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Highlights in this issue

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Prof. Sanjeeb Kakati

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ANNOUNCEMENT

API (Assam), in collaboration with the Indian Railway Medical Service Association, N.F. Railway, is pleased to announce that it is going to organize the **4th *East Zone Annual Conference of API (EZ APICON, 2014)*** along with the *24th Assam APICON*, to be held on **13th and 14th December 2014** at the Luitgarh Railway Officer's Club Auditorium, Maligaon, Guwahati.

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ASSAM JOURNAL OF INTERNAL MEDICINE

Official Journal of Association of Physicians of India, Assam Chapter

EDITOR : PROF. SANJEEB KAKATI

C O N T E N T S

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CASE REPORT

Editorial

The Dementia spectrum

Kamala Deka*

Dementia, is a recognizable cause of morbidity and disability across the world. As aging population has been increasing in developing Country (WHO), India may have dementia epidemic in near future. A wide range of etiology encompasses dementia spectrum with a commonality of cognitive decline.

Various etiological consideration has been examined in a hospital based population study from north east India[1] and provides replication of the findings that reversible dementia that are potentially treatable constitute a significant number in India and degenerative variety still stands as commonest one[2]. The fact is in India, dementia research demonstrates degenerative being the commoner type followed by vascular cause and few no. of reversible etiologies[3,4]. Among the early onset degenerative dementias Fronto-temporal group has occupied the top in the list than Alzheimer's or Vascular dementias in this study and otherst[4,5]. Alzheimer dementia is not common in India and a disease of the west[5]. However due to increasing trend of aging population Alzheimer dementia is no longer an uncommon type in India.

The MMSE (mini mental state examination) a popular screening tool is used to detect dementia. It is not a diagnostic tool as patients with Fronto-temporal or vascular dementia may retain high MMSE score until later in the disease. So the standard operationalised criterias used in the study for degenerative dementias especially Fronto-temporal, Dementia with Lewy body, Vascular and Alzheimer dementias were actually needed. Single Lewy body dementia was reported though LBD is common worldwide, difficult to differentiate from Parkinson's dementia at times. Regional difference may explain the less no LBD cases or the clinical diagnostic criteria that are used has high specificity but lower sensitivity[6] and in such, improved method of case detection may suggest helpful. Many a times presence of Parkinsonian symptoms early in Parkinson's disease and late in LBD may be able to differentiate the two. No mention about making the diagnosis of dementia with Parkinson's disease has been mentioned. Blank facial expression that we see in Parkinson's may obscure the intellectual decline, slow movements may conceal the retarded intellectual activity, associated depression which is an invariable phenomena in Parkinson's may appear to modify the symptoms of dementia in Parkinson's disease. So, to recognize dementia with Parkinson's disease careful examination along with standardized psychological test to detect associated depression and any standardized memory scale may be useful.

Further, etiological diagnostic confirmation has been made on medical, clinical, neuropsychological, brain imaging, EEG, Chest x-ray and lab tests and so on.Leutic infection, gluten related and anti TPO dementias are not commonly seen worldwide. Presence of these novel etiological factors warrant clinicians for detailed investigations for any such potentially treatable conditions keeping in mind the whole range so that early detection is made and possible expected outcome is achieved with treatment. Dementia due to vit B12 deficiency,

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thyroid related, neurocysticercosis are seen in India.

The use of specific operationalised diagnostic criteria, multiple rating scales for cognitive examination along with laboratory tests made a comprehensive assessment to the etiological diagnosis of Dementia.

Severity of dementia was also assessed on the clinical dementia rating scale (CDR) and confirmed a known fact that degenerative dementias are more severe than reversible dementias. Ideally CDR Score corresponds with MMSE Score and stages of the dementia may be ascertained. Here, in reversible type CDR score 1.2 (mean) corresponds with MMSE score 20.8 (mean) indicate mild dementia and CDR of 1.9 (mean) with MMSE score 15.6 (mean) indicate moderate dementia in degenerative group. The reversible group of dementias show improvement improved with increased MMSE score following treatment.

