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Dehradun, Uttrakhand

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President
International Medical
Sciences Academy
New Delhi, India

Prof. Hem Chandra
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JIMSA
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Association News

Appeal For IMSA Building Fund

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, Uttrakhand, 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.

Thanking you in anticipation and warm regards,

Yours Sincerely,

Dr. K.Jagadeesan, President, IMSA,WHQ

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09.07.17

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13.08.17

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Dehradun, Uttrakhand, India

April 12 & 13, 2019
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Editorial

Increasing burden of diabetes mellitus in India and the challenges ahead

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India is witnessing a transition in its disease profile. While the Indian health infrastructure grapples with the burden of infectious diseases, there has been a rapid rise in the non-communicable diseases in the last 25 years. This has been attributed to a faulty lifestyle such as improper dietary habit, reduced physical activity and increased stress levels. For this reason, non-communicable diseases are referred to as the 'life style disorders'.

The latest update on the IDF website from the South-East Asian Region states that 1 in 3 individuals above the age of 30 years is suffering from one or more lifestyle diseases, be it diabetes, high cholesterol, blood pressure, thyroid or cancer.[1] Diabetes mellitus is one of the most feared of these lifestyle disorders, primarily because of the microvascular and macrovascular complications associated with it. Implications of diabetes extend beyond the individual, since the socio-economic burden has to be shouldered by the family and therefore a significant burden is transmitted to the society and the nation.

Magnitude of Type-2 diabetes

According to IDF data among the 415 million diabetics in the world 78 million are in the South-east Asia region which is anticipated to rise to 140 million by 2040. IDF figures indicate that in 2015 there were 69.1 million diabetics in India when its population was 798.988 million (prevalence rate of 8.7%). [1] King et al [2] have mentioned that 19.4 million Indians were living with diabetes in 1995 (prevalence rate of 3.8%) and they predicted a steep rise of 195% over the next three decades. Thus, an estimated 57.2 million Indians are expected to suffer diabetes in 2025. However, in the last two decades (1995-2015), prevalence of diabetes has risen by 256% to 69.1 million. This rise is certainly alarming. In addition to this, another 36.5 million Indians are reported to have pre-diabetes. [3]

The National Urban Diabetes Survey (NUDS), studying 11,216 subjects using the stratified random sampling method, reported age-standardized prevalence of diabetes and IGT as 12.1% and 14.0% respectively, with no gender difference. [4] Subjects under 40 years of age had higher prevalence of IGT than diabetes (12.8% vs. 4.6%). A positive and independent association was observed between diabetes and co-variables such as age, body mass index, family-history of diabetes, waist-to-hip ratio, monthly income and sedentary life style. The first three variables also had an association with IGT. [4]

The Prevention of Diabetes in India Study (PODIS), which used multi-step sampling method, reported a prevalence of 4.3% for diabetes (urban 5.6%; rural 2.7%) and 5.2% for IGT (urban 6.3%; rural 3.7%). [5] Although the prevalence reported by PODIS was only 0.5% higher than the 3.8% reported by King et al, PODIS confirms that the prevalence of IGT is greater than that of overt diabetes. This indicates that if proper preventive measures (especially lifestyle modification) are not implemented at the population level, people with IGT will eventually develop diabetes, thereby contributing to the rising prevalence of diabetes. Also, it was remarkable to note that this 'at-risk' population was distributed not only in urban but also in rural areas.

The prevalence of diabetes as reported in the first-phase of Indian Council of Medical Research Indian Diabetes study (ICMR INDIAB study) was 10.4% in Tamil Nadu, 13.6% in Chandigarh, 5.3% in Jharkhand and 8.4% in Maharashtra. [6]. Rural-urban figures for the four centers are tabulated in Table 1. Diabetes prevalence varies from 10.9 to 14.2% in the urban areas and 3.0 - 8.3% in the rural areas. One-in-nine to one-in-seven urban individuals above the age of 20 years was found to suffer from diabetes. A greater problem is the high prevalence of IGT (prediabetes) which varied from 9.8 to 15.2% in urban areas and 7.1 to 14.7% in rural areas. The cumulative burden of diabetes and prediabetes taken

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Table 1

Urban-rural prevalence of diabetes and prediabetes: Results of first phase of ICMR - INDIAB study

Area	Prevalence of diabetes *			Prevalence of Prediabetes	
	Self reported	Newly diagnosed	Overall	Prediabetes alone	Prediabetes + diabetics
Tamilnadu					
Urban	8.5	5.2	13.7	9.8	23.5
Rural	4.1	3.8	7.8	7.0	15
Jharkhand					
Urban	8.4	5.1	13.5	10.7	24.2
Rural	0.7	2.3	3.0	7.4	10.4
Chandigarh					
Urban	6.6	7.6	14.2	14.5	28.7
Rural	3.1	5.2	8.3	14.7	23
Maharashtra					
Urban	3.7	7.2	10.9	15.2	26.1
Rural	1.7	4.9	6.5	11.1	17.7

* All values expressed as percentage of total population of the given area

together varied from 10.4 - 28.7%. These data indicate that a sizable population of India, both rural and urban, is at risk.

A survey by National Nutrition Monitoring Bureau found the prevalence of diabetes among men and women of rural India were 8.2% and 6.8% respectively. [3] High prevalence rates were observed in Kerala, Tamil Nadu and Gujarat (8.2 - 16.4%). Diabetes prevalence was reported to be increasing in the rural population at a rate of 2.02 per 1000 population per year. These data suggest that the large population of India, which is at the risk of or is suffering from diabetes, pose a formidable challenge to the nation. For every known case of type-2 diabetes mellitus, there is another person with undiagnosed disease.

The numbers game for type-1 Diabetes

As per the IDF, type-1 diabetes is also increasing worldwide at a rate of 3% every year, particularly in young children. [1] Over half a million children in the world were living with diabetes in 2015 when India had 70,200 children with type-1 diabetes. Thus India ranks second only to USA which has 84,100 diabetic children. The IDF has kept the goal that no child should die of diabetes. However, it is a fact that only 50% of the middle-income countries have full government supply of insulin essential to treat type-1 diabetes. India needs to tackle the numbers game of type-1 diabetes as well. Needs of the type-1 diabetes are different from that of type-2 diabetes and a multidisciplinary team is essential for proper management.

Peculiar conundrums of diabetes

It is a well-known fact that type-2 diabetes occurs at least 10-15 years earlier in Indians vis-a-vis Western populations. With better medical facilities and advancements, the average life span in India has also gradually increased over the years to 66.46 years in 2013 (68.25 years for women and 64.75 years for men). Consequently, the geriatric population of India is rapidly increasing from 8% in 2010 and it is expected to cross 20% by 2050. Prevalence of diabetes increases with age, and an ageing India therefore is bound to have a higher proportion of diabetics. Elderly people also suffer from several other associated degenerative disorders and compromised organ system functions. Thus diabetes and diabetes-related complications really lead to poor quality of life in the elderly people.

Due to unique phenotype of the Indian population, one third of type-2 diabetic patients can have normal body mass index. This indicates that insulin resistance is present even at lower body mass indices. Yajnik and Misra have observed that Indians, despite having low BMI, have greater visceral fat thereby contributing to insulin resistance and a greater prevalence of metabolic syndrome. South Asians, including Indians, are considered to be genetically predisposed to insulin resistance and development of diabetes. [7-10]

Childhood obesity and type-2 diabetes

Traditionally diabetes occurring in childhood and adolescence is thought to be of 'lean type' presenting with osmotic symptoms. It is also considered to be more prone for ketosis and requires insulin for prevention of ketoacidosis. Although maturity-onset diabetes in the young can occur over 30 years of age, young-onset diabetes has been virtually synonymous with type-1 diabetes. However, increasing urbanization, reduced physical activity (due to lack of outdoor playing spaces and lack of security in towns), easy availability of alternative modes of entertainment (eg. television, computers and video games) and faulty dietary habits (eg. increased consumption of sugary beverages, fast food items) have increased the incidence of childhood obesity in India. [11,12] Obesity is a phenotypic marker of type-2 diabetes in children and adolescents. There are two important corollaries to this pattern- (i) type-2 diabetes is now affecting all age groups leaving out none; (ii) such young population developing diabetes indicates that these children will have to live with the disease for the next 50-odd years of their life. Thus the burden of diabetes extends to enjoyable youth, productive middle age and the fading years of senior citizens.

Socio-economic impact of diabetes

Even well controlled diabetes is known to cause psychological stress of harboring a disease. It affects not only the individual, but also his/her family members. An otherwise good glycemic control may be complicated by episodes of hypoglycemia and associated weight gain (commoner with the earlier agents or with insulin). Obesity-related co-morbidities add up to diabetic problems. Cardiovascular and renal complications can occur despite adequate control of blood sugar. All these add-up to the burden and hamper the quality of life. Needless to say, if blood sugar levels are uncontrolled, hyperglycemia related complications and the acute diabetes complications in addition to the typical microvascular and macrovascular complications will occur.

Cost of monitoring blood sugar level, periodic investigations, medications and screening or preventive treatment of co-morbidities and complications significantly add up to the economic liabilities of the individual and the family. Apart from the monetary burden, individual suffering and loss of productivity (of either the patient or the care-taker) adversely affect the nation. The sheer numbers of diabetics in India demand a large chunk of the resources. Unlike the Western countries, India lacks an effective system of health insurance. About a decade back, diabetics could not be even insured; but now some insurance companies have started insuring diabetics as well, though selectively, at the cost of higher premium.

Studies outlining true prevalence of diabetes in India as well as its socio-economic impact are lacking. In India, 85-95% of all the cost of diabetic care is borne by individual patients. According to National Health Portal of the Indian Government [3] direct expenses of diabetic care consume 27-34% of incomes of rural and urban poor people, while the middle-to-high income groups in rural and urban areas consume 5-12.6% and 4.8-16.9% of income, respectively. The cost of diabetic care has been increasing over the years and the factors responsible for this include cost of hospitalisations, increasing distance from patients' dwelling to the health care setting, increasing frequency of health care visits and higher incidence of complications. In an article, published in this issue Fathima et al [13], observed that the annual median total cost per patient in rural area is Rs. 6228, 77% of which was contributed by the direct medical costs (Rs. 4800). The authors also reported that 8.54% of the family income was spent on diabetes care.

Impact of mortality

Even if the mammoth number of diabetics is not a burden, definitely mortality is! The Southeast Asia region, with its 1.2 million diabetic deaths (14.2% of all adult deaths) in 2013, ranks second among the seven IDF global regions. About 55% of these deaths occurred in age group < 60 years and 27% in < 50 years age group. High mortality of diabetes is related to poor overall health care in India, and 59.11% die within a week of hospitalization. Infections and chronic renal failure are the major causes of diabetic death in India as compared to cardiovascular and cerebrovascular diseases in the West. [14]

The way forward

Despite the mammoth burden of diabetes and prediabetes, it is imperative to look at future with optimism. Solution to this grim situation lies within it. Knowing very well that prediabetes and obesity can progress to diabetes, we must target this group. Early diagnosis and proper treatment of this group is essential for preventing diabetes-related complications. Early detection by community screening, although appears to increase the number of diabetics, effectively reduces the burden by avoiding complication. The Indian Diabetes Risk score [15-16] is a handy tool, which can be employed for screening.

The ICMR INDIAB study emphasized the importance of

focussing on low awareness and poor glycemic control. Also, the presence of other metabolic abnormalities needs to be diagnosed and adequately controlled to prevent the vascular complications associated with diabetes. To tackle the bull by its horns, we need to instill lifestyle modifications and inculcate healthy lifestyle. Simple lifestyle changes that can make a lot of difference in diabetic care includes shifting the television out of the bed room, avoiding snacking during television watching, avoiding sleeping soon after dining, having light meals at night, incorporating an exercise schedule (even within buildings if the outdoor space is lacking), light strolling after dinner, getting down a stop earlier while commuting to office and taking a flight of stairs instead of using the elevator.

Government can also play its part by levying hefty taxation on sugary beverages, sweets, confectionaries and fast foods. Healthy foods can be promoted by reducing taxations on fruits and vegetables. There is also a need to spread awareness about the disease, its diagnosis and complications. Ill-effects of obesity, smoking, tobacco and alcohol consumption should be highlighted by public propaganda. Together we can treat (secondary prevention), beat (primary prevention) and defeat diabetes (primordial prevention).

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ORIGINAL ARTICLE

Economic Evaluation of Care in Diabetes Patients – A Community Based Study in Rural Areas of Bangalore District

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ABSTRACT

Background: Diabetes mellitus is a chronic disease which lead to great economic burden on the individual, the family and the community. Studies of low-income Indian family estimate that 25% of family income is required for the care of one adult member with diabetes. This study was carried out to estimate the prevalence of type 2 diabetes mellitus in persons aged more than 30 years in the villages of Bangalore district. We also assessed the direct, indirect and intangible costs involved in the care of a person with diabetes mellitus in a rural setting of India.

Methods: Partial economic evaluation from a patient perspective was done in four villages under the Hesaraghatta Primary Health Centre, Bangalore Urban District.

Results: The prevalence of reported diabetes among individuals aged 30 years and above is 8.2%. Mean duration of diabetes mellitus was 6.7± 5.0 years. The annual median total cost per patient was Indian Rupees (INR) 6228 out of which the annual median direct cost was INR. 4800 (77%). Cost of medicines constituted a large proportion of 59% of the direct costs, followed by laboratory tests (19.4%), consultation charges (12.5%), hospitalization (7.7%), transport (2.9%) and food (1.9%). The indirect costs consisted of the mean amount spent while caring for a person with diabetes was INR. 54.2 and the mean cost for the patient losing his income as a result of hospitalization was INR. 814.

Conclusions: Care of diabetes imposes considerable economical burden on the low and middle income families in India. Cost of medicines is responsible for the major share of this expenditure. Price control of diabetic drugs will benefit patients.

Key words: Diabetes mellitus, rural area, cost of health care, health economics, community settings

Abbreviations: **HCE** - health care expenditure; **INR** - Indian rupees

Introduction

Diabetes Mellitus has been recognized by both the United Nations and the World Health Organization as an ever-increasing public health problem. [1] Globally, diabetes prevalence is increasing and is responsible for 5% of all deaths annually (World Health Organisation 2011). Prevalence of diabetes according to 2010 statistics was 285 million people and it is expected to increase to 438 million by 2030 (Diabetes Help 2010). Given the current projections, mortality due to diabetes is expected to increase by 50% in the next 10 years. [2]

Because of its chronic nature, the severity of its complications and the means required to control them, diabetes is a costly disease, not only for the affected individuals and their families, but also for the health authorities. [2] Unlike in the West, where mostly the older population is affected, the burden of diabetes in Asian countries is disproportionately high in young to middle-aged adults. [3] This could have long-lasting adverse effects on a nation's health and economy, especially for developing countries.

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In 2010, HCE on diabetes was 11.6% of the total HCE in the world. Global HCE of treating or preventing diabetes and its complications is estimated in 2010 to be at least 376 billion US. dollars. By 2030, this cost is projected to exceed 490 billion US. dollars.[4] The financial burden due to diabetes stems not only from the medicine cost, but also from charges due to consultation, investigation, hospital stay and surgery done for the complications of diabetes. Apart from this, indirect cost which arises from the time lost and loss of productivity also accounts significantly.[3] The cost of treating diabetes is enormous, as most of the patients with diabetes not only require anti-diabetic medications, but also need anti-hypertensives, aspirin, statins and other medications related to its complications. This becomes more expensive when the cost of insulin therapy is added that also requires rigorous monitoring.[4]

In recent times, changing economic trends have meant that evaluation of treatment costs is no longer a subject of mere academic interest, even in the developing world.[6] Community based studies in Rural India to assess costs of care in diabetes is lacking. Therefore this study was conducted to assess the prevalence and costs of care in type-2 diabetes mellitus in terms of direct, indirect and intangible costs from a patient perspective.

Methods

A cross sectional study was done in 4 villages falling under the administrative jurisdiction of Hesaraghatta Primary Health Centre located in Bangalore Urban District. A house-to-house survey was conducted to list all the permanent residents aged 30 years and above. From this, all individuals diagnosed with type-2 diabetes mellitus were identified. All households that were locked despite two visits by the study team were excluded from the study. A partial economic evaluation was done from a patient perspective to assess the different types of costs in diabetic care. A pretested semi-structured interview schedule was administered to the identified individuals suffering from diabetes for at least 1 year prior to the study. The interview schedule included questions related to socio demographic profile, care of diabetes control, healthcare expenditure for diabetes, diabetes care and household economy. The following costs were included in the study: (1). Direct medical costs which included money spent on consultation fees, laboratory investigations, hospitalization, drugs and insulin; (2) Direct non medical costs which included food and transport of patient and caregiver; (3) Indirect costs which included the income loss of patients because of hospitalization, income loss of caregivers while accompanying the patient for check-up or hospitalization or while caring for the patient; (4) Intangible costs which included reduced productivity, premature retirement.

The direct medical costs were estimated by documenting the expenditure on medicines, laboratory investigations and hospitalization. If the patient was taking the same medicines for the past one year average drug costs were estimated by calculating the drug costs including insulin cost, cost of syringes etc per month multiplied by 12 to get the annual cost. If patients were prescribed with any new medicines in the preceding 1 year or if any medicines were stopped, suitable modifications were made in the calculation. Information was collected on consultation fee per visit separately to private and public health centres and it was multiplied by the stated frequency of consultations in the past one year. Similarly expenses on investigations were multiplied by the frequency of tests to get an estimate of average annual costs for sugar level monitoring and laboratory investigations. The estimated cost of hospitalizations was based on the average hospital charge per event multiplied by number of hospitalisations per year.

Non-medical direct costs were difficult to estimate compared to medical costs as it was not possible to check for the validity of information given by the participants. However patients were asked regarding the average travel costs per visit in the recent past and it was multiplied by the number of total visits in the year. Similarly the cost of food taken outside during the visit for diabetic care in the recent past visit was multiplied by the number of visits. Patient was specifically asked whether food was taken outside for all the visits and accordingly the cost was estimated. Details of special food items prepared only for the diabetic patient at home was obtained and the cost of preparing them was calculated per month; later it was multiplied by 12 to get the average annual indirect cost of special food. All these estimated costs were added up to get the average annual direct non-medical costs incurred by a diabetic patient.

Data on indirect costs covered in this study include man-days lost due to diabetes and the loss of personal as well as family income. We have also calculated the number of days lost for the caregiver while accompanying the patient for diabetes care. Number of man-days lost has been estimated for workers only. Monetary value of man-days lost has been calculated by multiplying number of man-days lost with reported personal daily income (monthly income divided by 30). Loss in personal and family income is calculated by reported percentage loss in income with the monthly income multiplied by 12. An attempt was made to estimate the intangible costs by asking the diabetic patients if their diabetic status resulted in reduced productivity at work expressed in

terms of percentage and the number of years of premature retirement due to their diabetic status.

