difficulty in turning inside bed 2. 'Off' dystonia 3. Dyskinesias 4. Nocturia 5. Restless leg syndrome 6. Drug effects: hallucinations 7. Rapid eye movement disorder of sleep 8. Sleep disordered breathing 9. Depression, anxiety 	<p>Titration of dopaminergic therapy Slow release levodopa preparation, Doudopa, apomorphine Management of autonomic symptoms Dopaminergic therapy for RLS and PLMS</p> <p>Modification of drug therapy</p> <p>Clonazepam</p> <p>Continuous positive airway pressure Antidepressants and anxiolytics</p>
Excessive daytime somnolence	<ol style="list-style-type: none"> 1. Disturbed nocturnal sleep 2. Dopamine agonists(Sleep attacks) 3. Degeneration of primary brain regions regulating sleep 	<p>Improvement of nocturnal sleep Removal of medication if possible Stimulants: modafinil, methylphenidate</p>

Conclusion

Sleep disorders are commonly found in patients with PD. It is often ignored due to prominent motor symptoms in PD. However diagnosis and treatment of sleep disturbances may lead to improvement in both motor symptoms and quality of life of PD patients.

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Parkinson's Rehabilitation: Achieving the Goal of Independence in Daily Activities.

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ABSTRACT

There is a growing need to address rehabilitation issues to supplement medical therapy in Parkinson's Disease. The aim of this prospective follow-up study was to assess the impact of education about daily activities on the functional status in patients with Parkinson's disease. A total of 76 cases with Parkinson's Disease on a stable pharmacological regimen and moderately disabled participated in this study. All patients were educated about daily activities to practice at home. All the cases were evaluated with UPDRS II & VI (Activities of Daily Living) at baseline and at 3 months. The study demonstrated improvement in mean score of UPDRS II & VI, but it was statistically significant only in UPDRS VI. The activities of daily living like dressing, turning in bed and walking respond favourably to reduce the impact of disability. So, in conclusion systematic program of daily activity schedule is beneficial in moderately disabled Parkinson's disease.

Keywords: Activities of Daily Living, Parkinson Disease, Rehabilitation.

Introduction

People with Parkinson's disease need to exercise to prevent the negative effects of inactivity. Being active is one of the most important things you can do to maintain your physical and mental well-being. Exercise will not alter the progression of Parkinson's but it is essential for maintaining your quality of life. The goal of rehabilitation is aimed at effecting a change in the occupational performance, performance possibilities or performance competencies of the patient or caregiver. These changes can be at the level of the person himself, the activity or the social and physical environment. The strategies and interventions that are selected depend on the preference of the persons with Parkinson's or caregiver as well as the potential for changing aspects of the person, the activity and the environment. A combination of interventions usually applies. At level of the person, changing occupational performance can involve interventions directed at: i) improving and maintaining

skills during the performance of activities. ii) applying compensatory skills or strategies during the performance of activities. iii) increasing insight and knowledge in order to adequately deal with current and future limitations in daily activities (self-management).

Material and Methods

Patients of any age and either gender diagnosed with PD by the UKPD Society Brain Bank Criteria, on a stable pharmacological regimen attending Movement Disorder clinic, PGIMER & Dr Ram Manohar Lohia Hospital New Delhi, during period of study and for six months before entry into study and able to ambulate and function independently were included in the study. Patients having Hoehn and Yahr¹ 4/5 PD, dyskinesia, Parkinsonism variants, neurosurgical intervention, cognitive deterioration, psychiatric disturbances, head trauma, other neurological diseases and medical contraindications to exercise were excluded from the study. The procedures followed were in accordance with the ethical standards of the institutional committee. Patient demographics, including race, education, and employment status, were recorded. The Unified Parkinson Disease Rating Scale II, Schwab and England Activities of Daily Living Scale VI² were applied at baseline and at 3 months.

The program was of a 4-month period. All patients were

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taught the same set of daily activities for one month and were asked to continue the same exercises at home. During follow-up, the patients were evaluated at about the same time of the day and about the same duration after drug intake to decrease clinical fluctuations that may hamper evaluation.

Schwab and England Activities of Daily Living Scale VI rates independency from 0% = Vegetative functions such as swallowing, bladder and bowel functions are not functioning. Bedridden to 100% = completely independent. able to do all chores without slowness, difficulty or impairment. essentially normal. unaware of any difficulty.

Unified Parkinson Disease Rating Scale II. Activities of Daily Living (for both "on" and "off") rates speech, salivation, swallowing, handwriting, cutting food and handling utensils, dressing, hygiene, turning in bed and adjusting bed clothes, falling, freezing when walking, tremor and sensory complaints on 0-4 likert scale.