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Original Article

Etiological Spectrum of Dementia in North-East India – A Hospital Based Study

N P A Burji*, A Mahanta, M Das***, M Goswami****, A K Kayal*****

Abstract

Objective : To determine the pattern of dementia among patients attending a speciality clinic with respect to etiology and clinical spectrum

Methodology: A prospective analysis was carried from August 2009 to August 2011. All patients underwent neurologic ,cognitive and psychiatric evaluation according to a standard protocol . Screening was done by the Mini-Mental Status Examination . The DSM-IV criteria for diagnosis of dementia was followed. Standard published criteria was followed for diagnosis of specific subtype . Necessary hematological, biochemical and serological blood examination was done along with neuroimaging.

Results : A total of 30 patients Male :Female (7.66:1),mean age of 60.93 (±12.12 SD) years and mean education class VIII were diagnosed to have dementia .Of the various etiologies, the commonest was Frontotemporal Dementia (26.66 %), followed by Vascular Dementia (20%),Alzheimer Disease (13.33%), parkinsonism with dementia (10%). The reversible causes of dementia comprised 30 %(9 patients) which included leutic infection , hypothyroidism, vitamin B12 deficiency, gluten related Dementia, Anti –TPO antibody associated and neurocysticercosis.

Conclusion: Degenerative causes followed by the vascular causes were responsible for the majority of cases of Dementia.The novel findings in our study was the significant number of patients who had reversible causes of dementia (30%) .Key words: Degenerative, Vascular, Reversible.

INTRODUCTION :

Dementia is the development of multiple cognitive deficits that includes memory impairment and at least one of the following—Aphasia, Apraxia, Agnosia or disturbances in the executive functioning. The prevalence of dementia is increasing with the increase in global life expectancy. It is a disease strongly associated with increasing age. Dementia is a major public health problem among the elderly in industrialized countries. It can also have a devastating impact on developing countries whose population is aging most rapidly[1,2]. The most remarkable effects of the aging population are expected in the most rapidly developing regions such as China, India and Latin America[2]. By the year 2020, 70% of the world population aged above 60 years will be that in the developing countries, with 14.2% in India[3].

It is estimated that 24.3 million people worldwide have dementia, with 4.6 million new cases of dementia every year. Dementia is a clinical diagnosis whose evaluation involves assessment of the presenting problem; history about the patient that is provided by an informant, complete physical and neurological examination; evaluation of cognitive, behavioral, and functional status; and laboratory and imaging studies.[4]

It is estimated that around 1.5 million people are affected by dementia in India [2]. This number is likely to increase by 300 % in the next four decades. This aging population will place urgent demands on the health care systems in the developing countries most of which are ill-prepared for fulfilling such demands. According to the World Health Organisation (WHO) Global Burden of Disease report 2006, dementia is the third leading cause contributors to years if life lost due to disability (YLD) in the elderly in low-income and middle-income countries.

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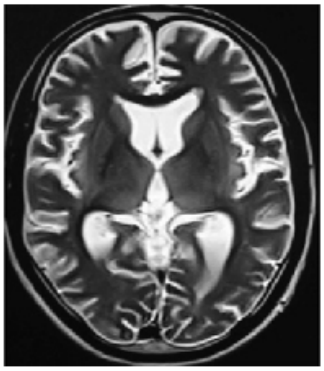
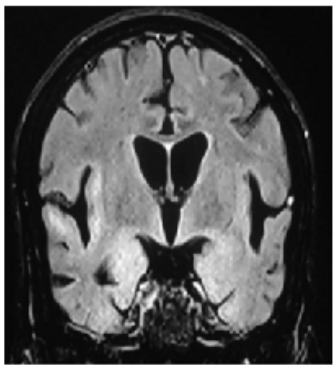
The WHO Report estimated that dementia is the second highest source of disease burden after tropical diseases.The World Health Organization (WHO) projects that by the year 2020, Asia and Latin America will have more than 55 million people with senile dementia[5].

Dementia due to potentially reversible etiologies is an important group of dementias to be identified not only because of the increasing number of such patients recently encountered, but also due to the potentiality for substantial improvement with treatment. The reported frequency of dementia due to potentially reversible causes varies from 0 to 23%[6-9]. In the recent years, more attention has been given for the early diagnostic evaluation of patients with dementia who may have reversible or treatable conditions. Guidelines suggest that all patients presenting with dementia or cognitive symptoms should be evaluated with a range of laboratory tests, and with structural brain imaging with computed tomography (CT) or magnetic resonance imaging (MRI)[10].