The sources of information were mainly the patient, family members or caregiver of the patient. The given information was crosschecked by inspecting the medicine and their price list. To check the validity of the costs reported, patients were asked to produce the bills for the investigations, consultations and hospital admissions.

The data collected were entered in Microsoft Excel and analyzed using standard statistical package (SPSS). Total expenditure was computed by adding the individual components of the direct, indirect and intangible costs. Costs were measured as continuous variables and checked for normality using Kolmogrof Smirnov test. All costs were described using percentages, mean and standard deviation, median and inter quartile range. Mann Whitney U test was used to compare the median between 2 groups. Spearman correlation coefficient was used to find out the correlation between total costs and other selected variables. Linear regression model analysis was done to find out association between total costs as dependent variable and the different variables influencing cost as the independent variables. P value < 0.05 was considered statically significant.

Results

Out of 571 households in the 4 villages, the study team was able to contact only 541 households. These 541 households contained 1065 individuals above the age of 30 years out of which 87 were diagnosed with diabetics. Thus the prevalence of diabetes in the 4 villages among individuals aged 30 years and above is 8.2%. Mean duration after diagnosis of diabetes mellitus was 6.7 ± 5 years. In 4 of the 87 diabetics, the duration of illness was less than one year and hence they were excluded from the study. The interview schedule was administered to the remaining 83 individuals.

The mean age of the study population was 57.1 ± 12.0 years and males constituted 61.4% of the study population. [Table 1] A total of 46 individuals (56.6%) were financially independent where as 32 individuals (38.6%) were completely dependent on their family members for their financial needs, the rest (4.8%) being partially dependent.

Around two thirds (67.5%) of the study population had their blood sugars under control (last blood sugar reading d" 200mg/dl) and 30.1% of the diabetic individuals had an associated co-morbid condition (the most common being hypertension). A large majority (83%) of the study population had a health check-up at least once in three months and about 60% of them accessed health care from private sector. Majority (98.8%) of the diabetics reported consuming medications regularly and they procured medicine mostly from private pharmacy (56.1%). About 83.1% of the diabetics had regular laboratory tests done and most of them availed the services of private laboratory (42.2%). Among the study cohort, 71 individuals (85.5%) had at least one complication of diabetes and the common complications encountered were neuropathy, peripheral vascular disease, eye complications and cardiovascular diseases.

The median total annual cost of diabetes was INR 6228 and the total median annual direct cost was INR 4800. The direct costs were contributed by medicines which constituted a large proportion of 59.06%, followed by laboratory tests 19.38%, consultation charges 12.46%, hospitalization 7.7%, transport 2.94 and food 1.96%. (Fig.1) The mean annual indirect cost was INR 1546. The mean amount spent while caring diabetic patient was INR 54.17 and the mean cost for the patient losing his income as a result of hospitalization which was INR. 813.98. The mean cost for the caregiver while accompanying the patient for hospitalization was INR. 27.71 and the mean cost of the caregiver losing his income while accompanying the patient for checkups was INR. 650.60. The total costs did not show a statistically significant difference among the two sexes (p=0.246), regularity of check ups (p=0.195), presence of complications of diabetes (p=0.858) and presence of co-morbidities (p = 0.249).

Our study showed that 58.5% of the study population had experienced less productivity at work due to their diabetic status and 2 persons had premature retirement (<60 years) due to their diabetic status. Three patients (7.3%) had given-up some form of diabetic care due to financial difficulty and all 3 of them had discontinued taking medications and laboratory tests in the last one year.

Discussion

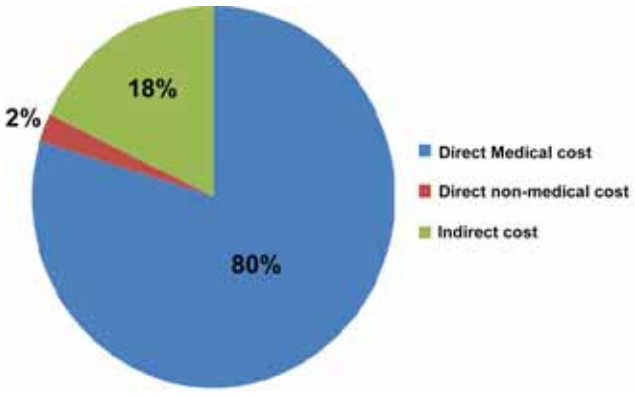

Fig. 1: Pie chart displaying the components of the annual cost of diabetic care

Table 1: Age and gender distribution of the study population

Age group (in years)	Males (%)	Females (%)	Total
31-40	3 (60)	2 (40)	5
41-50	16 (64)	9 (36)	25
51-60	21 (75)	7 (25)	28
>60	11 (44)	14 (56)	25
Total	51 (61.4)	32 (38.6)	83

Table 2: Estimation of the annual cost of diabetic care*

Costs	Total Expenditure	Inter quartile range	Mean cost †
Direct Costs	589,441	2314 - 8980	7101
Direct Medical Costs (total)	572,509	2064 - 8896	6897
• Medicines	319,724	936 - 5760	3852
• Lab tests	82,605	300 - 1200	995
• Consultation	46,680	200 - 800	562
• Hospitalisation	123,500	0 - 700	2872
Direct Non Medical Costs (total)	16,932	40 - 250	204
• Food	4,895	0 - 100	59
• Transportation	12,037	22 - 150	145
Indirect Cost (total)	128,355	0 - 2141	1546
• Cost incurred by caregiver	4,495	0 - 0	54
• Income loss to patient during hospitalization	67,560	0 - 0	814
• Income loss to caregiver during hospitalization of patient	2,300	0 - 0	28
• Income loss to caregiver while accompanying patient for check up visits	54,000	0 - 0	650

* All costs are expressed in Indian National Rupees (INR)
† Mean cost is calculated per patient per year

Indian studies estimate that, in low-income families as much as 25% of family income is allocated to the care of one adult diabetic.[5] The costs of diabetic care is not only a financial problem but also a social burden. Intangible costs (pain, anxiety, inconvenience and generally lower quality of life etc.) also have great impact on the quality of lives of the patients and their families; however they are the most difficult component of health care to quantify.[5]

There are three different types of costs involved in care of a diabetes patient. Direct costs to individuals and their families include medical care, drugs, insulin, hospital charges and other consumables. Costs range from relatively low-cost items, such as primary-care consultations and hospital outpatient episodes, to costly items, such as long hospital inpatient stays for the treatment of complications. Indirect costs include loss of job or loss of work efficiency. Sickness, absence, disability,

premature retirement or premature mortality can cause loss of productivity. Intangible costs include pain, anxiety, inconvenience and other factors which decrease the quality of life. Some activities may have to be foregone in favour of treatment, discrimination may be experienced in the workplace, obtaining jobs may be more difficult, and professional life may be shortened because of complications leading to early disability and even death. Personal relationships, leisure and mobility can also be negatively influenced.[5] For most countries, the largest single item of diabetes expenditure is hospital admissions for the treatment of long-term complications, such as heart disease, stroke, kidney failure and foot problems. Many of those are potentially preventable given prompt diagnosis of diabetes, effective patient and professional education and comprehensive long term care.[5]

Research of economic evaluation is still in its infancy in India. Literature shows that most of the studies are conducted in hospital settings. This may lead to non inclusion of data from patients who do not seek regular care which is very often the case in diabetes. The strength of our study is that it is a community based study which included not only patients seeking care at both government and private institutions but also those who did not seek any form of care at all. Unlike many previous studies, in the present study costs of diabetic care were assessed under three different subheadings namely the direct, indirect and intangible. The costs were assessed only from the patient's perspective. Finally, costs were assessed as comprehensively as possible and every effort was made to ensure that important components of cost were estimated with some degree of accuracy.

In our study, the direct costs were contributed by medicines which constituted a large proportion (59.0%) followed by laboratory tests and consultation charges. In a study done in coastal Karnataka, the total cost of care of diabetes was estimated at INR.14517.42 per person annually and the greatest bulk of it (42.4%) was attributed to the cost of drugs. [6] The average costs cited in the above study was much higher than that of our study. As the study of Sachidananda et al [6] was done among patients attending a diabetic clinic in a tertiary care hospital, it could have resulted in higher costs. Further, patients attending hospitals are more likely to have complications which increase the cost of care. The present study was done in the community with persons who visited both government and private clinics for diabetic car. Therefore the average cost of our study is lower than that of the study done in a hospital setting.

In a study done by the London School of Economics in 2010 in 5 European Union countries to assess prevalence of diabetes and the cost of its care, cost of inpatient care was consistently higher than that of outpatient care in all the countries.[7] This was attributed to increased medical care required in diabetes-related complications. Outpatient costs on the other hand, as well as diabetes medications, can be less than half of inpatient costs due to the relatively low costs of maintaining good glycemic control (medication and regular monitoring).[7] This study identified costs due to absenteeism, early retirement and expenditure on social benefits which amounts to a total of •98.4 billion across the study countries in 2010.[7] In a study done in Thailand, the total cost of diabetic care was estimated to be US $ 418,696 in 2008. [8] Of this, 23% was direct medical cost, 40% was direct non-medical cost and 38% was indirect cost.[8] This study was done in a hospital setting and hence show higher cost as compared to our study.

Kumpatla et al showed a huge increase in the cost of treating diabetic complications.[9] They showed that diabetic foot complications costed an average of INR. 19,020 per patient, those who had two complications spent four times more, and those with renal disease, cardiovascular and retinal complications spent three times more than patients without any complications. [9] But in our study there was no significant difference in the total costs incurred among persons who had complications of diabetes and those who did not ($p = 0.858$). This difference could be due to community based assessment which tends to dilute the concentration effect of hospital based studies.

Several studies done to assess the cost of diabetic care have been done in hospital settings. [10] In contrast the present study is one of the few studies done in the community. Thus it provides a realistic estimate of cost as experienced by diabetics. Further community-based studies are required to get a better picture of the economic implications of diabetic care and a larger sample size is also needed to generalise the results.

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ORIGINAL ARTICLE

Determination of serum zinc concentration in SGA neonates as compared to AGA neonates

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ABSTRACT

Background: Birth weight is the single most important determinant of infant survival in developing countries. Zinc is required to maintain the normal structure and function of multiple enzymes including those that are involved in transcription and translation of genetic material and cell division and growth and development.

Methods: The objective of the study was to compare serum zinc levels in small for gestational age (SGA) babies with respect to appropriate for gestational age (AGA) babies in a tertiary hospital whether term or preterm. 100 term and preterm newborns who are small for gestational age will comprise the study group and 100 newborns who are appropriate for gestational age will comprise control group. Venous blood sample will be collected immediately after birth and zinc levels will be determined by colorimetric method.

Results: The mean (±SD) serum zinc levels of the study and the control groups were 56.8 ± 40.6 µg/dl and 107.4 ± 72 µg/dl respectively. Statistically highly significant difference was found in the mean serum zinc levels between the two groups**.** The serum zinc level was lower in the study group as compared to control group. The mean serum zinc levels of the preterm SGA group and term SGA group were 46.26 ±22.54 µg/dl and 63.35± 47.47 µg/dl respectively. Statistically significant difference was found in the mean serum zinc levels between the two groups.

Conclusions: Maternal zinc level affects the weight of the baby but not the length or head circumference of the baby. So, zinc supplementation during pregnancy leads to normal birth weight.

Key words: Pre-term, Birth weight, Zinc level

Abbreviations: **SGA -** Small for gestational age; **AGA -** Appropraite for gestational age

Introduction

Birth weight is the single most important determinant of infant survival in developing countries. It is estimated to be responsible for >70% Perinatal deaths, 90% of neonatal deaths and 50% of infant deaths [1]. Low birth weight is associated with poor subsequent growth in infancy and childhood with increased morbidity from infectious diseases and compromised cognitive and behavioural development [2].

Zinc is required to maintain the normal structure and function of multiple enzymes including those that are involved in transcription and translation of genetic material and cell divison [3]. SGA born babies have small liver and inadequate stores of zinc**,** limited capacity to absorb and retain micronutrients and increased requirement for catch up growth, thus making them vulnerable for zinc deficiency [4]. This contributes to substantial neuro-cognitive, pulmonary and ophthalmologic morbidity.

Due to effect on DNA and RNA synthesis, Zinc helps in cell replication and differentiation of chondrocytes, osteoblasts, fibroblasts. In cell transcription and synthesis of somatomedin C, osteocalcin, alkaline phosphatise and

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metabolism of carbohydrates, proteins and fats. Zinc helps in hormonal mediation by growth hormone synthesis, secretion and action on somatomedin C in liver cells and activation of somatomedin C in bone cartilage. It helps in synthesis of Insulin, thyroid hormone and Vitamin D, all of which are required for growth [5].

MATERIALS AND METHODS

The study was conducted on 100 newborns with birth weight small for gestational age as study group delivered in Deptt. of Obstetrics and Gynaecology, Govt. Medical College, Patiala and admitted to neonatal section of Deptt. of Paediatrics, Govt. Medical College Patiala. 100 newborn term AGA and preterm AGA babies are taken as control group. Approval of ethics committee was taken along with written consent from the parents.

A detailed antenatal and clinical history of the mother covering personal history, past history of any medical illness like pregnancy induced hypertension, diabetes mellitus etc., previous obstetrical history like previous number of childbirths, abortions, perinatal loss, mode of delivery, family history of tuberculosis/diabetes mellitus/ hypertension etc., socio-economic status and present complaints were taken to rule out other causes that can affect zinc levels in babies.

Inclusion criteria

All SGA babies whether term (37-41 weeks) or preterm (<37 weeks) were included as test and AGA babies both term and preterm were taken as controls.

Exclusion criteria

All newborns whose mothers were having intrauterine infections, toxaemia of pregnancy, Diabetes mellitus, hepatitis, smokers and all birth- asphyxiated babies.

Serum Zinc level were estimated by 56.8 ± 40.6 µg/dl and 107.4 ± 72 µg/dl respectively in the hospital laboratory.

RESULTS

The male and female percentage of babies in the study group were 55% and 45% and in the control group were 56% and 44% respectively. The mean gestational age in the study group was 37.13± 2.33 weeks and 36.82 ± 2.50 weeks in the control group. The maximum number of cases in both the groups were in the 39th week period of gestation.

The mean (±SD) serum zinc levels of the study and the control groups were 56.8 ± 40.6 µg/dl and 107.4 ± 72 µg/dl respectively. Statistically highly significant difference was found in the mean serum zinc levels between the two groups. (Normal zinc levels- 60-120 µg/dl).

Table 1: Serrm zinc levels in study and control grow

Group	No.	Mean ±SD (µg/dl)	't' value	'p' value	Sig.
Study	100	56.8 ±40.6	-6.1	<0.001	HS
Control	100	107.4± 72			

Table 2: Serrm zinc levels in preterm SGA and Term group babies

Group	No.	Mean±SD (µg/dl)	't' value	'p' value	Sig.
Preterm SGA	38	46.26± 22.54	2.07	<0.05	Sig.
Term SGA	62	63.35± 47.47			

The mean serum zinc levels of the preterm SGA group and term SGA group were 46.26 ±22.54 µg/dl and 63.35± 47.47 µg/dl respectively. Statistically significant difference was found in the mean serum zinc levels between the two groups.

Table 3: Serrm zinc levels in term and preterm babies

Group	No.	Mean±SD (µg/dl)	't' value	'p' value	Sig.
Term	120	81.13 ± 52.25	-0. 277	> 0.05	NS
Preterm	80	83.68 ± 77.88			

The mean (SD) serum zinc levels of the term and preterm group whether they are SGA born or AGA born babies are 81.13 ± 52.25 µg/dl and 83.68 ± 77.88 µg/dl respectively. A statistically non significant difference was found in the mean serum zinc levels between the two groups (p> 0.05, as computed from the SEDM and the t-test).

DISCUSSION

In the present study, the mean (±SD) serum zinc levels of the study and control groups were 56.8 ± 40.6 µg/dl and 107.4 ± 72 µg/dl, respectively. Statistically, highly significant difference was found in the mean serum zinc levels between the two groups. A similar study done by Elizabeth et al (2007) showed the same observation in SGA babies taken in study group and term normal weight babies taken in control. Serum zinc levels were 70.25 ± 24.5, 78.09 ± 18.39 and 92.24 ± 19.4 in µg/dl respectively. The difference between study and control group was statistically significant.[6] Another study by Akram et al

(2011) compared serum zinc levels in babies born SGA with the babies born large for gestational age(LGA). Zinc levels were 78 µg/dl in the SGA group and 92 µg/dl in the LGA group. The difference was statistically significant in the two groups [7]. A study was done by Ozdemir et al (2007) on the estimation of zinc levels from cord blood in SGA, AGA and LGA babies. Zinc levels were below 100 µg/dl in the SGA group and 150 µg/dl in the AGA group. It showed significantly lower zinc levels in the SGA group. The method used for estimation was atomic absorption spectrophotometery [8].

CONCLUSION

The present study demonstrates that maternal zinc level affects the weight of the baby but not the length or head circumference of the baby. As prognosis of the baby in neonatal period and infancy regarding morbidity and mortality depends on the birth weight, various factors affecting the weight in utero should be duly considered. However more studies are required.

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ORIGINAL ARTICLE

Role of MRI in differentiation of Acute Disseminated Encephalomyelitis from Multiple Sclerosis

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ABSTRACT

Background: The differentiation between Acute disseminated Encephalomyelitis(ADEM) and Multiple sclerosis(MS) is of utmost importance from clinical prognostic view - point. The different imaging modalities are available; however, MRI plays significant role in prognosis. ADEM is monophasic acute inflammation and demyelination of white matter following recent viral infection or vaccination. Clinical presentation consistent with ADEM can also be the first manifestation of multiple sclerosis (MS), Particularly in children. MS in most patients is a long-lasting chronic disease, characterised by relapses, which may finally transform into a progressive disease. Symptoms unlike MS are more symptomatic rather then focal and include fever and multifocal neurological symptoms. ADEM is considered a disease caused by immune responses of the brain after a severe viral, bacterial or parasitic infection. It can also occur after vaccination MRI can also be helpful in differentiating ADEM from MS. Indistinct lesion borders, gray matter involvement, and diffuse or multilesional enhancement all favor the radiologic diagnosis of ADEM over MS also ADEM have diffuse bilateral lesion Pattern whereas MS have unilateral lesion pattern. ADEM lesions tend to resolve or remain unchanged with no new lesion formation, whereas appearance of new lesion is common in MS.

Method : Retrospective analysis of MRI scans of 18 children diagnosed with MS and 10 children with ADEM was performed T-2/FLAIR hyperintense lesions were categorised according to location, description, number and size.T1W1 before and post gadolinium administration were evaluated for the presence of lesion and post gadolinium enhancement. MRI findings were compared between the groups. Exclusion criteria is patient with orbit tumour and patient with implant.