Prescription of Daily Activities

Rest and Sleeping: Keep the bedroom floor clear of things that could cause tripping and falling. For example, don't leave shoes, books or papers on the floor.

To get into bed: Approach the bed as you would a chair; feel the mattress behind both legs. Slowly lower yourself to a seated position on the bed, using your arms to control your descent. Lean on your forearm while you allow your

trunk to lean down to the side. As your trunk goes down, the legs will want to go up, like a see-saw.

To get out of bed: Bend knees up, feet flat on the bed. Roll onto your side toward the edge of the bed by letting the knees fall to that side. Reaching across with the top arm. Turn your head and look in the direction you are rolling. Lower feet from the bed as you push with your arms into a sitting position. A straight back chair anchored at the side of the bed or a bed rail can help you roll more easily.

To roll or turn over in bed: Bend your knees up with feet flat. Allow knees to fall to one side as you begin to roll. Turn your head in the direction you are rolling and reach top arm across the body.

To scooting over in bed: Bend your knees up with feet flat. Push into the bed with feet and hands to lift your hips up off the bed. Then shift hips in the desired direction. Finish by repositioning feet in the direction your hips moved.

Helping handle/bed rail provides assistance with rolling and support for pushing yourself to an upright position. It attaches between the mattress and box spring. An inexpensive alternative to a bed rail is a straight-back chair laced to the bedframe.

Getting around: If balance or strength is affecting your ability to walk, a mobility aid such as a cane, walker or wheelchair can help you keep moving. A straight cane with a rubber tip is better. Handgrips should be

Table 1: Demographic characteristics of study participants

n = 76	Percentage Mean (SD)	Range
Sex	68 M / 8 F	
Age (yr)	65.6(+/- 11.4)	41-86
Duration of PD (yr)	8.3(+/-7.2)	0-27
Highest level of education		
College or higher	51.6	
High school or technical school	32.8	
Employment status		
Unemployed, disabled, or retired	67.7	
Employed	32.3	
Living situation		
Alone	11.3	
With spouse or family	88.7	
Total UPDRS	53.4(+/- 22.9)	25-121
Hoehn and Yahr stage	2.7(+/-1)	1 to 5

comfortable, and the height of the cane should be adjusted for the best support.

Choosing a Chair: Chairs should have a stable base and average height chair with firm, smooth cushions and sturdy armrests.

Changing positions: Stiffness, rigidity and slowed movement can make it difficult to do activities such as getting in and out of a chair. Here are some helpful tips to make these activities easier. When possible, it is best to work with a physical or occupational therapist to learn the best techniques.

Sitting to standing: Scoot hips forward near the edge of the chair. Feet should be shoulder width apart, and flat on the floor. Position feet behind bent knees. Lean forward until head is positioned “nose over knees”. Push forward and up from armrests using both hands. Keep head down (looking at floor) initially when rising. Rocking back and forth can provide the extra “momentum” needed to stand up.

Standing to sitting: Take large steps as you approach the chair. Avoid short, shuffling steps. Make a wide turn and position yourself so chair is centered directly behind you. You should feel the chair against the back of both of your legs before sitting. Reach back for the armrests as you lean forward from the waist. Use arm rests to slowly lower body into the chair, this helps avoid “crash” landings.

To getting in and out of a car: First make sure the car is parked far enough away from the curb so that you can step onto the level ground before you go into, or get out of, the car. Have the seat far enough back so that you have enough room for your legs.

To get into a car: Turn and back in toward the seat so that your buttocks are leading the way. Reach back for

the seat or dashboard and slowly lower yourself to sit. Never hold on to the moving door. Reach over to the inside edge of the seat and begin lifting one leg in at a time. Use a pillow to make low seats higher

To get out of a car: Reach inner arm for the dashboard and begin moving one leg at a time out of the car. Your body should be in the car and your legs should be out on the ground. Scoot forward to the edge and lean forward while pushing up from the seat or dashboard. Never pull up on the car door.

Recreation and Exercise Moving, stretching and exercising as much as you can will also help prevent secondary effects that may develop such as: poor posture and balance, decreasing range of movement (losing flexibility), decreased strength particularly in the muscles that hold you upright, resulting in a tendency to stoop forward, decreasing endurance (being out of breath or fatigued). Plan your physical activities and exercise during “on” times when your medication is working well. Exercise groups are a good option for some people. Leisure activities such as gardening, playing with children or pets, painting, or walking offer enjoyable options for movement and exercise.