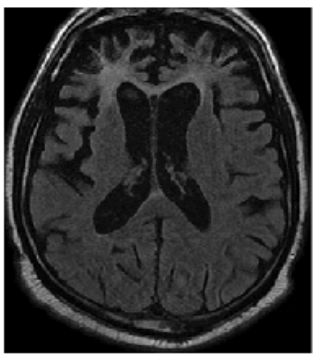
Fig 3 : Spectrum of Reversible Dementia.



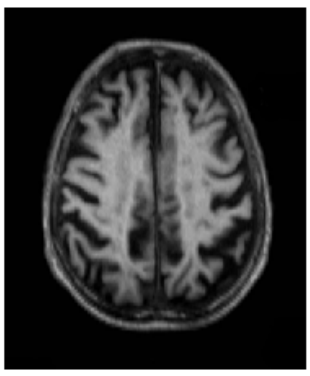
T2W image of MRI brain having increased signal intensities involving bilateraltemporoparietal region of the leutic dementia



MRI FLAIR hyperintensity in bilateral medial temporal lobes of leutic dementia



Frontal atrophy on fMRI of a patient with behavioural variant of FTD (bvFTD)



Parietal and Temporal atrophy in AD

Study on the clinical profile of dementia in India as a whole are limited and data from Northeastern India is also lacking. Keeping in view of the above facts this prospective study was aimed to estimate the clinical profile of dementia and various etiological factors of the dementing illness.

MATERIALS AND METHODS :

This prospective study was performed in Gauhati Medical College Hospital from August 2009 to August 2011 .All subjects were from the Dept. of Neurology and its allied departments like Medicine and Psychiatry. The history, general and neurological examinations was conducted. The most common language that the study population could understand was Assamese. For neuropsychologic assessment, we used the Mini-Mental Status Examination (MMSE) for screening [11,12] and the Addenbrooke Cognitive Examination - Revised (ACER) for further testing of cognitive subdomains (orientation, attention, memory, verbal fluency, language and visuospatial function). The history, general and neurological examinations, including neuropsychological assessments was recorded in a semistructured proforma.

An MMSE score below 24 (out of a possible score of 30) was evaluated for further clinical diagnosis. The MMSE has been used as a screening tool to detect dementia [11-14] which is stated in many studies . We also administered the functional ability questionnaire (Everyday Abilities Scale for India, EASI) to the family members [15]of the subjects who were cognitively untestable because of sensory impairment, illness, or severe dementia. Alzheimer's disease was diagnosed using - DSM-IV plus NINDS –ADRDA criteria and for Vascular dementia - DSM-IV plus NINDS-AIREN criteria were taken[10,16 ,17]. The diagnosis of Fronto-temporal Dementia (FTD) was based on Lund-Manchester criteria and for dementia with Lewy bodies (DLB)[18] , International consensus consortium criteria for dementia with Lewy bodies was used. Hachinski Ischaemic Score was taken for differentiation of the Dementia of Alzheimer's Type (DAT) and Vascular Dementia (VaD).The Clinical Dementia Rating scale was used to evaluate the severity and to assess the outcome after treatment . The Clinical Dementia Rating (CDR) is a five-point scale in which CDR-0 connotes no cognitive

impairment, and then the remaining four points are for various stages of dementia:

CDR-0.5 = Very mild dementia
CDR-1 = Mild Dementia
CDR-2 = Moderate Dementia
CDR-3 = Severe Dementia

Patients diagnosed to have pseudodementia and Mild Cognitive Impairment (MCI) were excluded from the study.

Laboratory tests included: Complete blood count and Erythrocyte sedimentation rate, Fasting lipid profile, Sr. urea, Sr. creatinine, Sr. sodium, Sr. potassium, Sr. calcium, Sr. phosphorus, Fasting and postprandial blood sugar, Liver Function Tests, Thyroid Hormone Assay,Anti-TPO (Anti-thyroid peroxidase antibodies), Serum Vit B12 levels, Serum folic acid levels, Venereal Diseases Research Laboratory (VDRL) test, Sr *T Pallidum* haemagglutination (TPHA),Serum Ammonia levels and HIV screening. Cerebrospinal fluid (CSF) analysis was done which include cell count, cell type protein, sugar and VDRL. Electro Encephalogram (EEG), Chest X -ray and Ultrasound abdomen and pelvis were done. Neuroimaging in the form of computed tomography (CT) or magnetic resonance imaging (MRI), or both, were performed.