Results: ADEM was seen in children <10 years (M=F) MS >10 years (M<F). History of prior infection was very frequent in ADEM whereas variable in MS. MRI findings to distinguish MS from ADEM are absence of diffuse bilateral lesion pattern, Presence of two or more periventricular lesions. However lesion margin are poorly defined (fuzzy) in ADEM and well defined (Dawson finger) in MS. Post-gadolinium shows patchy/absent enhancement in ADEM and focal enhancement in MS. On followup studies ADEM lesions are resolved whereas MS shows new lesions.

Conclusion: MRI is useful in differentiating patient with first attack of MS from those with Monophasic ADEM.

Key words: ADEM, MS, MRI

Abbreviations: **ADEM** - Acute disseminated Encephalomyelitis, **MS** - Multiple sclerosis

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Introduction

The differentiation between Acute disseminated Encephalomyelitis (ADEM) and Multiple sclerosis (MS) is of utmost importance from clinical prognostic significance. The different imaging modalities are available however MRI plays significant role in prognosis. ADEM is monophasic acute inflammation and demyelination of white matter following recent viral

infection or vaccination.[8] Clinical presentation consistent with ADEM can also be the first manifestation of multiple sclerosis (MS),Particularly in children. MS in most patients is a long-lasting chronic disease, characterised by relapses, which may finally transform into a progressive disease. [7]

Patients of ADEM, unlike MS are more symptomatic and include fever and multifocal neurological symptoms. ADEM is considered a disease caused by immune responses of the brain after a severe viral, bacterial or parasitic infection. It can also occur after vaccination [3][4] MRI can also be helpful in differentiating ADEM from MS. Indistinct lesion borders, gray matter involvement, and diffuse or multilesional enhancement all favor the radiologic diagnosis of ADEM over MS [9] also ADEM have diffuse bilateral lesion Pattern whereas MS have unilateral lesion pattern [6] and ADEM lesions tend to resolve or remain unchanged with no new lesion formation, whereas appearance of new lesion is common in MS. [8]

Aim and Objective

1. Role of MRI in differentiation of ADEM from MS
2. Role of Diagnostic differentiation between ADEM and MS for prognostic reason

Method and Material

Retrospective analysis of MRI scans of 18 patients diagnosed with MS and 10 patients with ADEM. T-2/ FLAIR hyperintense lesions were categorised according to location, description, number and size.T1W1 before and post-gadolinium administration were evaluated for the presence of lesion and post-contrast enhancement. Exclusion criteria is patient with orbit tumour and patient with implant.

RESULT

It was observed that 8/10 patients diagnosed as ADEM, were < 10 years age whereas 2 patients were between 20 to 35 years. 15/18 patients diagnosed as MS were female and between 20 to 50 years Whereas 3/18 patients were male between 50 to 70 years. History of prior infection was very frequent in ADEM whereas variable in MS. 11/18 patients diagnosed as MS had bilateral optic neuritis whereas 3/18 patients had unilateral optic neuritis and 4/18 patients had no findings of optic neuritis on MRI Scan. On MRI scan 9/10 patients diagnosed as ADEM had diffuse bilateral lesion Pattern whereas 1/10 had unilateral lesion. 10/18 patients diagnosed as MS had unilateral lesion pattern whereas 8/18 patients had diffuse bilateral lesion pattern. 7/18 patients diagnosed as MS had lesions in periventricular region 5/18 patients had lesions in corpus callosum region 3/18 patients had lesions in infratentorial region located at surface of pons and at the base of fourth ventricle 2/18 patients had multifocal basal ganglion lesion 1/18 patient had cortical infarct lesion. 6/10 patients diagnosed as ADEM had lesion in basal ganglia and 4 /10 lesion were seen in thalamus location.On MRI scan 16/ 18 patients diagnosed as MS had well defined lession (dawson finger) whereas 2/18 patients had illdefined margins. 7/ 10 patients diagnosed as ADEM had poorly defined (fuzzy) margin whereas 3/10 lesions had well defined margins. On followup MRI 8/10 patients diagnosed as ADEM had no lesions whereas 2/10 lesion remain unchanged with formation of no new lesions. 11/ 18 patients diagnosed as MS showed formation of new lesions 7/18 lesions remain unchanged.

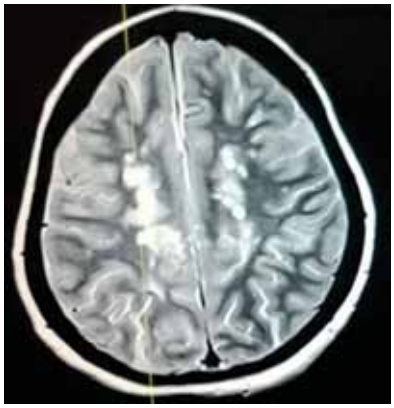

Fig. 1: Case of Multiple Sclerosis

Fig. 2: Case of Multiple Sclerosis

Fig. 3: Case of Multiple Sclerosis

Fig. 4: Case of Multiple Sclerosis

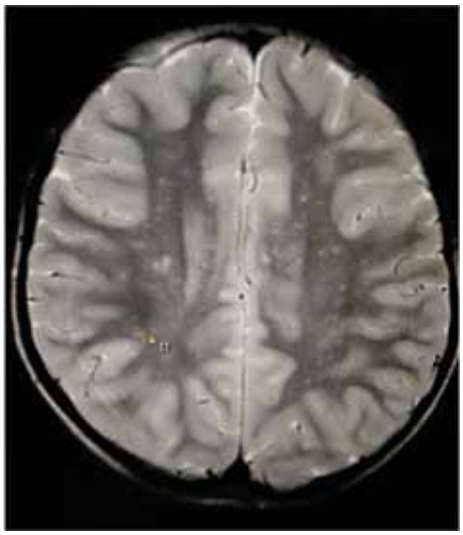

Fig. 5: Case of MRI ADEM

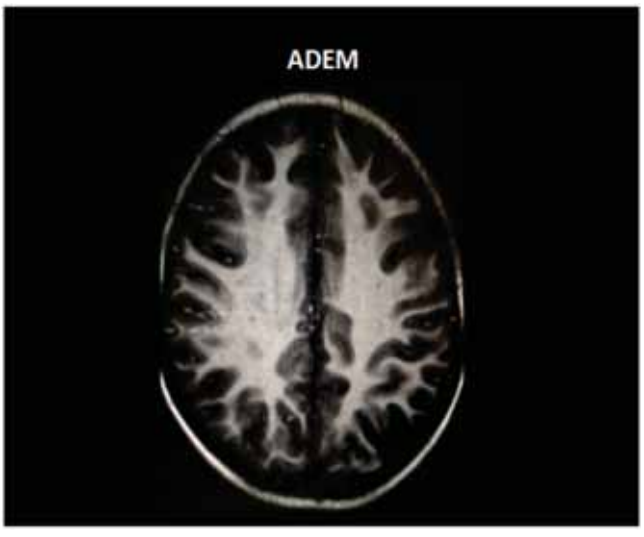

Fig. 6: Case of MRI ADEM

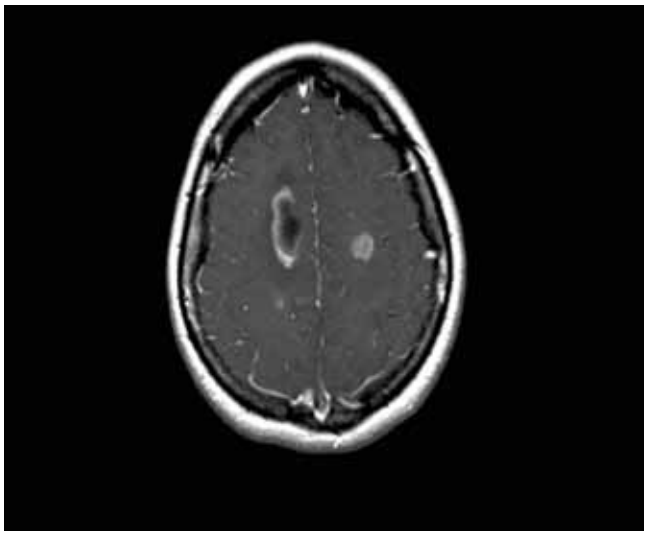

Fig. 6: Case of MRI ADEM

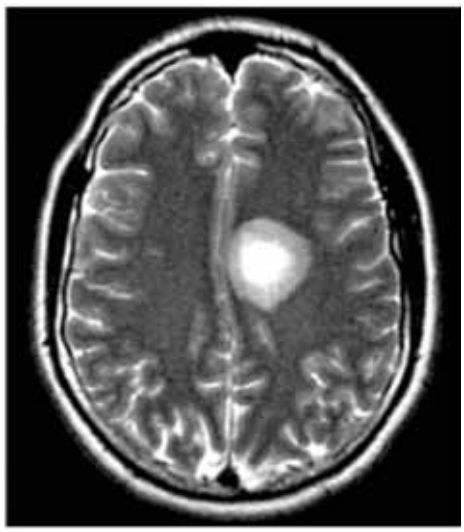

Fig. 8: Case of MRI ADEM

Discussion

In our study 8/10 patients diagnosed as ADEM were less then 10 years whereas 2/10 patients between 20 to 35 years; age distribution was similar to that reported by Marchioni E et al [1] which states ADEM is common in children and rare in adults. In present study 15/18 diagnosed as MS are females between 20 to 50 years whereas 3/18 patients were male between 50 to 70 years which is similar to Balcer L.C et al [2] in which MS was seen commonly in females between 15 to 49 years. History of prior infection was very frequent in ADEM whereas variable in MS which is similar to Schwarz S et al [3] and de seze J et al [4] study in which 73 percentage reported a recent infection or vaccination.11/18 MS patients have bilateral optic neuritis 3/18 have unilateral optic neuritis 4/18 patients have no finding of optic

neuritis which is similar to Frohman EM et al study. [5] On MRI scan 9/10 patients diagnosed as ADEM have diffuse bilateral lesion Pattern whereas 1/10 patient had unilateral lesion. 10/18 patients diagnosed as MS have unilateral lesion pattern whereas 8/18 patients have diffuse bilateral lesion pattern which is similar to study of Callen DJ et al [6] in which ADEM lesions had diffuse bilateral pattern mostly whereas MS lesions showed unilateral pattern. 7/18 MS lesion are located in periventricular region 5/18 lesions are located at corpus callosum 3/18 lesions in infratentorial region located at surface of pons and at base of 4th ventricle 2/18 lesions are multi focal basal ganglia lesions 1/18 are cortical infarct. 6/10 lesions of ADEM are located in basal ganglia and 4/18 lesions are located at thalamus. Which is similar to study of Polman CH et al [7] which states MS lesions are common in periventricular region, corpus callosum and infratentorial region and ADEM lesions are common in basal ganglia and thalamus.In present study 16/18 MS lesions are well defined (Dawson finger) whereas 2/18 lesions are ill defined. 7/10 ADEM lesions have poorly defined margins whereas 3/10 lesions have well defined margin which is similar to Polman CH et al [7] Which states ADEM has mostly indistinct lesion border whereas MS has well defined Dawson finger appearance. In present study on follow up MRI 8/10 patients diagnosed as ADEM had no lesions whereas in 2/10 lesions remained unchanged with formation of no new lesions. 11/18 MS lesions showed formation of new lesions 7/18 lesions remain unchanged which is similar to Wingerchuk DM et al [8] study which states ADEM lesions tend to resolve or remain unchanged with no new lesion formation, whereas appearance of new lesion is common in MS.

Conclusion

In most but not all cases, ADEM occurs only once, while patients with MS have, repeated attacks of inflammation in their brains. In most cases, ADEM patients do not develop new scars on a repeat MRI scan whereas MS patients typically experience new scars on their follow-up MRI scans. Typical symptoms of ADEM such as fever, headache and confusion, vomiting, and seizures are not usually seen in people with MS, although they can be seen in paediatric MS onset especially in patients younger than 11 years. Pattern of MRI abnormalities helps in differentiating these two disorders. Most patients with MS are treated with ongoing medication to prevent attacks. Patients with ADEM do not require such medication.ADEM occurs more frequently in males; MS more frequently in females.ADEM is more common in children; MS is more common in adults.

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Ethics:	There is no ethical violation as it is based on voluntary anonymous interviews
Funding:	No external funding
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ORIGINAL ARTICLE

Role of Color and Duplex Doppler in Differentiating Malignant from Benign Thyroid Nodules

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ABSTRACT

Background: Thyroid nodules is a common clinical problem and the chances of malignancy in solitary thyroid nodule is quite variable and recently reported to be as high as 34.4%. Ultrasonography is the most useful and a commonly employed modality to evaluate thyroid nodules with the main concern to differente malignant from benign nodules. Doppler is expected to enhance its diagnostic capabilities through the Doppler parameters (RI, PSV) of the intra- and the peri- nodular vessels. The type of flow in the nodule, correlation of the nodule size and the vascularity and changes in the Doppler characteristics with malignancy are the areas to raise curiosity in the minds of the researchers.

Aim: The purpose of study was to evaluate the role of color and spectral Doppler in differentiating benign from malignant thyroid nodules.

Methods: Doppler examination was performed on 61 nodules in 47 patients and recorded the pattern of vascularity on colour Doppler while resistivity index and peak systolic velocity was determined in both the peri-nodular and intra-nodular regions using spectral Doppler. All of these nodules subsequently underwent ultrasound guided fine needle aspiration cytology.

Results: Out of 61 nodules, 9 nodules were malignant while 52 nodules were benign on fine needle aspiration cytology. Six (66.67%) of nine malignant thyroid nodules and five (9.62%) of 52 benign nodules had predominately intranodular flow. Predominately intranodular flow had sensitivity, specificity, PPV and NPV of 66.67%, 90.38%, 94% and 54.54% respectively for diagnosing malignancy in thyroid nodules. Malignant thyroid nodules had significantly (P value=0.015) higher intra nodular mean systolic velocity (22.08cm/sec) in comparison to benign nodules (13.49cm/sec) while there was no significant (P value=0.953) difference in mean systolic velocities in peri nodular region. The resistivity indices of malignant nodules in both peri nodular (0.84) and intra nodular region (0.77) was not significantly different than those of benign nodules (peri nodular RI= 0.83 and intra nodular RI=0.83) with P vaues of 0.963 and 0.527 respectively.

Conclusion: The pattern of vascularity and intra-nodular peak systolic velocity can be used as color and spectral Doppler parameters to differentiate benign from malignant thyroid nodules. Intranodular flow is strongest predictor for diagnosing malignancy among color Doppler parameters.

Key words: Thyroid Nodules, Diagnosis, Ultrasonography, Doppler, Blood Flow velocity, Vascular resistance

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Introduction

Thyroid nodules are a common medical problem with approximately 8% of the adult population having clinically apparent nodules. The prevalence of palpable thyroid nodule in South India is about 12.2% [1]. The detection of these nodules has increased multifold due to the use of Imaging in the recent times [1,2]. Most of the thyroid nodules hyperplastic, with only 5-20% being true

neoplasms [3].Thyroid cancer has been reported variably but occurs in approximately 5% of all thyroid nodules independent of their size [4] and the prevalence is known to increase in the recent times. [4, 5] . The incidence of malignancy in solitary nodule is seen in up to 16.5% of population residing in the costal region in an Indian study [6]. Keh et al have reported 34.4% of thyroid nodules to be malignant in their retrospective study [7]. Therefore, it is extremely important to differentiate the malignant thyroid nodules from the benign lesions.

The role of gray scale ultrasound (USG) in diagnosing thyroid malignancies has been extensively studied and features like hypoechogenicity, microcalcifications, and irregular margins have been found to be associated with malignant thyroid nodules. [8,9,10]

Malignancy is known to be associated with increased cellular proliferation leading to increased vascularity. Addition of Doppler to gray scale USG can increase the diagnostic performance of US for better detection of the malignant nodules. Five patterns of vascularity have been described in the thyroid nodules on color as well as on power Doppler examination. [11] .Even if fine needle aspiration cytology (FNAC) does not reveal malignancy nodules suspicious of being malignant on US can be kept under close Doppler follow-up and FNAC repeated whenever required.

Researchers have shown that increased intranodular flow on color Doppler examination helps in diagnosing thyroid malignancies[12,13]. While others concluded that malignant thyroid nodules have significant higher resistive index (RI) than benign thyroid nodules[14,15]. Further, power duplex Doppler facilitates scanning of thyroid nodules at risk of malignancy with increased sensitivity (92.3%) and specificity (88%) [11] . Chamas et al also demonstrated that RI was higher in malignant as compared to the benign thyroid nodules [11]. On the contrary Tamsel et al did not find any significant difference between the malignant and benign thyroid nodules with respect to the mean RI of either the intranodular (mean RI = 0.60) or the perinodular (mean RI=0 .58) vascularity[16]. Similarly the mean peak systolic velocity (PSV) was also not significantly different in both the intra- and perinodular vessels between the benign and the malignant nodules [16]. As there are conflicting reports regarding the role of Doppler in diagnosing thyroid malignancy, we conducted this study to determine the color and spectral Doppler characteristics of thyroid nodules for differentiating malignant from benign nodules.

Patients and Methods

After obtaining due clearance from the institutional review board, we performed a cross sectional study on 47 patients referred to the department of Radiology for a sonographic evaluation of thyroid gland due to clinical suspicion of thyroid disease. Patients with thyroid nodules detected clinically or on sonography and consenting to be a part of the study were included while patients refusing consent or having a FNAC proven diagnosis of the type of thyroid nodule were excluded from the study.

Sonographic Technique

The sonography was performed using Philips ATL HDI 5000 Bothell, WA, USA; equipped with a phased array linear broadband 5 MHz to 12 MHz transducer. The routine sonographic evaluation of the thyroid and the detected nodule was done. All the nodules were subjected to Doppler study. In cases where multiple nodules were detected one representative nodule was selected and interrogated on Doppler examination. All the nodules having different sonographic appearance underwent Doppler in multi-nodular goiter. The flow settings were adjusted to detect even the slow flow in the nodule without appearance of color artifacts. The vascular pattern of thyroid nodules was classified as one of the following as shown in table1.

Table 1: Type of vascularity in the thyroid nodules

Type of vascularity	Description
Avascular	Absence of any color or vascularity
Peri-nodular	Nodules with only peri-nodular or predominant peri-nodular flow
Both peri- and intra-nodular	Presence of both intra and peri-nodular vascularity
Intra-nodular	Nodules with predominately intra-nodular flow or only intra-nodular flow.

Each vascularized nodule was then evaluated on Duplex Doppler examination. The resistivity index (RI) and the peak systolic velocities (PSV) were recorded for three vessels both in the peri-nodular and the intra-nodular region and the average RI and PSV was then calculated for peri-nodular and intra-nodular vascularity. The nodule evaluated on Doppler was recorded diagrammatically on a representative image of the thyroid gland and the same nodule/ nodules were subjected to US guided FNAC.

Fine Needle Aspiration Cytology

Ultrasound guided FNAC was done from the nodules in collaboration with an experienced pathologist. FNAC was taken from every nodule that had been evaluated by color and spectral Doppler. If the sample taken at first attempt seemed inappropriate, a maximum of three attempts per nodule were made.