Bathing: Shower/commode chairs allow you to sit in the shower while you bathe. Dry off using several small towels rather than one large towel.

Bathroom safety instruments: handrails; shower curtains to make transferring easier and to prevent slipping by keeping your floor dryer; non-skid rubber bath mat. All bath rugs should have a rubber backing; nightlight. A 3-in-one bedside commode converts to a toilet frame, raised toilet seat, or shower-chair. This particular commode style is usually covered by insurance if you have a prescription from your physician.



Eating: Schedule meals during “on” times, or when medication is working best. Cut food into small-bite size pieces so that it is easier to chew and swallow. If you have swallowing problems, don't drink thin liquids or use a straw. Sit up as straight as possible when eating, and stay upright for at least 30 minutes after each meal. Helpful eating aids: Insulated dish keeps food at the right temperature for slow eaters. Hi-lo scoop plate keeps food from sliding off. Rocker knife cuts meat and other foods with a simple rocking motion. A pizza cutter also works well. Easy-to-hold angled utensils make mealtime easier.

Handwriting: Think “big strokes” when writing. Use lined paper. Vary the size, shape, and weight of your pen. Change pens when your hand tires. Change your grip on the pen. Place the pen between your index and middle finger and wrap your thumb around the bottom of the pen for better stabilization and support. Try a roller point, ball point, or felt tip. Decide which one works best.

Results

Seventy six patients (68 males and 8 females) with mean (\pm SD) age of 65.6 (\pm 11.47) years satisfying the inclusion criteria participated in the study. Four patients dropped out of the study because of not able to follow-up due to other health reasons. Descriptive statistics including mean and standard deviation distribution were found out for each quantitative variable. To compare scores related to UPDRS section II & VI (activities of daily living) between baseline and 3 months, paired t-test was used. There was improvement in mean score of both UPDRS II & VI (activities of daily living) at 3rd month, but improvement in UPDRS II score was statistically non-significant while UPDRS VI mean score showed statistical significant improvement.

Discussion

The present study showed regular rehabilitation exercise program helped to improve ADL in patients with

moderate Parkinson's disease. The programs must be designed and tested to determine how best to reinforce self-management gains and sustain improvement of patients and family caregivers as the disease progresses. People with Parkinson's disease responded to a six-week program of self-management rehabilitation with health related quality of life benefits beyond best medical therapy³. According to Borrione P et al tailored physical activity is a valid tool to be included in the therapeutic program of PD patients, considering that this approach may ameliorate the symptoms as well as the overall physical incapacity, reduce the risk of falls and injuries, and ultimately improve quality of life⁴. The Environmental Skill-Building Program reduces burden and enhances caregiver well-being in select domains and has added benefit for women and spouses⁵.

A limitation of this study is that neurobehavioral symptoms including mood, anxiety, and optimism were not assessed, and cognitive function was only assessed with the MMSE. The presence of extrapyramidal symptoms including bradykinesia, rigidity, and dyskinesia may pose unique challenges to patient insight regarding motor performance. Another potential source of discrepancy is that performance in the home environment may be different than the office setting. The capacity for compensatory strategies in the home may not be reflected in performance-based tasks in the office. The use of assistive devices, familiar routine, assistance from family, and variable lighting conditions may raise or lower the level of function in one place compared to another. Because clinical assessment of daily function and disability are integral to clinical decision-making and clinical trials, the accuracy of current methods of functional assessment are an important area for further study.

Suggestions for special caregiving concerns: Gradually add minutes of activity to your program. Minutes count and your goal is to build up your activity level to a total of

Table 2 : UPDRS Unified Parkinson Disease Rating Scale (n-number of cases, SD-Standard deviation, \$ Non significant between baseline and 3 month.)

		Baseline	3 month
UPDRS Section II ADL	Mean (n=76)	9.86	8.79
	SD	2.99	2.67
	p value		0.022 ^{\$}
UPDRS Section VI (Schwab & England ADL)	Mean (n=76)	78.81	80.02
	SD	2.67	2.99
	p value		0.009

30 to 60 minutes a day. It is never too late to become active. Here are some specific ideas you can try to increase your activity level: Take a walk 20 steps in your normal way, then take 20 long steps, then 20 normal steps, then swing your arms for 20 steps. Repeat for the duration of your walk. Get off the bus one stop early or park the car one block away. Use the stairs instead of the elevator. Lift cans of soup, or any small weight, to exercise your arms (see strengthening exercises section). Do leg exercises while watching television. Join an exercise class. One of the best ways to stay motivated is to exercise with others. Play your favourite music and dance or move to the beat.

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