The statistical analysis was done using SPSS 14 software and the mean, Standard Deviation (SD) and p Values were applied.

RESULTS :

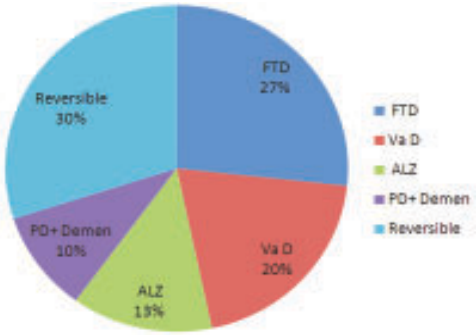
A total of 30 patients were diagnosed to have dementia. 23 patients were male and 7 were females forming a ratio of 7.66:1. The mean age of the patients were 60.93 (±12.12 SD) years. The age group was ranging from 43yrs to 88 yrs. There were 15 cases between 40-60 yrs and 15 cases were above 60yrs (Table 6). The p value when compared between 40-60 yrs and that >60yrs were not significant (0.5808). This suggested that there were equal distribution of cases among these groups The mean education class was VIII. None had a positive family history. The spectrum of dementia of our study is illustrated in fig 1 of the various etiologies, the commonest was Frontotemporal Dementia (FTD) occurring in 8(26 .7%) patients .The mean age of the patients with FTD was 54.4+/-7.6 yrs ranging from 45-71yrs. The second commonest cause was Vascular Dementia which comprised 6 cases (20%) . The risk factors for the patients with vascular dementia were: hypertension (4), diabetes mellitus (2) and dyslipidemia (2) . 4 patients (13.3%) had Alzheimer Disease and Parkinsonism with dementia in 3 patients (10%). The reversible causes of dementia comprised 9 patients (30 %) [fig 2]. They included 1) CNS Leutic infecction -3 patients (Diagnosed on the basis of criteria in the table 4) 2) Thyroid related –

Table 6 : Age Profile of our Cases

Age	Male	Female
40-50 yrs	5	3
50-60 yrs	5	2
60-70 yrs	6	1
>70yrs	7	1

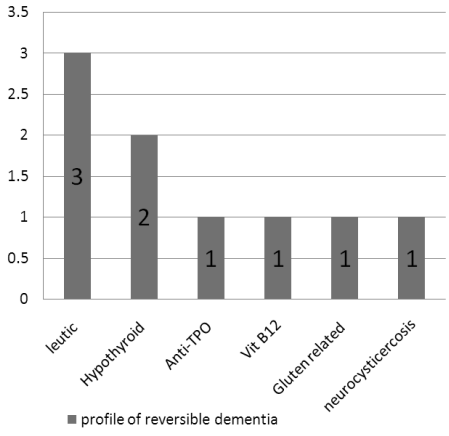
The mean age group was 60.93yrs +/- 12.12 SD
The age group varied from 43 yrs to 88 yrs
p value when compared between 40-60 yrs and that >60yrs was not significant(0.5808) suggesting equal distribution of cases among these groups
There were 15 cases between 40-60 yrs and equal number of cases above 60yrs

Fig 1 : Spectrum of Dementia in our study



FTD- Fronto Temporal Dementia
Va D- Vascular Dementia
ALZ –Alzheimer's Dementia
PD+ Demen – Parkinsons disease with Dementia
Reversible –Reversible Dementia

Fig 2 : Spectrum of Reversible Dementia



Hypothyroid-2, Anti Thyroid Peroxidase (TPO) antibodies -1

3) 1 patient each of vitamin B12 deficiency, gluten related Dementia and neurocysticercosis.

The mean age of presentation of the reversible dementia was 56.66yrs (ranging from 40-70yrs). The mean duration of symptoms of our 30 patients before presenting to the Clinic was 19.7 (± 9.78 SD) months which was ranging from 4 to 32 months .There were 21 patients in the degenerative group and 9 of reversible dementia .We also compared the differences in the mean duration of illness among the degenerative and reversible group . The duration of illness when compared between the two groups suggested that degenerative group [24.3(±8SD)] months' had longer duration of illness as compared to reversible group [9 (± 3 SD)] months It was also statistically significant (p value < 0.001)

The mean MMSE score at the time of initial examination was 18.2 ± 3.67 SD (range 8 to 22). The mean MMSE when compared (table 1) between the two groups suggested that degenerative group [15.6 (±2.6SD)] had low MMSE as compared to reversible group [20.8(± 3.5 SD)]. It was also statistically significant (p value < 0.001). The Clinical Dementia Rating Scale (CDR) score between two groups on admission is shown in table 2. The degenerative group had a higher CDR score suggesting more severity of illness in them.