Both air dried and Pap wet fixed slides were made with the aspirated material. The air dried and the Pap slides were fixed by methanol and ethanol respectively and the air dried slides stained by May Gounwald Giemsa stain. The pathologist was blinded to imaging findings categorized the nodules as: malignant, benign or indeterminate based on the cytology result. A repeat FNAC was done in nodules with previously non-diagnostic/indeterminate results.

A statistical analysis was done using mean test for quantitative data and Fisher's exact test for qualitative data.

Results

A total of 47 patients of which 42 (89.36 %) were females, twenty nine patients had solitary thyroid nodule while eighteen had multinodular thyroid disease. The mean age of the patients was 41.43 years. A total of 61 nodules were identified for evaluation with color and spectral Doppler examination.

FNAC revealed that out of 61 nodules 45 were benign, 8 were malignant and 8 were non- diagnostic on first FNAC examination. One out of eight non- diagnostic nodules underwent surgery and was malignant on histopathological examination, while the rest seven were benign on repeat FNAC examination. Therefore the final study constituted of 52 benign and 9 malignant nodules.

No vascularity was appreciated in 4 (7.69%) of the nodules, all of which were benign. 57 (93.44 %) nodules

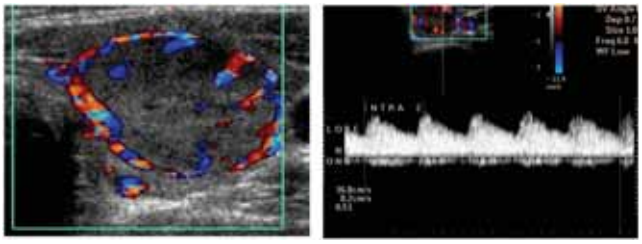

Fig. 1: a Fig. 1: b

Fig. 1: a) Showing an isoechoic solid nodule in the left thyroid lobe with peri nodular vascularity on color Doppler Imaging and b) Duplex Doppler shows a low resistance flow, with RI of 0.51. Histological diagnosis was a benign- Adenomatous nodule.

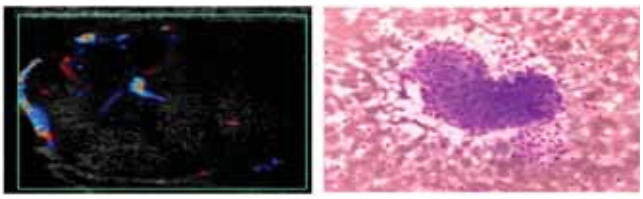

Fig. 2: a Fig. 2: b

Fig. 2: a) Color Doppler image showing both intra and perinodular vascularity and b) Photomicrograph at 200x of cytological specimen from thyroid gland showing increased number of thyroid cells with benign features s/o adenomatous goiter, benign nodule

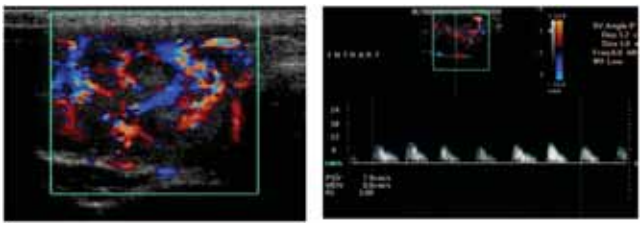

Fig. 3: a Fig. 3: b

Fig. 3: a) Right thyroid lobe shows an isoechoic nodule with showing predominately intra-nodular vascularity and & b) Duplex Doppler shows high resistance flow with RI of 1. Cytology revealed a Medullary carcinoma- a malignant nodule.

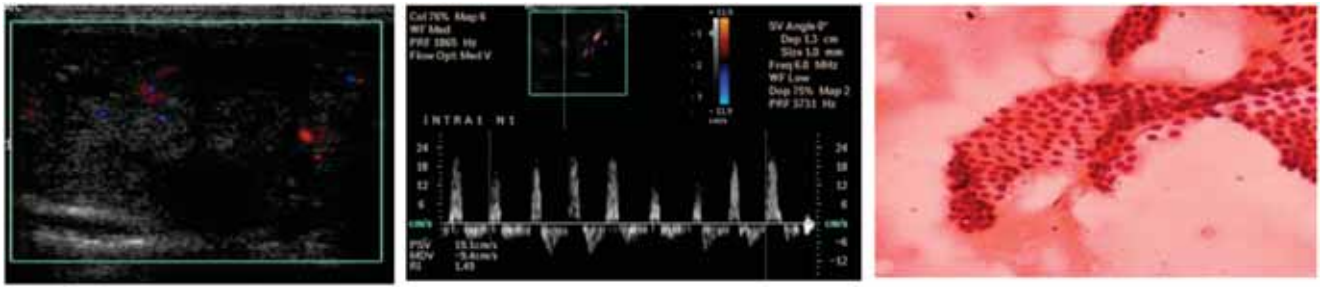

Fig. 4: a Fig. 4: b Fig. 4:c

Fig. 4: a) Color Doppler image showing predominately intra-nodular vascularity heteroechoic thyroid nodule with lobulated margins and foci of micro b) Duplex Doppler image showing high resistance intra-nodular flow (RI of 1.49) c) Photomicrograph at 200x of Pap smear showing thyroid cells with spindle shaped nuclei with grooving and irregular margins suggestive of papillary carcinoma a malignant thyroid nodule.

showed vascularity on colour Doppler imaging. Six (66.67%) of nine malignant thyroid nodules and five (9.62%) of 52 benign nodules had intranodular flow. Perinodular vascularity was present in 31 (50.82%) nodules and all were benign in nature. (Fig. 1) Of 15 nodules displaying equal peri-nodular and intra-nodular vascularity 12 were benign and 3 were malignant. (Fig. 2) Therefore, 23.07% of the benign and 33.33% of the malignant nodules demonstrated both perinodular and intranodular vascularity. (Table 2). All the 9 malignant nodules had intranodular vascularity. (Fig. 3 & 4).

Table 2: Distribution of color Doppler characteristics among benign and malignant thyroid nodules

Colour Doppler Characteristics	Benign (n=52) No. (%)[+]	Malignant (n=9) No. (%)*	Total (n=61) No. (%)[@]
Absent vascularity	4 (7.69%)	0 (0%)	4 (6.56%)
Perinodular vascularity (predominately peri-nodular or only peri-nodular flow)	31 (59.62%)	0 (0%)	31 (50.82%)
Equal distribution of vascularity	12 (23.07%)	3 (33.33%)	15 (24.59%)
Intranodular vascularity (predominately intra-nodular or only intra-nodular flow)	5[++] (9.62%)	6[#] (66.67%)	11 (18.03%)

[+]Percentage is indicated taking 52 as 100 %, [++]All had predominant intra-nodular flow
* Percentage is indicated taking 9 as 100, @ Percentage is indicated taking 61 as 100 %
[#] two of the six malignant nodules showing predominately intranodular vascularity had only intranodular vascularity.

Table 3: Predictive value of color Doppler characteristics suggestive of malignancy in a thyroid nodule.

Color doppler characteristics	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Intranodular vascularity	100	32.69	20.45	100
Perinodular vascularity	88.89	7.69	14.28	80
Predominately intranodular vascularity	66.67	90.38	94	54.54

Table 4: Duplex Doppler parameters of resistive index and peak systolic velocity in thyroid nodules for both malignant and benign thyroid nodules.

	Resistive Index (RI) (mean±2 SD)			Peak systolic velocity (PSV) (mean±2 SD in cm/s)		
Type of vascularity	Benign	Malignant	P value	Benign	Malignant	P value
Perinodular	0.83±0.19	0.84±0.17	0.963	18.75±10.156	19.60±16.86	0.953
Intranodular	0.83± 0.27	0.77±0.28	0.527	13.49± 6.58	22.08± 15.51	**0.015***

* Significant

For diagnosis of a malignant nodule intra-nodular flow had the highest sensitivity of 100%, but a very low merely 32.69% specificity. The PPV and NPV were 20.45% and 100% respectively for intra-nodular blood flow. Presence of perinodular vascularity had a sensitivity, specificity, PPV and NPV of 88.89%, 7.69%,80%, 14.28% and 80% respectively for diagnosing malignancy in thyroid nodules. Predominant intra-nodular flow had a sensitivity, specificity, PPV and NPV of 66.67%, 90.38%, 94% and 54.54% respectively for diagnosing malignancy in thyroid nodules. (Table 3)

Discussion

A total of 61 nodules of which 9 patients were subject to malignant and the rest 52 were benign on FNAC. Color and spectral Doppler examination in order to differentiate between benign and malignant thyroid nodules.

It was found that intranodular vascularity had strong association with malignancy in thyroid nodules (P value =0.015).

When studied as an independent predictor it had sensitivity, specificity, PPV and NPV of 66.67%, 90.38%, 54.54% and 94% respectively for detection of malignancy in thyroid nodules. According to our findings intranodular flow is strongest predictor for diagnosing malignancy among color Doppler parameters.

In concordance to our findings Cerbone et al 1999 [15], reported that predominately intranodular blood flow has a sensitivity of 98.1% and specificity of 100% for differentiating benign from malignant thyroid nodules. They explained that malignant lesions have more cellular proliferation in intranodular region. They also reported that there was no significant correlation between nodule size and vascularity. Papini et al [12] was in agreement and also reported intra-nodular vascularity to be significant predictor of malignancy in thyroid nodules. They reported a sensitivity of 74.2% and specificity of 80.8% for this feature.

Though Frates et al [13] showed that solid hypervascular thyroid nodules have high risk of malignancy, but found that it is a non specific sign as more than 50% of the hypervascular solid thyroid lesions were benign.

However, present results are not concordant with findings of Wienke et al [17] and Iannuccilli et al [18] who found that benign nodules also exhibited high grades of intranodular vascularity. Similarly, Tamsel et al [16] found that intranodular vascularity is the most common vascular pattern for both benign and malignant thyroid nodules and it cannot be used as a predictor of thyroid malignancy because of its low specificity. This difference in the studies can be attributed to the relative larger size of the thyroid nodules in their study as compared to ours; as with growth of the benign nodule there is progressive recruitment of intra-nodular vessels.

Chan et al [19] reported that all papillary thyroid carcinomas in their study had some intrinsic blood flow, and they concluded that a completely avascular nodule is very unlikely to be malignant. This was in total agreement to the findings in our study as all the avascular nodules in our study were benign and all the malignant nodules showed presence of intra-nodular blood flow.

The presence of large quantities of stenosis and occlusions in the neo-vascularisation of thyroid carcinoma tend to increase the vascular resistance and thus the resistive index.

The duplex Doppler findings showed no statistical significant difference between benign and malignant thyroid nodules when resistivity index was studied as parameter for diagnosing malignancy both in the peri-nodular as well as the intra-nodular blood flow. In concordance to these results, Tamsel et al [16] reported that duplex Doppler can not differentiate between benign and malignant thyroid nodules and they did not find any statistically significant difference between resistive indices of benign and malignant thyroid nodules.

However there is gross disagreement with Cerbone et al [15] and Chammas et al [11] with respect to the RI between the benign and malignant thyroid nodules. The former reported a significant difference in RI of benign and malignant thyroid nodules and suggested a cut off value of 0.75 for RI of intra-nodular vessels for predicting malignancy in thyroid nodules. Chammas et al [11] found that thyroid nodules with RI of >0.77 in intranodular location were at high risk for malignancy.

A statistically significant difference was found in the peak systolic velocities in intranodular location of benign (13.49 cm/sec) and malignant thyroid nodules (22.08cm/sec), but peak systolic velocities in perinodular location were not significantly different. When a cut off value of 16cm/sec was taken for peak systolic velocity in intranodular location for diagnosing malignancy in thyroid nodules, it had a sensitivity of 55.55% and specificity of 78.85%.

Cerbone et al [15] suggested Power Doppler to have a higher sensitivity (100 vs. 91%) and specificity (95.1 vs. 86.2%) than color Dopler in investigating thyroid nodules in comparison to the results of cytology. However, in our study neither power Doppler nor duplex power Doppler was used to evaluate the thyroid nodules.

Conclusion

From our study we concluded that intranodular flow and increased intranodular peak systolic velocity can be used as predictors of malignancy in thyroid nodules, while resistivity index is not helpful as both benign and malignant thyroid nodules can show high resistivity index in both the peri-nodular and intra-nodular blood flow. Intranodular flow is strongest predictor for diagnosing malignancy among color Doppler parameters.

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ORIGINAL ARTICLE

Enhanced Image Registration Technique for Medical Image Segmentation

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ABSTRACT

In image processing and image analysis the final result is obtained by combining information from various sources. To obtain better result from combined information, image registration plays an important role, which is an earlier step in image segmentation. Generally it follows feature extraction, feature matching, and transformation and sampling, dominant extracted features and matching algorithm gives the better registration accuracy. In proposed system contourlet transform and mutual information is combined to increase the accuracy in registration process. Contourlet transform extracts efficient curves and edges from MRI images, these features from MRI brain images helps to match the information using mutual information. Performance comparison of proposed results shows high performance using Contourlet transform.

Key words: Feature extraction, Transform modeling, Image resampling, Contourlet transform, Mutual Information.

Introduction

The processing of transforming different forms of data into single co-ordinate system is called image registration. Data may be different images, data which is reconstructed from different sensors, depths, times or viewpoints [1]. It is used in computer vision applications, medical imaging, biological imaging, and satellite imaging. Registration is important in order to match or fuse the data which is obtained from different measurements. The discrete expansion of curvelet transform is known as contourlet transform, first it is processed in continuous domain [2] using multi-scale filtering and then undergo with a block ridge let transform [3] on each band pass data, later it is directly used in frequency partitioning without using ridge let transform . Instead of key points contourlet transform captures curves; apart from curvelets and contourlet many transformation techniques are developing which efficiently represent geometrical regularity, for example band lets, the edge-adapted multiscale transform [3] etc, but these techniques need edge detection stage which is followed by adaptive representation.

Contourlet representation is a type of fixed transform, these characteristics made contourlet transform to be effectively applied in a computer vision tasks, similar to wavelets. In computer vision techniques Magnetic Resonance Imaging (MRI) play an important role, it is used in medical examination, assistant diagnosis of brain tumors and breast cancers owing to its high resolution to soft tissues and no damage to human body during acquisition. Medical knowledge and experience of doctors can obtain the information's like sizes, locations, shapes and other pathological features of brain tumors. According to the information in MRI images are used to make scientific and reasonable therapeutic treatment. Because there are several MRI examinations for every patient in the whole therapeutic treatment, each of which can give data in multiple sequences, it is a large amount of data to be dealt with for the doctors. Long time of hard work will inevitably lead to mistakes in the diagnosis of the tumor contours for the doctors. Moreover, it is subjective for the doctors to determine the state of the diseases according to their medical knowledge and clinical experiences. Therefore, developing an automatic or a semi-automatic computer-aided diagnosis system is meaningful in real medical treatments, which can release the workload of doctors and improve the accuracy by

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giving objective results. Resulting problem is an important point in the research field of medical image processing and medical engineering; a lot of algorithms and work have been proposed to try to solve this problem.

Proposed Methodology

Brain tumor detection and classification algorithms normally uses T2 weighted MRI slice image, but in some cases we cannot assure that we can get correct vision of tumor, it is necessary to fuse the multiple information like T1 weighted MRI slice and PD MRI slice to get accurate result. Here we use registered images for good image fusion result, so image registration is important pre-processing step in our system. The block diagram of our methodology is shown in Fig. 1. The three slices of a image is considered for the input image. These images are preprocessed. The Contourlet transform is applied to the preprocessed images and edges are extracted to carry out registration process. These registered slices of images are fused then region growing method is applied for brain tumor segmentation by user interface selecting the location.

Normally image registration techniques carried out by following 4 general steps, i.e. from Feature Extraction to Image Resampling. In feature extraction method salient and dominant features are extracted from sensed and reference images automatically and both image features are compared. In proposed methodology Contourlet Transformation model is used to represent the geometrical parameters. The medical images are characterized by curved shapes and contours. Further limited work has been done in the area of medical imaging using Contourlet Transform.

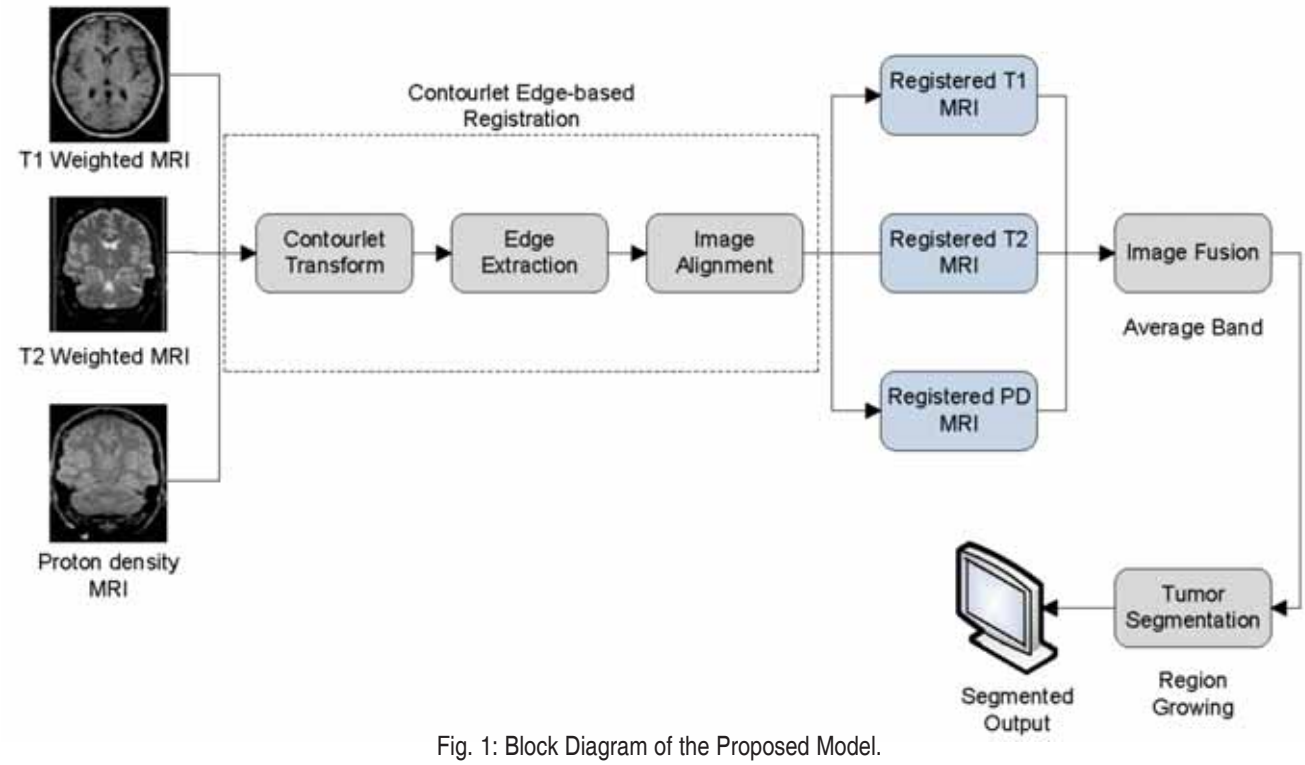


Fig. 1: Block Diagram of the Proposed Model.

Proposed Algorithm Steps:

- Input as source image and image to be registered
- Apply contourlet transform on both the image and reconstruct the images
- Calculating the joint histogram values of both reconstructed images
- Mutual information and its maximum value are determined
- Scale and the angle of rotation are computed
- Rotate the image to angle of rotation computed and scale the image to scale value obtained

A. Contourlet Transform

In signal processing tasks like de-noising, compression, enhancement and feature extraction, by Contourlet Transform the image is represented efficiently. This transforms aimed to improve the sparse representation

of the image over the Wavelet Transform. The ability to capture directional information is limited in the wavelet transform, which is the main drawback. The multi-scale and directional representation of image is considered in which the intrinsic geometrical structures is captured. The properties of the Contourlet Transform: directional property i.e., basis functions at number of directions, and the anisotropy property i.e., basis functions at appears at different aspect ratios. When the other geometrical representations are compared to the Contourlet Transform, it is observed that the Contourlet Transform is efficient wavelet and relatively simple.

The purpose for which is to represent as directional multi-resolution image that captures and represent the singularities efficiently with smooth object boundaries. Its lower redundancy and the filter bank construction which is efficient make the contourlet transform an attractive computational framework. The dimensional filter bank used in contourlet transform decomposes the image into many subbands at multiple-scales. It is accomplished with Laplacian pyramid and directional filter bank at each scale. The first directional decomposition and second multi-scale decomposition stages are independent because of this contourlet transform. Each scale can be decomposed into arbitrary power of 2's number of directions and different scale can be decomposed into different numbers of directions. This helps contourlet to achieve flexibility in process of decomposition.

Here f(t) is the 2D function and it decomposes to scale J with j1 directions using discrete contourlet transform [7].

$$a_J[\vec{n}] =< f, \phi_{J,n} >= \int_{\vec{t}} f(\vec{t}) e^{-j} \phi(2^{-j}\vec{t} - \vec{n}) \overrightarrow{dt} \quad (1)$$

$$c_{J,k}{}^{(l_j)}[\vec{n}] =< f, \lambda^{(l_j)}{}_{J,k,n} >= \int_{\vec{t}} f(\vec{t}) e^{-J} \lambda^{(l_j)}{}_{j,k,n}(\vec{t}) \overrightarrow{dt} \quad (2)$$

$$\lambda^{(l)}{}_{j,k,n}(\vec{t}) \overrightarrow{dt} = \lambda^{(l)}{}_{j,k}\left(\vec{t} - 2^{j-1} S_k^{(l)}(\vec{n})\right) = \sum_{\vec{x} \in Z^2} d_k^{(l)}(\vec{x}) \mu_{j,x}\left(\vec{t} - 2^{j-1} S_k^{(l)}(\vec{n})\right); \vec{n} \epsilon Z^2 \quad (3)$$

$$\mu_{j,2n+k_l}(\vec{t}) = 2^{-j} \psi^{(i)}(2^{-j}\vec{t} - \vec{n}) \quad (4)$$

$$S_k^{(l)} = \begin{cases} diag(2^{l-1}, 2) & for\ 0 \le k < 2^{l-1} \\ diag(2, 2^{l-1}) & for\ 2^{l-1} \le k < 2^l \end{cases} \quad (5)$$

Where $0 \le i \le 3$, $0 \le k \le 2l-1$, $j = 1,2,\ldots,J$, and i k is a downsampling rate with ratio 2 for each dimension.

$$k_0 = (0,0)^T$$
$$k_1 = (1,0)^T$$
$$k_2 = (0,1)^T$$
$$k_3 = (1,1)^T$$

Therefore, we rewrite "(3)" as

$$\lambda^{(l)}{}_{j,k,n}(\vec{t}) = \sum_{i=0} \sum_{\vec{x} \in Z^{23}} d_k(\vec{x}) 2^{-j} \psi^{(i)}(2^{-j}\vec{t} - 2^{-1} S_k \vec{n} - 2^{-1}\vec{x} + 2^{-1} k_i) \quad (6)$$

$a_j[\vec{n}]$ and $c_{J,k}{}^{(l)}[\vec{n}]$ are approximation coefficients and detail coefficients respectively as in "(2)". $W_{j,k}^{(l)}$ is several directional subband which is decomposed using filterbank. Contourlet function $\lambda^{(l)}{}_{j,k,n}(\vec{t})$ has compact support with width of c2 j and length of j +l - 2 in the scale j. Also, it has L-order directional anishing moment (DVM). For a contourlet function $\lambda^{(l)}{}_{j,k,n}(\vec{t})$ constructed from an iterated filter bank as in "(5)", it has an L-order DVM along direction u if the discrete-time Fourier Transform $W_k^{(l)}(e^{j\omega_1}, e^{j\omega_2})$ the associated filter $W_k^{(l)}[\vec{n}]$ also has L-order zeros along the line $u1w1 + u2w2 = 0$.

$$\gamma_{j,k}^{l}(\vec{t}) = \sum_{\vec{m} \in z^2} w_k^{(l)}[\vec{m}] \emptyset_{j-1,m}(\vec{t}) \quad (7)$$

The frequency partition is done using contourlet transformation is as shown in Fig. 2. The scales are divided to four, eight directional subbands and from coarse-scales to the fine-scales respectively.

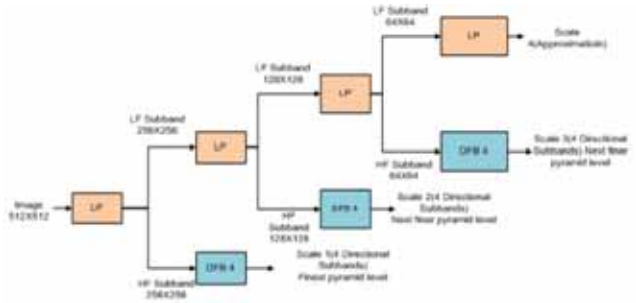


Fig. 2: Decomposition process of Contourlet Transform for a 512 X 512 image using Laplacian pyramid. [7]

Contourlet Transform uses pyramidal Directional Filter Bank (DFB) for a local, multi resolution and directional expansion of image. The DFB is combination of Laplacian Pyramid captures the discontinuity points, and directional filter banks links these discontinuities into linear structures. Fig. 2 shows the dataflow of Contourlet Transform for a 512X512 image.

B. Mutual Information

The measurement of amount of common information between two images is known as Mutual Information (MI). The different transformation estimates during the image registration are evaluated [7]. These transformations determine that variations in the degree of overlap between images which is better than entropy. Mutual information can also be considered as measurement of how one image explains well the other

image; it is computed using "8". It is maximized at the optimal alignment.

$$I(A,B) = \frac{H(A) + H(B)}{H(A,B)} \quad (8)$$

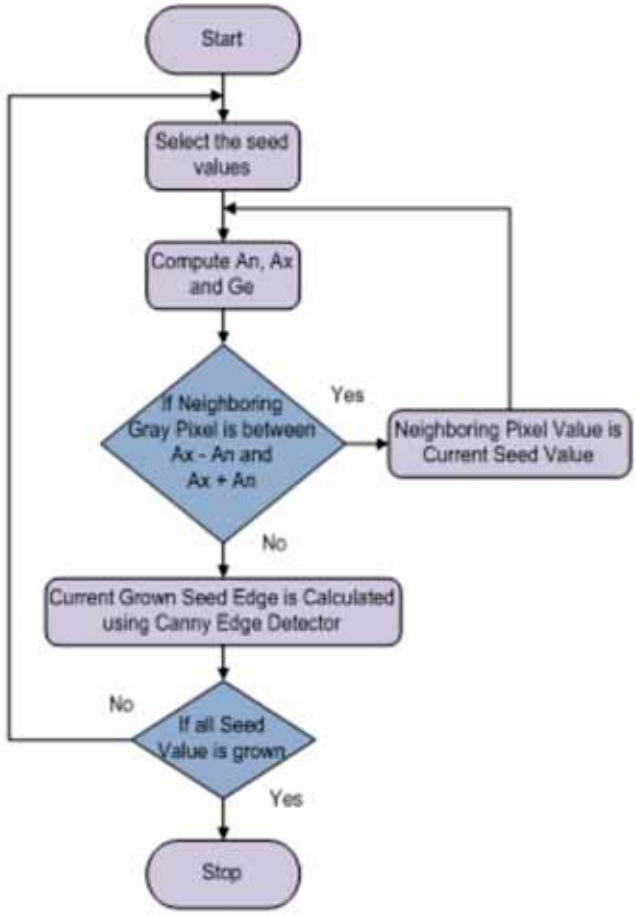


Fig. 3: Flow Diagram Region Growing Algorithm. [14]

C. Region Growing Algorithm

Region growing segmentation algorithm provides better segmentation results for MRI brain images, because algorithm performs well with respect to noise. Fig. 3 shows the flow diagram of region growing; the first procedure is to select seed point or set of seed points in an image, the initial growing procedure starts from the exact location of seed point, before that expected value(Ax), entropy(An), and hyper entropy(Ge) are computed using cloud model computing[15]. By using this computed characteristics region of interest is calculated and segmented using canny edge detector.

Results

The proposed method takes the multimodal images as its input. The contourlet transform is applied to both the source and target images or sensed and reference images. The images are reconstructed after truncating to the most significant bits. Then the image registration is done using mutual information.

There two types of data: source image and target image. Target image is the one which is to be registered. Source image is the one which is taken as reference image for registration. Source image can be considered as the original image and Target is the rotated and scaled image, which is to be registered to the source image considered.

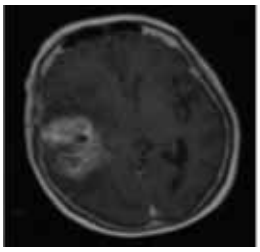


Fig. 4 (a) Source Image

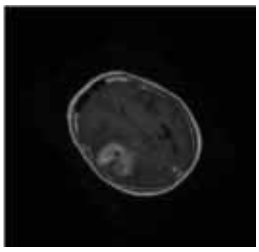
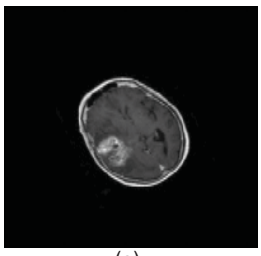


Fig. 4 (b) Target Image

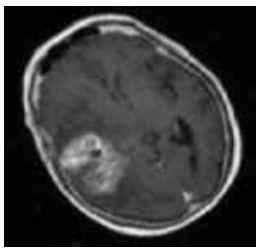
The image in Fig. 4(a) is the source image and image in Fig. 4(b) is the target image that to be registered. Here image registration is done using mutual information. Therefore the source image must be mapped to target image and registration is done. Joint histogram features are calculated from both source image and target image and extracted features are normalized using "(9)", where J_h is the joint histogram similarly Marginal Entropy is calculated from normalized histogram using "(10)" where N_h is the normalized histogram. Mapping source image with target image during registration process is done by using mutual information as shown in "6".

$$N_h = \frac{J_h}{No\ of\ rows * No\ of\ Coloums} \quad (9)$$

$$Hy = Hy + N_h * (log2 * \quad (10)$$



(a)



(b)

Fig. 5: Intermediate Results (a) Image when rotated and scaled (b) Image when matched with the Reference Image

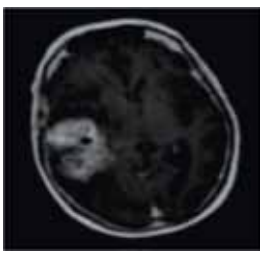


Fig. 6: Registered Image

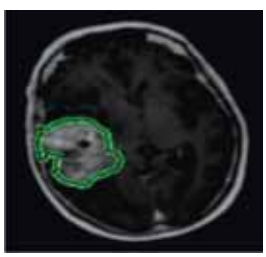


Fig. 7: Tumor Region Detected Image

The image in fig 5(a) represents original image when rotated and fig 5(b) represents the resultant image when source and target image are mapped. The Fig. 6 represents the final registered image and the Fig. 7 represents the area of tumor detected in brain.

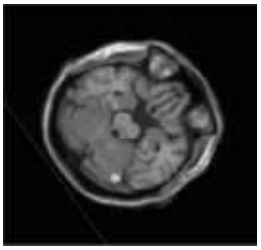


Fig. 8a: T1 Weighted Image

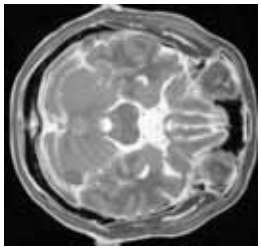


Fig. 8b: T2 Weighted Image

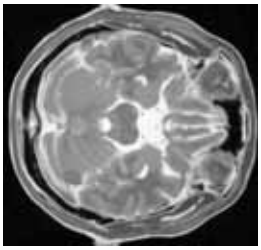


Fig. 8c: PD Image

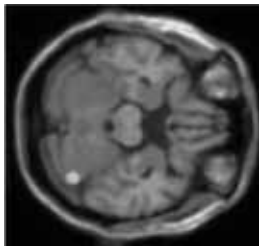


Fig. 9a: Registered Image

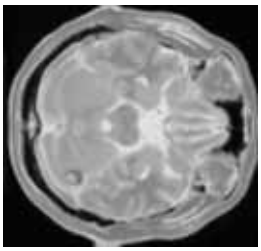


Fig. 9b: Fused Image

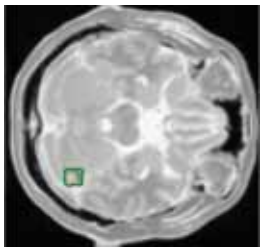


Fig. 9c: Segmented Image

Fig. 8(a), Fig. 8(b) and Fig. 8(c) are the T1 weighted, T2 weighted and PD MRI images respectively, here T1 image is registered using proposed algorithm, the registered image is as shown in Fig. 9(a), Fig. 9(b) shows fused information of T1 weighted, T2 weighted and PD image respectively, here image is fused in spatial domain by using average fusion rule. Similarly Fig. 9(c) shows the segmented image using region growing algorithm. In our approach T2 weighted image is taken as reference image, T1 weighted image is mapped with T2 weighted image for registration. The table1 shows the comparison between the existing and proposed algorithm and methodology.

Table 1 : Existing and proposed algorithm presentation

	Algorithms	Presentation
Existing	Graph based image registration and segmentation [13]	Prior knowledge is necessary
Existing	Combination of k-means and fuzzy C-means [11] , [12]	Better accuracy, but uses two segmentation algorithm, so computation time is more
Proposed	Enhanced Contourlet Edge based Registration + Region Growing	Detail Segmentation, low computation time

Conclusion

The target image is different in angle and size when compared to the source image. It is registered to the standard image using pyramidal directional filter bank which is combination of LP and DFB of contourlet transform. During image registration, Mutual Information as similarity measure is evaluated. Mutual Information technique and the contourlet-based edge extraction method are proposed for the multimodal image registration. MI technique has become a important and standard reference, mainly in medical imaging. It is considered with other like feature methods to get more gain with higher reliability, accuracy and robustness. Here it is combined with the contourlet transform. It is observed that as it can extract fine, accurate and continuous edges from the registered images, it can be effectively used in the feature extraction step of registration applications. Region growing algorithm is used to detect the tumor boundary after fusing the registered information. To increase accuracy, features of image can be extracted followed by Mutual Information as similarity measure.

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CASE REPORT

Erdheim - Chester Disease : A Rare Case Report

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ABSTRACT Erdheim-Chester Disease (ECD) is an extremely rare disorder that can affect many different organs of the body. It is rare form of non-Langerhans-cell histiocytosis. characterized by excessive production and accumulation of specific cells (histiocytes) whose normal function is to fight infections. The histiocytes accumulate in the loose connective tissue of many organs of the body and as a result this tissue becomes thickened, dense and fibrotic. Without successful treatment the disease is debilitating and can result in organ failure. ECD is often described in the medical literature as an extremely rare[1]. ECD usually presents in adults aged 40-60.In our case report the age of the patient is 26 years. Here we present a case of ECD in a 26 years male patient with progressive course over a period of 4 years developed features suggestive multiorgan involvement(lungs,ear,eye,brain,muscle, skeletal system)

Key words: Erdheim Chester disease; Interferon alpha; Interleukin-1; BRAF

Abbreviations: **ECD** - Erdheim-Chester Disease

Introduction

Erdheim–Chester disease (also known as Erdheim–Chester syndrome or polyostotic sclerosing histiocytosis) is a rare disease characterized by the abnormal multiplication of a specific type of white blood cells called histiocytes, or tissue macrophages (technically, this disease is termed a non-Langerhans-cell histiocytosis)[1]. Usually, onset is in middle age. The disease involves an infiltration of lipid - laden macrophages, multinucleated giant cells, an inflammatory infiltrate of lymphocytes and histiocytes in the bone marrow, and a generalized sclerosis of the long bones. it was first described in the literature in 1930 by the Austrian pathologist, Jakob Erdheim, and the American pathologist, William Chester [2]. Because it is so rare and because it is not discussed in the common textbooks of medicine, many doctors have never heard of it. It is usually difficult to diagnose. For these reasons, some feel the disease could be under-diagnosed and may not be as rare as thought.

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Case Report

26 years old male, presented with complaints of difficulty in breathing noticed increasing dyspnea on exertion and orthopnea, which has progressively worsened over the past 3 years. He also experienced drooping of eyelid right side with decreased vision since 1 month duration. The drooping of eyelid was insidious in onset, progressive in nature without, diurnal variation. Similar illness – drooping of Right eyelid with double vision 2 years back. There is a history of hearing loss (Rt>Lt), ear pain, ear discharge, headache, bone and joint pain. Giddiness and intermittent tinnitus, difficulty in speech, on and off since 3 years. Patient was on steroid treatment from which he got relief from symptoms for 2 to 3 month. There is no history of fever, vomiting and seizure. On examination pt is oriented and conscious, alert, afebile, Extra ocular movement - Rt eye restricted in all the direction except abduction, Lt eye-normal in movement in all the direction. Fundus – bilateral papillo-edema present. Pupils – Rt 4mm fixed, lt 2mm reactive. Vision acuity - Rt 6/12,lt 6/12.Bilateral conductive deafness present. Motor and Sensor system-normal. CVS, RESPIRATORY, PER-ABDOMEN ON clinical examination –Normal. Investigation-All blood investigations were in normal range. LUMBAR PUNCTURE DONE CSF ANALYSIS SHOWS-GLUCOSE-68, PROTEIN-48, CHLORIDE-126,CELL COUNT-14CELL (All are lymphocyte). PFT SHOWS RESTRICTIVE LUNG DISEASE.CHEST X-ray PA

view- Diffuse bilateral inflates present. CSF-Antimyobacterial and anticysternal antibodies negative by ELISA, CSF VDRL - non - reactive, serum RA-negative, serum ANA-negative, Bone marrow Histology - There was complete replacement of the fatty marrow by a variable degree of fibrous tissue and prominent proliferation of foamy histiocytes, including multinucleated forms. AUDIOMETRY - rt profound hearing loss, bilateral middle ear pathology, left moderate conductive hearing loss. CT SCAN BRAIN - There is evidence of T2 hypo intense thick nodular lesion seen along the dorsal aspect of cliws reaching down to sinus and orbital apex, lesion is isotense on TIWI and shows intense homogenous enhancement following in contrast.

Figure 1: Chest –X-ray PA VIEW. Showing Diffuse bilateral infiltrate.

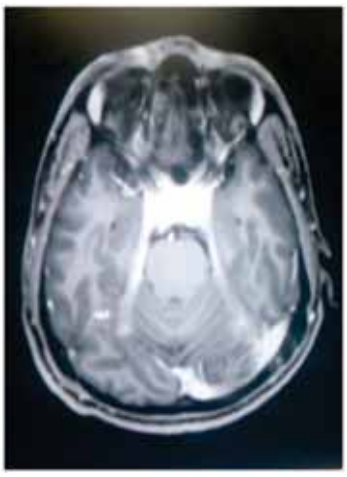

Figure 2: MRI done in year 2010 Imaging feature are suggestive of Pachymeningitis involving sellar parasellar orbital apex and clivus.

Figure 3: *MRI done in year 2014 Imaging shows Nodular Thickening and Enhancement of the skull base Pachymeninges.*

Discussion

Erdheim-Chester disease (ECD), a non-Langerhans form of histiocytosis, is a multisystemic disease characterized by various manifestations such as skeletal involvement with bone pain, exophthalmoses, diabetes insipidus, renal impairment and central nervous system (CNS) and/or cardiovascular involvement [1,4,10]. Prevalence is unknown. More than 500 cases (<15 pediatric) have been reported since 1930. ECD usually presents in adults aged 40-60 with a 3:1 male to female ratio[4]. Clinical course varies from asymptomatic to multisystemic, life-threatening forms. The pathognomonic feature of ECD is osteosclerosis of the long bones manifesting as bone pain, mainly affecting the distal lower limbs (50% of cases). Pituitary gland infiltration leads to diabetes insipidus and rarely hyperprolactinemia and gonadotropin insufficiency [11]. Constitutional symptoms include fever, weakness and weight loss [5]. Infiltrations in other organs can lead to intracranial hypertension, exophthalmos, papilledema, adrenal insufficiency, xanthelasmas and papulonodular skin lesions. CNS involvement can cause cerebellar and pyramidal syndromes, headaches, seizures, cognitive impairment, cranial nerve palsies and sensory disturbances [6,10]. A frequent cardiovascular involvement is the "coated aorta". Renal arteries can also be involved, leading to reno-vascular hypertension. Pericardial involvement may be complicated by a tamponade. Pseudo-tumoral infiltration of the right atrium is also seen. Dyspnea, due to lung infiltration, has been reported. Pseudo retroperitoneal fibrosis is sometimes complicated by bilateral hydronephrosis. Etiology is unknown but it is thought to be either a reactive or neoplastic disorder. Elevated levels of interferon-alpha (IFN-alpha), interleukin (IL)-7, IL-12, monocyte chemo attractant protein-1 and decreased levels of IL-4 found in

ECD patients support an associated systemic immune Th-1 oriented perturbation[9,12]. Recent findings of mutations in the BRAF proto-oncogene in > 50% of ECD cases clearly add further complexity to the pathophysiology of ECD [7].

The hallmark histological finding is the xanthogranulomatous or xanthomatous infiltration of tissues with spumous histiocytes. Immunohistochemical staining of a biopsy sample is CD68-positive and CD1a-negative[3 5].

Bone x-rays usually display bilateral and symmetric cortical osteosclerosis of the long bones, while technetium 99m bone scintigraphy shows almost constantly evidence of symmetric and abnormally strong labelling of the distal ends of the long bones of the lower limbs (and sometimes the upper limbs)[8]. Abdominal CT scan may show a "hairy kidney'' appearance (in 50%) which can be biopsied. Differential diagnosis includes Langerhans' cell histiocytosis, Rosai-Dorfman disease, Takayasu arteritis, Wegener's granulomatosis, primary hypophysitis, chronic recurrent multifocal osteomyelitis (see these terms), malignancies, neurosarcoidosis, mycobacterial infections and metabolic disorders.

First line treatment is the administration of standard or pegylated IFN-alpha for all forms of ECD with higher doses (9 million units, 3 times per week) required on a long-term basis for those with CNS and cardiac localizations (if well tolerated) [13]. Bisphosphonates may be given to alleviate bone pain. Cladribine can be given to those with orbital involvement that have been resistant to other forms of treatment. Anakinra can improve symptoms of mild forms of ECD in patients where IFN-alpha was ineffective. Recently, infliximab and vemurafenib have been used with some success, this latter drug seeming very promising for patients with a BRAFV600 mutation. PET scans are recommended for the assessment of disease activity.ECD has a variable prognosis but is overall poorer in those with CNS involvement. Before IFN-alpha, the mean survival after diagnosis was 19.2 months. Nowadays, with IFN-alpha treatments, the mortality rate is only 26%, and 5-year survival is 68%.

Conflicts of interest:	Nil
Ethics:	Patient confidentiality and safety were protected
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Guarantor:	Dr. Mamatha B. Patil will act as guarantor on behalf of all co-authors

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CASE REPORT

Capillary Hemangioma in the External Auditory Canal

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ABSTRACT Hemangiomas are common tumours encountered in infancy. Majority of the hemangiomas present in the head and neck region but rarely reported in the ear. We report a case of capillary hemangioma manifesting as an external auditory canal mass.

Key words: Hemangiomas, Capillary

Introduction

Hemangiomas are benign vascular lesions that are most commonly encountered in infancy and childhood. They are relatively common in the head and neck region but rarely reported in the ear. 40% of the Haemangiomas of the external ear arise from the lamina propria of the tympanic membrane and is pedicled in the posterior part. In 40% of cases, hemangiomas involve both the external auditory canal and tympanic membrane. In 20% of cases it usually involves only the skin of the posterosuperior wall of external auditory canal.

Case Report

A 60 year old male presented with a 2month history of blocking sensation and diminished hearing in the right ear and 3days history of right ear pain.On examination a pink pedunculated mass was visualized completely occluding the external auditory canal which was soft, bled on touch, could be probed all around the mass except in the posterosuperior aspect. Tuning fork test revealed conductive hearing loss on the right side. Pure tone audiogram revealed 30 dB conductive hearing loss on right side. CT temporal bone(plain) revealed a homogenous soft tissue mass in the external auditory canal without any bony erosion and an intact tympanic membrane and ossicles.

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Figure 1: CT temporal bone (plain)

Operative procedure

The mass was found to be arising from the skin of external auditory canal in the posterosuperior aspect and was completely excised with clear surgical margins via the transcanal approach and was sent for histopathological examination.Histpoathology revealed the tumour to be composed of dilated vascular channels and endothelial cells arranged in clusters and nodules with areas of fibrinoid necrosis which are characteristic of **Capillary Haemangioma.**

Histopathology

Figure2 : (10Xmagnification) Tumour shows squamous epithelium lining and composed of dilated blood vessels and endothelial cells with slit like lumen. Endothelial cells are arranged in clusters and nodules

DISCUSSION

Freedman (in 1972) was the first to report the presence of hemangioma in the external auditory canal,he

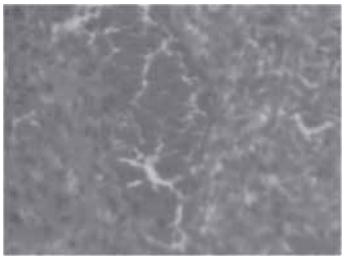

Figure 3 : (40X magnification) :shows dilated vascular channels lined by endothelial cells

described two male patients in their 60´s with lesion that started from the posterior wall of the external auditory canal affecting the tympanic membrane , since then very few cases have been reported. In 1987, Hawnk and van Nostrand described the first case of hemangioma of external auditory canal with no involvement of the tympanic membrane, and up to now 3 such cases have been reported.[3]

Haemangiomas are the most common tumours of infancyand childhood, with more than 60% of all haemangiomas occurring in the head and neck region. Complete resolution occurs in 50% of cases by 5 years of age; 70% by 7 years of age, with the remaining 30% resolving to varying degrees over time[1].

Hemangiomas are characterized by increased numbers of normal or abnormal blood vessels[9]. The natural history is characterised by rapid proliferation followed by a slow period of spontaneous involution. Growth during the **proliferative phase** is embodied by endothelial hyperplasia and an increase in the number of mast cells . During the **involution phase**, endothelial cell activity decreases with normalisation of mast cell count and inltration of the cellular parenchyma by brofatty tissue[1].There may be difficulty in histologically distinguishing it from vascular tumours.Malignant transformation is rare.There are several histologic and clinical variants.The most common variant, capillary hemangiomas,occur in the skin,subcutaneous tissue and mucous membranes of oral cavities and lips as well as in the liver,spleen and kidneys[9].

But the hemangioma of the tympanic membrane and/or the external auditory canal occur predominantly in adulthood (average age 56), with a predilection for the male sex (M:F, 2:1). 40% of external auditory hemangiomas, arise from the lamina propria of the tympanic membrane and is pedicled in the posterior part of the tympanic membrane .The hemangioma may aect the external auditory canal involving only the skin of the posterosuperior wall (20% of cases). It can sometimes involve both the external auditory canal and tympanic membrane(40% of cases)[7].

Temporal bone hemangiomas tend to arise from the geniculate ganglion,internal auditory canal and beginning of chordatympani due to their vascularity[5]. Typically, the ear hemangiomas do not have bone invasion properties [7].Treatment of these lesions is by complete excision with clear surgical margins. Different approaches can be considered according to the location of the lesion. Small lesions can be removed by transcanal approach, whereas transmastoid, translabyrinthine, middle fossa approach or combined methods must be considered for advanced lesions[7].

CONCLUSION

This is a rare case presentation of capillary hemangioma occuring in the external auditory canal. Hemangiomas should be differentiated from other vascular lesions like glomus jugulare, high jugular bulb, aberrant internal carotid artery and other arteriovenous malformations. Extent of the lesion should be evaluated radiologically and managed by clear surgical excision and confirmed by histopathological examination.

Conflicts of interest:	Nil
Ethics:	Patient confidentiality and safety were protected
Funding:	Self funded
Guarantor:	Dr. Vellavedu Umapathy Shanmugam will act as guarantor on behalf of all co-authors

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CASE REPORT

Uncommon Nasopharyngeal Mass - Extranodal Natural Killer/T-cell lymphoma: A Case Report

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ABSTRACT Extranodal natural killer/T cell lymphoma, nasal type, is a non-Hodgkin lymphoma, most commonly affecting the nasal cavity, paranasal sinuses and nasopharynx. Clinically it is characterised by destruction of fascial tissues,commencing in the midline, in most cases it arises as a malignant transformation of natural killer cells (NK); sometimes as malignant transformation of cytotoxic T cells. Extranodal NK/T cell lymphoma, nasopharyngeal type, is rare, more so in black persons. This case of extranodal NK/T cell lymphoma, nasopharyngeal type, manifested in elderly female.

Key words: Natural Killer cells, Cytotoxic T cells, T cell lymphoma

Abbreviations: **NK -** Natural Killer cells; **EBV** - Epstein - Barr virus

Introduction

Extranodal natural killer/T cell (NK/T cell) lymphoma, nasopharyngeal type, is a rare non-Hodgkin lymphoma originating in nasopharynx. It is strongly associated with Epstein-Barr virus (EBV) infection.Its prevalence is higher in countries in South-East Asia and in Central and South America than in Europe and in North America; it occurs in middle-aged persons and affects males more frequently than females [1-5].

Extensive local invasion, regional lymph node involvement, elevated serum lactate dehydrogenase, raised EBV DNA titres and systemic signs (fever, night sweats, weight loss) are associated with a poor prognosis [3,4,6] and the overall prognosis is poor. The five year survival rate is reportedly between 38% and 85%.

Case Report

A 50 yrs old female presented to our OPD with C/O bleeding from left nostril and headache for 2 yrs,which was insidious in onset and intermittent for 2 years. Bleeding is of moderate quantity and it occured spontaneously and stopped on its own.

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Post nasal examination showed a nodular mass arising over right lateral nasopharynx. Blood parameters were within normal limits. Diagnostic nasal endoscopy showed red nodular mass in the nasopharynx more towards right lateral nasopharynx (fig 1).

Fig. 1

Fig. 2: CD_{20} (B Cell Maker)

Fig. 3: CD_3 (T Cell Maker)

DISCUSSION

Most cases arise from natural killer cells, only a few arise from cytotoxic T-cells. Clinically, extranodal NK/T cell lymphoma, nasal type, is characterized by progressive midline facial destruction, initial signs and symptoms include nasal stuffiness, epistaxis and pain, owing to the progressive tumour growth in the nose.As the tumour mass enlarges, it invades and destroys structures in the upper airway and becomes progressively necrotic with a purulent discharge. Signs and symptoms are related to the sites involved. Secondary infection and haemorrhage are not infrequent. Metastasis is uncommon [7].

As extranodal NK/T cell lymphoma, nasal type, may clinically mimic other destructive disease entities affecting mid-facial structures including other Lymphomas, Nasopharyngeal Squamous Cell Carcinoma, Tertiary Syphilis, Wegener granulomatosis and Fungal infections, the definitive diagnosis must be based on histopathological, immunological and molecular studies [5]. Localised extranodal NK/T cell lymphoma, nasal type usually responds favourably to radiotherapy.

Extranodal NK/T cell lymphoma, nasal type, is characterized histopathologically by angiocentric and angiodestructive growth, by tumour cells that vary in size and may harbour EBV in a clonal episomal form, and by inflammatory cell infiltrate of plasma cells, histiocytes and eosinophils. Immunophenotypically, the malignant tumour cells,like the natural killer cells from which they originate express CD2, cytoplasmic CD3 and CD56. As NK cells and T cells may arise from common progenitor cells, NK cells may express some T cell antigens, and T cells may express some NK cell antigens, so that cells of extranodal NK/T cell lymphoma, nasal type, may express both NK cell and T cell antigens.

CD-3 is a T-cell antigen and it is used to identify T-cells. It stains both membrane and cytoplasm. In immature cells they are cytoplasmic positive and as the cell matures, they move to the surface and becomes surface positive. CD-3 has been expressed, (both membranous and cytoplasm) in B- cell lymphomas with expression of EBV[8]. CD-20 is a marker for B-cell and used to identify B-cell lineage. CD-20 is positive in a small subset of non- neoplastic T-cell and very rarely in T-cell lymphomas [9]. The more reliable criteria for NK/T cell malignancy is the presence of significant cytological atypia in a diffuse population of T-cells and aberrant loss of BCL-2 expression. The present case shows CD-3 surface antigen negativity and shows CD-20 positive with cytological atypia.

Staging: The extent of invasion by the tumour is classified by stages:

T1 refers to a tumour confined to the nose & nasopharynx;

T2 to additional tumour invasion of the maxillary and anterior ethmoidal sinuses and/or the hard palate

T3 to further tumour invasion involving the posterior ethmoidal sinuses, sphenoidal sinuses, orbit, maxillary alveolar process of bone, and buccal tissues; and T4 to tumour invasion extending to the mandibular alveolar process bone, to the infratemporal fossa, and to the cranial fossa.

Our patient is in T1 stage and since it is amenable to curative radiotherapy, our patient was referred to cancer institute for further management.

EBV infects the NK/T cells and establishes latent infection before the initial transformation of NK/T cells has occurred, prior to the clonal divergence and clonal expansion of the cancerous cells. The presence of this clonotypic EBV genome in a latent form in the tumour cells strongly supports, but does not prove the pathogenic role of EBV in NK/T lymphomagenesis. The active involvement of EBV in the pathogenesis of extranodal NK/T cell lymphoma, nasal type, is further supported by the direct positive correlation between EBV load in the tumour and the extent of the disease,and by the high titres of IgG antibodies to EBV in persons with the disease. Plasma titre of EBV DNA serves as a marker of tumour viral burden and fluctuates with the status of the disease and the response to treatment because EBV DNA fragments are released from apoptotic tumour cells and escape into the circulation . In our hospital we do not have facilities for in-situ hybridization studies, hence we do not know whether the cells of the extranodal NK/T cell lymphoma, nasal type, of our patient carried EBV encoded early RNA.

We presented this case because NK /T CELL LYMPHOMA is rare in females and that too in the nasopharynx.

Conflicts of interest:	Nil
Ethics:	Patient confidentiality and safety were protected
Funding:	Self funded
Guarantor:	Dr. Munirathinam Kumar will act as guarantor on behalf of all co-authors

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CASE REPORT

Colloid cyst in Third Ventricle

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ABSTRACT Colloid cyst are relatively rare benign intracranial tumor located in the front part of third ventricle at foramen of monro. Clinical presentation may be non-specific and heterogeneous may produce hydrocephalus , brain herniation and may lead to sudden death. We describe case of Road traffic accident in which colloid cyst was diagnosed accidentally with no associated hydrocephalus.

Key words: Colloid cyst, Hydrocephalus, third ventricle

Introduction

Colloid cyst are rare congenital and benign intracranial tumors, representing upto 2% of all intracranial neoplasm[1-3]. Location is usually in the anterior and anteriosuperior part of the third ventricle[4,5] occuring in 3rd to 5th decade of life. The cyst may cause obstruction of the foramen of monro leading to hydrocephalus and may lead to sudden death. The symptoms may be non-specific on related to rate of hydrocephalus[4,6]. Collid cyst may be a complete accidental finding as well, when the patient performs imaging of the head due to the complaints that are unrelated to the cyst. The incidence of colloid cyst is increasing owing to extensive use of modern diagnostic methods such as CT and MRI of head[4]

Case Report

A 22 year old male patient underwent Road traffic accident CT Brain was done which revealed multiple fracture involving superior wall, lateral wall of left maxillary sinus, lateral wall of left orbit and nasal bone with left maxillary and left sphenoidal hemosinus with well defined hyperdense lesion measuring 6.5x4.9mm taking density of 66 HU to 75 HU just below the septum pellucidum at foramen of monro suggestive of ? Colloid cyst ?? Hematoma repeat CT was adviced. on repeat scan colloid cyst was confirmed. Patient complaint of slight headache.

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Under general anaesthesia in supine position, endoscopic excision of the cyst was done. The interior of the cyst is aspirated followed by removal of the cyst, wall and material and was sent for histopathological examination. Postoperative the patient was neurologically intact with no sign of raised intracranial pressure and was discharged a week late.

Fig. 1 : Axial CT Image

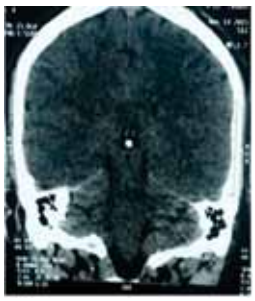

Fig. 2 : Coronal CT Image

Fig. 3 : Sagittal CT image

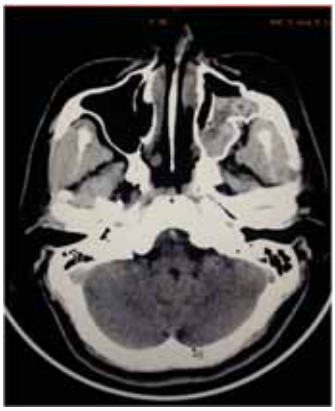

Fig. 4 : Axial CT Image

Discussion

Colloid cyst of the third ventricle are rare lesion comprising upto 2% of primary brain tumor[1-3]. Most commonly found posterior to the foramen of monro in the anterior aspect part of third ventricle[4,5]. Its location in the third ventricle help to distinguish the colloid cyst from other cyst such as enterogenous cyst, ependymal cyst and Rathke cleft cyst that superficially resemble it but occur in different location. Because of its location, it can cause obstructive hydrocephalus and increased intracranial pressure. Most reported cases occur in the 3rd to 5th decade of life[3]. Familial cases of colloid cyst are extremely rare. Patient can be asymptomatic .Symptoms include headache relieved by a change in position, vertigo, memory deficit, diplopia, behavior disturbances and in extreme cases sudden death. Cyst are 3-40mm in diameter. Spherical and smooth walled. Cyst size does not appear to be reliable predictor of outcome, as even small ones may result in sudden death[6]. Both CT and MRI imaging may be used in the diagnosis of colloid cyst. MRI features are variable and may show intracystic fluid levels may show isosignal intensity. On CT scan, most are well defined hyperdense. Calcifications are rare in colloid cyst. Early detection and total excision of the colloid cyst through an open craniotomy , or by endoscopy, as in our case carries an excellent prognosis. Acute hydrocephalus associated with the colloid cyst may require emergency drainage.

Conclusion

Early detection and prompt treatment with complete removal of he cyst led to improvement of symptoms and excellent prognosis.

Conflicts of interest: Nil

Ethics: Patient confidentiality and safety were protected

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CASE REPORT

The Eagle Syndrome : A Clinical Dilemma - Case Report and Review of literature

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ABSTRACT Elongated styloid process causing craniofacial symptoms is called as Eagle syndrome. We report a 45-year-old female with this syndrome. Clinically the elongated styloid process was mistaken for submandibular calculus. The diagnosis was made by computed tomography. Styloidectomy relieved her symptoms.

Keywords: Eagle syndrome
Abbreviations: USG - ultrasonography; CT - computerized tomography; ESP - Elongated styloid process

Introduction

Eagle syndrome is a clinical condition characterized by craniofacial and cervical symptoms caused by elongated styloid process (ESP) or calcification of the stylo-hyoid ligament. It was first described by Eagle in 1937. According to him elongated styloid process is present in approximately 4% of the population.[1,2] However, other investigators have reported an incidence rate varying from 1.4 to 18.2 %.[3,4] Most of the patients with ESP are asymptomatic and only a small percentage of them (4 to 10%) actually become symptomatic.[5] Eagle syndrome is diagnosed only if the patient is clinically symptomatic and not merely by the presence of ESP. Patients may present with wide range of symptoms such as ear ache, sore throat, vague foreign body sensation in pharynx, hyper-salivation, painful movement of the neck and tongue, odynophagia and headache. These symptoms are due to pressure effect of ESP on adjacent soft tissues. In the absence of appropriate imaging technique clinical diagnosis is often missed due to the vague nature of symptoms.

Case Report

A 45-year-old female presented with 3 months' history of pain on the right submandibular region. Neck movements were also painful. USG examination of the neck was unremarkable. Sialolithiasis of right submandibular gland was suspected clinically. Non-contrast spiral CT scan of the neck with three-dimensional reconstruction was done using 64-slice multidetector computed tomography scanner (Somatom Definition AS-Siemens). Coronal, sagittal images as well as thick multiplanar reconstruction image and volumetric rendering technique images were obtained. These images revealed dense ossification of the right styloid process and stylohyoid ligament. The grossly thickened styloid process was extending up to the hyoid bone (Fig. 1, 2). The right styloid process was measuring 7.4 cm whereas its left counterpart was normal in length (2.6 cm). The maximum thickness of the right styloid process was 1.2 cm. Bilateral submandibular salivary glands and other soft tissue structures of neck were normal. In addition there was presence of anterior bridging osteophytes and calcification of anterior longitudinal ligament of the cervical spine involving C3 to C7 cervical vertebrae. (Fig. 3) She was relieved of her symptoms after undergoing surgical removal of the right ESP.

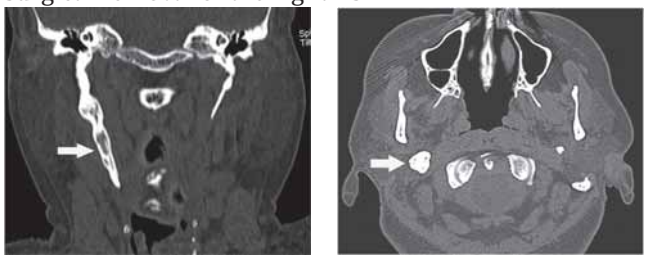

Fig 1: Plain computed tomography showing elongated and thickened right styloid process (arrows) in coronal (A) and axial (B) sections.

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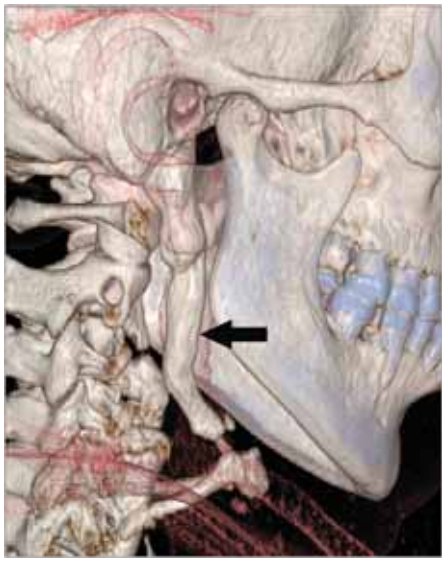

Fig 2: Volumetric rendered technique image showing elongated styloid process on the right side (arrow)

Fig 3: Mid-sagittal CT images of the same patient showing anterior bridging osteophytes and calcification of anterior longitudinal ligament of the cervical spine involving C3 to C7 vertebrae

Discussion

Styloid process is a conical bone which arises from the mastoid portion of temporal bone just anterior to stylomastoid foramen and then courses downwards, forwards, and medially. The stylohyoid ligament connects the tip of styloid process and the lesser cornue of hyoid bone. The tip of styloid process is often cartilaginous. Ossification of the cartilaginous tip or calcification of the stylohyoid ligament causes an appearance as if the styloid process is thickened and elongated. Length of the styloid process tends to increase with advancing age and therefore, ESP is more prevalent in the age group of 31 to 50 years. [6] Symptomatic patients are usually females above the age of 40 years. [7-9] However, few authors have reported a higher incidence of ESP in males. [6]

ESP exerts pressure effect on the adjacent soft tissue structures such as the internal jugular vein, carotid artery and cranial nerves V, VII, IX, X and XII. Pressure symptoms consequent to nerve compression constitutes the Eagle syndrome. In our patient the close proximity of the styloid tip to the submandibular gland was clinically mistaken for salivary calculus.

The styloid process normally measures 2.5 to 3 cm in length [2]; but it varies in length from person-to-person and even from side-to-side of the same person. Kaufman et al. have defined 30 mm is the upper limit for normal styloid processes. [3] Therefore, styloid elongation can be diagnosed if its length exceeds 3 cm. [2,3] Partial ossification of stylohyoid ligament is not uncommon, but complete ossification is quiet rare. [10] The actual cause of styloid process elongation is poorly understood and the various theories proposed are: 1) congenital elongation of the styloid process due to persistence of a cartilaginous analogue of the embryologic precursors of the styloid, 2) calcification of the stylohyoid ligament due to an unknown reason, and 3) growth of osseous tissue at the insertion of the stylohyoid ligament.[5]

Based on various radiological features, Langlais et al classified ESP into 3 types. Type-I is characterized by uninterrupted elongation of the bone; type-II is characterized by the styloid process apparently being joined to the stylohyoid ligament by a single pseudo articulation giving the appearance of an articulated elongated styloid process, and type III consisting of interrupted segments of the mineralized ligament, sometimes creating the appearance of multiple pseudoarticulations. [11] Our case belongs to type-I ESP with pseudoarticulation with hyoid bone.

Researchers have correlated the ligamentous ossification of the stylo-hyoid complex and osteophytes of the cervical spine, concluding that variable types of styloid process-stylohyoid ligament complex abnormalities have significant correlation with ligamentous ossification and osteophytes of the cervical spine including cervical diffuse idiopathic skeletal hyperostosis (DISH). Enlargement of this ligament were significantly correlated with transverse and antero-posterior dimensions of ligamentous ossification or osteophytes of the cervical spine at various levels.[12] This association was also present in our case.

There are two types of Eagles syndrome (not to be confused with the 3 types of ESP) as described originally by Eagle: (1) the classic styloid syndrome presenting as pharyngodynia localized to the tonsillar fossa due to a

post- tonsillectomy fibrous scar causing compression/ irritation of the cranial nerve endings against the ESPs; (2) stylo-carotid syndromes (Carotidynia and Ernst syndromes) are due to compression of the internal and/ or external carotid arteries and their perivascular sympathetic fibers, resulting in a persistent pain radiating to the carotid territory. [13,14] Such patient may complain of headache, chronic neck pain, pain upon turning the head, pain radiating to the eye, ear pain and vertigo. [13,14]

Eagle Syndrome can be suspected clinically on the basis of symptoms, diagnosed on physical examination and confirmed radiologically by conventional radiography or more sensitive CT scan. The elongated styloid process can be felt in the tonsillar fossa, and palpation may aggaravate symptoms. Several radiographic projections have been used for visualisation of ESP and they include lateral head-and-neck view, Towne view, panoramic view, lateral-oblique mandible view, and posterio-anterior head view. [15] Superimposed bony anatomical structures will make it difficult to visualize ESP in plain radiography. Reconstruction of three dimensional views using multidetector computed tomography is an extremely valuable imaging tool. Details of the length, angulation, and relation to adjacent structures can be obtained accurately from a 3D-CT scan. [15]

Differential diagnoses to be considered in patients with similar clinical picture include temporomandibular joint diseases, trigeminal, sphenopalatine or glossopharyngeal neuralgias, temporal arteritis, chronic pharyngotonsillitis, otitis media, external otitis, mastoiditis, dental pain, improperly fitting dental prostheses, submandibular sialadenitis or sialolithiasis, true pharyngeal foreign bodies, and tumors of the pharynx or tongue base. [14, 15, 16] Most of these causes can be diagnosed or ruled-out by CT.

Eagle syndrome can be managed by surgical and nonsurgical methods. Nonsurgical treatments involve reassurance to the patient, analgesics, and steroid injections. Surgical treatment option includes styloidectomy which can be performed either trans-pharyngeally or extra-orally. The latter is thought to be superior because of decreased risk of deep neck space infection and better visualization of the surgical field. [5,14,16]

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Ethics:	Patient identity is protected and the intervention done is part of routine clinical care
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CASE REPORT

Extensive Necrotizing Fasciaitis (NF) following Administration of Intra-Muscular Injection in a Acute febril illness with Thrombocytopenia due to Dengue fever.

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ABSTRACT We report the case of 22 years of young male patient, non-addict, without any long standing disease like diabetes, hypertension or peripheral vascular disease, presented with fever with chills for three days and taken Intra-muscular injection at Right buttock for fever and developed extensive necrotizing fasciitis (NF) with crepitus (sub-cutaneous emphysema) in Right legs, thigh, rt. Lateral abdominal wall and back. He was diagnosed to have acute febrile illness with thrombocytopenia due to acute dengue infection. He also developed septicemia, rhabdomyolysis and subsequently acute renal shutdown within short time. Extensive surgical debridement, antimicrobial therapy and sustain low efficiency dialysis (SLED) given. Despite rapid diagnosis and treatment, the patient died of septic shock with multiorgan failure.

Keywords: Necrotizing Fasciitis, Thrombocytopenia, septicemia

Abbreviations: **NF** - Necrotizing Fasciitis, **SLED** - Sustain Low Efficiency Dialysis

Introduction

Necrotizing fasciitis or NF, commonly known as flesh-eating disease, flesh-eating bacteria or flesh-eating bacteria syndrome [1]. Wilson used the term *necrotizing fasciitis* without assigning a specific pathologic bacterium that caused the disease [2]. It is an uncommon, devastating soft tissue infection primarily involving superficial fascia, subcutaneous fat and deep fascia that relatively spares skin and underlying muscle. NF a term introduced by Wilson, was first described as "hospital gangrene" in the American Civil War era [3]. It most frequently occurs in the abdominal wall, extremities and perineum, where the pathogen may be introduced in the subcutaneous space via disruptions of overlying skin. Besides direct inoculation, haematogenous spread from a distant site may probably occur. The disease predominantly develops in diabetics, alcoholics, immunosuppressed patients, illicit drug users and patients with peripheral atherosclerotic vascular disease. Despite rapid diagnosis and treatment, case fatality rate is high and any delay may correlate with worse outcome [1]. The morbidity and mortality of necrotizing fasciitis is 20%-60% [4]. Lethal outcomes in patients with NF who undergo therapy with NSAIDs are reported [5]. NSAIDs enhance the production of TNF-α in the presence of endotoxins by impairing the normal feedback loop during the production of TNF and other cytokines, and, therefore, acting as an additional stimulus to the proinflammatory cascade. In addition, they are potent inhibitors of neutrophil granulocyte chemotaxis and phagocytosis by inhibiting the lipoxygenase pathway, which is specially true for diclofenac and indomethacin. In the same way, they also decrease leukotriene production by leukocytes, which are compounds known to play a role in the inflammatory response. Finally, they confound the progression of disease by suppressing fever and pain through inhibition of prostaglandin synthesis, thus blurring the signs of onset of serious infection.

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Case Report

A 22 years of male patient presented with history of fever with chills since 3 days, right leg swelling and unbearable pain since 2 days and drowsiness since 1 day. Patient was apparently alright 3 days ago when he developed fever with chills. For which he went to his family doctor who

gave some tablets and Intra-muscular injection in right buttock. Next day, fever continued but in addition he developed pain in right buttock (at the site of injection) and swelling, which increased rapidly and involved right lower limb along with unbearable pain. There was no relief, for which he went back to same family doctor who gave another intramuscular injection in right deltoid. Day third, patient develops swelling and tightness of entire right lower limb massively. He got admitted to a nursing home with drowsiness from where he was transferred to our hospital for further management. In casualty, he was in shock with poor general condition. BP was not recordable, pulse was 146/min, tachypnoea, SPO2 90%, RBS 95mg/dl with icterus. After giving IV fluid of 2 litres, patient was transferred to ICU. In ICU, fluid challenge was given followed by 250ml/hr NS started and patient was stabilized with inotropic (nor-adrenaline) support. He was given meropenem and clindamycin intravenous as broad sprectum antibiotics. His pulse rate came down to 116/min, Blood pressure rose to 110/70. hemodynamic monitoring started. There was hemorrhagic as well as echymotic patches at the site of injection as well as on right thigh (as shown in picture). Right thigh and leg was extreme painful. There was extensive crepitus over right lateral wall of abdomen, right thigh and leg along with sole. Necrotizing fasciitis was diagnosed . Patient passed only 10 ml of hemorrhagic urine since admission . Surgical reference was made for urgent surgery with high risk consent. Patient was given 1 packed cells, 4 FFP and 6 units of random donor platelets transfusion given prior to surgery. Within 2 hours after hemodynamic stabilization, surgery was performed. Multiple incisions were made through out right lower limb. There was extensive necrotizing materials, pus and lots of gases evacuated. Muscle biopsy and necrotic materials send for histopathological investigation. Smear from pus revealed no Gram positive spore bearing organisms (Gas gangrene). Pus Culture did not grow any organism.

Results

On admission : Haemoglobin - 9.7 PCV-24.4,WBC-18800,Platelets-58000, IGM LEPTOSPIRA- 2.4 NEGATIVE(N=<9 UNIT),DENGUE IgM positive, NS1 antigen and IgG dengue negative, HbsAg non reactive, HIV non reactive, anti HCV- non reactive, SGPT-95, Alkaline phosphate-37, Total Protein 10.6 (hemoconcentrated), Albumin 5.9, Globulin 4.7,SGOT 1060,GGTP 800, INR-2.81, PT-28.3s, INR 2.81, aPTT-50.5s (N : 23-33), CPK-12965 (normal: 55-175 IU/L), BT 3'55'', CT 9'58'' , CLOT RETRACTION 56% (N: 48-64%), FDP 18.2mg/l (N:0.05 -0.3 mg/l), PLASMA FIBRINOGEN 293 (N 250-450 mg/dl), ABG : PH 6.9, PCO2 22, HCO3 6.5 SEVERE METABOLIC ACIDOSIS, Urine routine examination : Protein 4+, epithelial cells 2-3, Leucocytes-6-7, Erythrocytes >200/hpf, Bacteria Present Few granular cast present. Post operation : Haemoglobin-2.4,PCV -6.5, WBC-11900, Platelet-52000,

USG Lower Limb- Extensive diffuse subcutaneous oedema extending into intramuscular planes with air bubbles noted in all aspect of right thigh & lower limb. Fascial layer thickening is noted. Above finding suggestive of necrotizing fasciitis. **X-RAY Chest Supine AP -** No significant abnormality seen except there is pneumomediastinum seen outlining heart and aorta. Air also seen in neck and outlining diaphragm. **Tissue Specimen**-Histopath of tissue biopsy revealed haemorrhagic and necrotic muscle tissue with infiltration of neutrophils seen suggestive of necrotic muscle.

Post-operatively, patient was transfer to ICU and Sustained low efficiency dialysis (SLED) started in view of anuria along with packed cell transfusion. Patient was on ventilator with Inotrope support. Patient developed sudden onset bradyarrythmia and arrested. However patient could not be revived and patient died within 24 hours of admission.

Figure 1 : Necrotizing fasciitis due to IM injection in a 22-years of male patient. Note Site of injection (Right buttock) minimal erythema, swelling and echymotic patches

Figure 2 :Necrotizing fasciitis due to IM injection in a 22–years of male patient. Note minimal erythema , swelling and echymotic patches over right thigh and involving upper part of lower leg.

Figure 3 : Necrotizing fasciitis due to IM injection in a 22-years of male patient. Note minimal erythema, swelling and echymotic patches over right thigh and involving upper part of lower leg.

Discussion

This patient was presented with Acute febrile illness with thrombocytopenia due to dengue fever. Patient received Intra-muscular injection for fever. Patient deteriorated rapidly and developed necrotizing fasciitis involving lower limb with crepitus formation all over right limb and retro-peritoneal spread of air into mediastinum. Patient also developed rhabdomyolysis (as evidence by CPK- 12965 (normal: 55-175 IU/L) leading to Acute Renal shutdown. Despite early diagnosis and immediate intervension , our patient could not be survived. It most frequently occurs in the abdominal wall, extremities and perineum, where the pathogen may be introduced in the subcutaneous space via disruptions of overlying skin. Besides direct inoculation (intra-muscular injection) haematogenous spread from a distant site may probably occur. Pain out of proportion to physical findings in a patient with evidence of a systemically toxic condition should raise the clinical suspicion of necrotizing fasciitis. Overt sign of necrotic tissue, such as crepitus occurs only in 30% of the patients. Radiographic imaging like Ultrasonography, CT scan is helpful to diagnose necrotizing fasciitis. Treatment modalities comprise early aggressive surgery with frequent wound debridement, broad spectrum antibiotic coverage, hyperbaric oxygen, and supportive care [6].

Conclusion

Whenever there is epidemic of acute febrile illness with thrombocytopenia, intra-muscular injection is to be avoided . If there is pain at the site of injection, careful observation should be made and if require proper reference should be given. Recent studies [7] have highlighted the importance of correct IM drug administration, in order to minimize the risk of potentially serious complications. Appropriate clinical practice needs to reflect considerations about appropriate needle length and gauge, to ensure that patients get the benefit of drug administration without adverse effects. Muscle tissue is usually spared the harmful effects of substances injected into it, probably because of its abundant blood supply. However, deep IM injections can cause abscesses and granulomas, whereas more superficial IM injections may result in increased incidence of local reactions, such as irritation, inflammation and necrosis. The clinical presentation may be masked, as changes in the overlying skin may only be observed later in the disease process. Clinical differentiation between necrotizing fasciitis and non-necrotizing fasciitis is more important as NF require immediate treatment. Current guidelines continue to emphasize timely diagnosis, effective surgical debridement and appropriate initial antibiotics coverage as proven way to optimize the patients outcome.

Conflict of interest:	All authors declare no COI
Ethics:	Patient identity is protected and the intervention done is part of routine clinical care
Funding:	No external funding
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HEALTH

Improve Children Health – Best Investment for Better Tomorrow

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ABSTRACT India is home to the largest number of children in the world. In recent years there has been an increased focus on issues that affect children and on improving their health. Healthy children are more ready and able to learn and, in the longer term, are more likely to become healthy adults who will contribute as a productive citizenry and workforce to the continued vitality of society. The major health problems in children are varied from infections, malnutrition, mental problems to sexual abuse and gender violence. The needs and care of children are very different at different ages. Despite many improvements, India lags far behind other industrialized nations in this and other measures of children's health. Today for measuring child health we use various indices but it is well known that there are no comprehensive, agreed-upon measures or indices as to what constitutes child health. The strategies for child health intervention focus on improving skills of the health care workers, strengthening the health care infrastructure and involvement of the community through behavior change communication to implement policies and programme more efficiently and achieve objective on time. It is seen that despite major gains over the past century, children's health varies widely across population subgroups and lags well behind that of many other industrialized nations. Unless we act now, our children will continue to fall farther behind, putting our society, economic prosperity, and national security at even greater risk.

Keywords: Child Health Issues, Strategies for Intervention, Surveillance

Abbreviations: **WHO -** World Health Organisation; **NDC** - National Policy for Children

Introduction

India is home to the largest number of children in the world, significantly larger than the number in China. In India, childhood has been defined in the context of legal and constitutional provisioning, mainly for aberrations of childhood. It is thus a variable concept to suit the purpose and rationale of childhood in differing circumstances. Essentially they differ in defining the upper age limit of childhood.[1]

Biologically, childhood is the span of life from birth to adolescence. According to Article 1 of UNCRC1 (United Nation's Convention on the Rights of the Child), "A child means every human being below the age of 18 years unless, under the law applicable to the child, majority is attained earlier" [2]. The definition of the child as given by the UNCRC has definite bearing not only on child development programmes and on budgetary provisions for them, but also on production of statistics as applicable to different cross sections of children in terms of reference ages. The age-groups of 0-14 years, 15-59 years and 60 and above years form a commonly accepted standard in reporting demographic indicators.

Children are vital to the nation's present as well as its future. In recent years; there has been an increased focus on issues that affect children and on improving their health. It is in the national interest to have healthy children. Healthy children are more ready and able to learn and, in the longer term, are more likely to become healthy adults who will contribute as a productive citizenry and workforce to the continued vitality of society[3]. Children have begun to be recognized not only for who they are today but for their future roles in creating families, powering the workforce, and effectively contributing in development of the country. Mounting evidence that health during childhood sets the stage for adult health not only reinforces this perspective, but also creates an important ethical, social, and economic imperative to

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ensure that all children are as healthy as they can be [3]. Healthy children are more likely to become healthy adults. Health during childhood must be a major concern both because children are important in their own right and because the nation cannot thrive if it has large numbers of unhealthy adults.

Within this context, it is reasonable to ask what it means for children to be healthy and whether the country is adequately supervising and appraise the health of its children. Do current surveillance and monitoring approaches provide the sufficient information necessary to evaluate ongoing programmes and policies to ensure that common precedence and available resources are aligned with children's needs and deployed to optimize their health? Are there ways possible to get methods for sculpture policies and practices in better manner specially designed to make children healthier?

Child Health issues: Changing Trends

When we take a look at the changing scenario in child health in India over the twentieth century, the improvements are nothing short of breathtaking. In 1910, the infant mortality rate was 145.6 per 1,000 live births; by 2014 (SRS), the rate had dropped to 39 deaths per 1,000 live births [4].

In India, an estimated 27 millions of children are born every year. As per Census 2011, the share of children (0-6 years) accounts 13% of the total population in the Country [5]. Majority of these births took place in the underprivileged rural and urban communities, where the parents are not always able to provide adequate care. Newborn and infant mortality rates are particularly high in such situations. An estimated 12.7 lakh children die every year before completing 5 years of age. However, 81% of under-five child morality takes place within one year of the birth which accounts nearly 10.5 lakh infant deaths whereas 57% of under-five deaths take place within first one month of life accounts 7.3 lakh neo-natal deaths every year in the Country [5].

The World Health Organization (WHO) estimates that India is one of the highest ranking countries in the world for the number of children suffering from malnutrition. The prevalence of underweight children in India is among the highest in the world, and is nearly double that of Sub Saharan Africa with dire consequences for mobility, mortality, productivity and economic growth [6].

As per NFHS 3, 48% of children under age five years are stunted (too short for their age) which indicates that, half of the country's children are chronically malnourished. Acute malnutrition, as evidenced by wasting, results in a child being too thin for his or her height. 19.8% of children under five years in the country are wasted which indicates that, one out of every five children in India is wasted. 43% of children under age five years are underweight for their age[7].

The major child health problems in different age groups have been summarized in **Figure 1.**

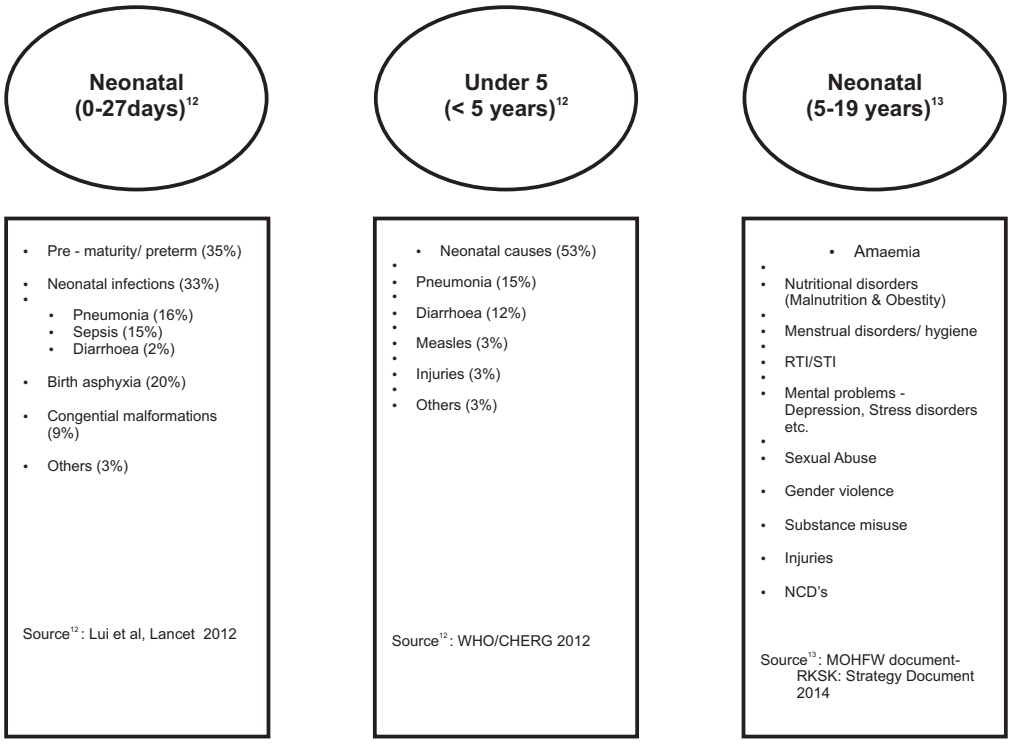

Figure 1: Major child health problems in India

Health Needs Of Children In Current Situations

The needs and care of children are very different at different ages. The important health needs at various ages can be considered as follows:

Newborn: Maternal nutrition and adequate antenatal care. Safe delivery, immediate care of the neonate and subsequent management during the first 1-3 months.

Infancy and pre-school period: Feeding and nutrition (supplements of iron, vitamins), immunization, proper management of common infections (diarrhea, respiratory, skin, eye, ear, parasitic), and attention to development.

Older children: Adequate nutrition, treatment of acute and chronic diseases (e.g. tuberculosis, malaria, water borne diseases).

Adolescents: Physical and emotional health, treatment of acute and chronic diseases, family life counseling.

Parents, grandparents, and other family members are usually committed to providing every possible facility to the children in their families, and to ensuring that they are always healthy and have the opportunities that they need to execute their potential. Yet it is considerably varied between communities in their commitment to the collective health of children and in the resources that they make available to meet children's needs. This is clearly reflected in the methods in which communities address the issues related to children, specifically concern to their health. If the family, community and country entertain efficiently and effectively with all these child health need only then we can expect a perfect society in future.

Why Urgent Action Needed

The past century has seen vast improvements in our children's health. The infectious diseases that once killed huge numbers of children have largely been conquered.

Dramatic improvements have occurred over the past several decades in such areas as reducing infant mortality, reducing mortality and morbidity from many infectious diseases and accidental causes, increasing access to health care, There have been steady increases in the proportion of immunized children, and both acute mortality and long-term disabilities resulting from certain infectious diseases have been greatly reduced.[8] Yet despite these improvements, India lags far behind other industrialized nations in this and other measures of children's health. Some national indicators raise questions about the health of the nation's children and point to the need for continued appraisal. The children behind each of these statistics face serious barriers to healthy childhoods and healthy, productive adult lives.

Children are generally viewed as healthy when they are assessed by adult standards, and there has been a great deal of progress in reducing childhood death and diseases [3]. But the country should not be blinded by these facts—several indicators of children's health point to the need for further improvement, children in the country do not fare as well as their counterparts in many other developing countries on many aspects of health, and there are marked disparities in health among children within the country. Recent improvements in children's health need to be sustained and further efforts are needed to optimize it. To accomplish this, the nation must have an improved understanding of the factors that affect health and effective strategies for measuring and using information on children's health.

Measuring Child Health – Think Differently

Today for measuring child health we use various indices but it is well known that there are no comprehensive, agreed-upon measures or indices as to what constitutes child health [9]. Some widely used indices to measure child health are summarized in **Figure 2.**

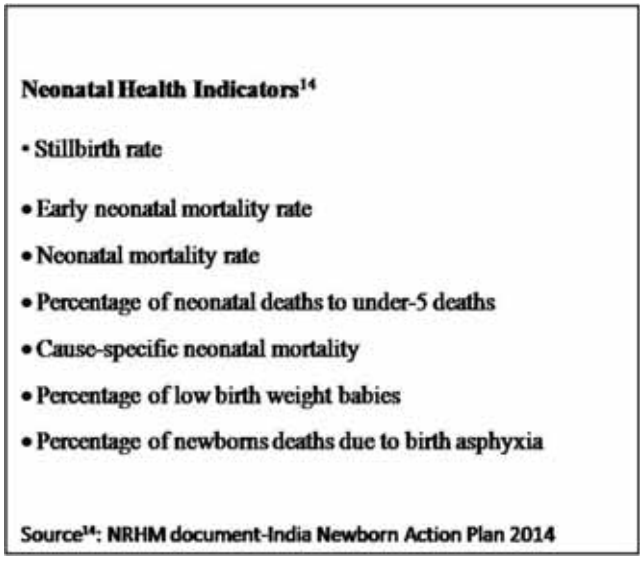

Figure 2: Neonatal health indicators

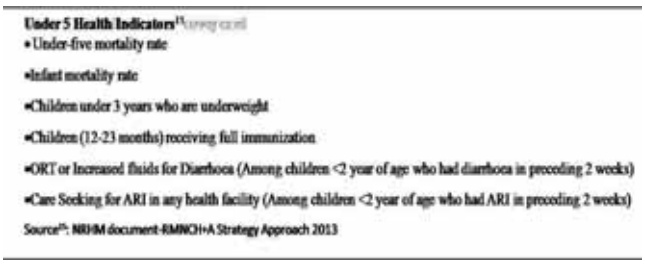

Figure 3: Under 5 health indicators

Figure 4 : Adolescent health indicators

As these indices reflect that major emphasis is on physical dimension of child health but we should not forget that this is only the tip of iceberg, other dimensions are also play crucial role especially in case of children's health.

It is evident that child health is the result of a dynamic set of factors [10]. A multitude of biological, behavioral, and environmental factors can either pose a risk to children's health or act in a protective or health-promoting capacity. For example; children's social environments can be characterized by a number of influences that can be viewed as safe, health-promoting, risky, or detrimental. Many factors (e.g., peers) can be either a risk to health or a protective factor, depending on the specific circumstances [3].

All these factors are responsible not only for changing trends but also for epidemiological transition of childhood diseases as well. The past century has witnessed dramatic changes in child and adolescent mortality and illness. One hundred years ago, infectious diseases were the leading causes of childhood disease and death. Today, social and environmental factors are the principal drivers of child health. Along with the old problems like persistence of communicable diseases, there is an urgent need to address the emerging issues like the threat of non-communicable diseases, HIV AIDS and other nutritional disorders[8]. Thus child health experts and advocates now focus on the precursors, promoters and precipitators of diseases, as well as how all these affects children's health and development throughout childhood and adolescence. So that we can approach and deal childhood health problems in a better manner.

The Nation's Investment in Children: Current Scenario

Earlier, the National policy for Children (NPC) 1974 recognized the need for national programmes for children so that they grow to become 'robust citizens, physically fit, mentally alert and morally healthy" Further, National Charter of Children adopted by India in 2004 underlined the intent to secure for every child right to enjoy a healthy and happy childhood and all root causes that negate the healthy growth of children To affirm this commitment, Government of India adopted in the NPC 2013, a long term, sustainable, multi-sectoral, inclusive and integrated approach for the development and protection of children i.e. 0-18 age group. Survival, health, nutrition, development, education, protection and participation are the key priorities of the policy. It reiterates the State's commitment to ensure equitable access to essential, preventive, promotive, curative and rehabilitative health care for all children[11]. Concurrent assessment of child health services under national health programmes highlight various areas of default and demands to deal the issue in a proper manner. In this view Important Thrust areas under Child Health programme are

Thrust Area 1 : Neonatal Health

- Essential new born care (at every 'delivery' point at time of birth)
- Facility based sick newborn care (at FRUs & District Hospitals)
- Home Based Newborn Care

Thrust Area 2: Nutrition

- Promotion of optimal Infant and Young Child Feeding Practices
- Micronutrient supplementation (Vitamin A, Iron Folic Acid)
- Management of children with severe acute malnutrition

Thrust Area 3: Management of Common Child hood illnesses

- Management of Childhood Diarrhoeal Diseases & Acute Respiratory Infections

Thrust Area 4: Immunisation

- Intensification of Routine Immunisation
- Eliminating Measles and Japanese Encephalitis related deaths
- Polio Eradication

The strategies for child health intervention focus on improving skills of the health care workers, strengthening the health care infrastructure and involvement of the community through behavior change communication to implement policies and programme more efficiently and achieve objective on time.

We Must Act Now

In brief, we find that despite major gains over the past century, children's health varies widely across population subgroups and lags well behind that of many other industrialized nations. Furthermore, although public health-care expenditures for children have grown steadily, this growth has come from expanded eligibility for publicly financed health insurance and substantial increases in the cost of health care. Rising health expenditures have coincided with the erosion of public investment in education, housing, and social services, all of which are thought to affect health, especially among the poorest children.

Unless we act now, our children will continue to fall farther behind, putting our society, economic prosperity, and national security at even greater risk. So it is the time of intense and dominant commands like

1. **Well equipped infrastructure Establishments to handle recent child health needs and provide direct care** – Attending infections on need, addressing new emerging chronic conditions by ensuring kids take medication when carried in eg. Asthma, Diabetes etc, first aid in case of injuries at play and school, maintenance of health records, good day care services as small retiring room in case of sickness etc.
2. **Regular and Periodic Health screenings** – Plan and schedule tests – nutritional status, sugar, vision, hearing, dental exams etc – Any early diagnosis will help us to manage problems more easily as well as avoid spending resources at a later date.
3. **Well Coordinated School Health Plan** – Inquire about medical facilities in school before admissions, Review of sanitation and hygiene facilities in school, Mid-day meal nutrition, coordinate with local medical authorities and NGOs to track and communicate epidemics and infections in local neighbourhood and early preventive measures for it.
4. **Awareness and Health Education** – Educate and aware the kids on age-relevant health topics – prevention measures, food nutrition, hygiene, open communication on medical condition, medication and side effects, safety measures etc.
5. **Proper coverage of allied health areas** – Training and awareness programmes should be conducted depending on resource availability and contextual importance – ragging, anger management, psychological counseling, smoking, alcohol, sex, drugs, suicide tendency etc.

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