Table 1 : Comparison of mean MMSE among the two groups during admission

	Reversible	Degenerative
Mean MMSE	20.8	15.6
SD	3.56	2.62
N	9	21

p value = 0.0001 (statistically very significant)
MMSE— Mini–mental state examination
N=number of cases,SD=Standard Deviation

Table 2 : CDR score depicting the severity of the disease between two groups

	Reversible	Degenerative
CDR score	1.2	1.9
SD	0.46	0.35
N	9	21

p value –0.0029 (Statistically significant)
CDR—Clinical Dementia Rating Scale
N=number of cases, SD=Standard Deviation

DISCUSSION :

This prospective study was aimed to estimate the clinical profile of dementia and various etiological factors of dementing illness in this part of India . A total of 30 patients were diagnosed to have dementia. The study period was from August 2009 to August 2011 in the tertiary care hospital. None had a positive family history .In contrast, the study done by Das et al showed that one fifth of their patients had a positive family history[19]

The commonest was the Frontotemporal variant occurring in 8(26 .6%) patients. This prevalence is in accordance with the study by Ratnavalli et al who showed ,the prevalences of early-onset FTD was 15 per 100,000 (8.4 to 27.0 %) with the age ranging from 45 to 64 years . [20] Frontotemporal dementia (FTD) is a clinically and pathologically heterogeneous syndrome, characterized by progressive decline in behavior or language functions associated with frontal and temporal lobe degeneration. An interesting study, in terms of the distribution of FTD across four ethnic groups, claimed that it was as common in Asians as Caucasians in the younger dementic group.[21-24]

In the study of Chow et al, the median age of onset of FTD was about 58 with 22% of the patients having an age of onset after age 65.7.[25] Acccordingly, the patients with Frontotemporal Dementia in our study were 54.4+/-7.6 yrs ranging from 45-71yrs . Our study also closely correlates with the Chinese studies where the FTD group had a younger age at onset . 3 patients were less than 50 yrs age in our study . The Male : Female ratio was 5:3.This is in accordance with study done by Ratnavalli where the mean age at the onset of FTD was 52.8(+/- 4) years and there were male preponderance (14:3)[20] .

The study from Das et al which focussed on young onset Dementia also showed that before the age of 50, it was FTD and neither VaD nor AD was the most common cause [19] . In one study done in Oman by Shelley BP , front temporal dementia constituted 9.5% of the cases[26] .

The patients in our study had behavioural variant of FTD (bvFTD) and progressive non-fluent aphasia (PNFA) type of FTD. Many Chinese studies have shown that behavioural variant of FTD is the commonest among FTD.[27,28]

The second commonest cause was Vascular Dementia which comprised 6 cases .Five (5) of the patients had Multi-infarct type and 1 had Subcortical infarct, suggestive of Binswanger's disease . Alzheimer's Disease was present in 4 patients in our study. Alzheimer's disease (AD) is the most common sub-type of dementia with approximately two-third of dementia cases above 65 years being diagnosed as AD[29]. But in our study it constituted 13.33%. All patients of AD were above 65yrs of age in our study. 2 patients were male and 2 were females . It is generally believed that men and women are equally at risk of Alzheimer's disease.

All the patients were from urban locality. This is in accordance with the recent research in India and Africa which showed that the risk of Alzheimer's disease is higher for urban as compared to rural areas.[30, 31] In the study by Das et al where the Young onset Dementia (<65yrs) were studied Alzheimer's disease constituted 31(33%) of the 93 cases. Family history of Alzheimer's disease was present in 7 patients. In our study , the age group of the patients with Alzheimers Dementia was more (66+/-1.3 yrs) as compared to patients with Frontotemporal dementia 52.8(+/- 4) yrs. This is in accordance with study by Shinagawa S et al which showed that in the early-onset group, the rates of patients with Alzheimer's disease were relatively low and the rate of patients with Frontotemporal lobar degeneration was relatively high.[32]

3 patients were diagnosed to have parkinsonism with dementia constituting 10% of our cases. They were of Multisystem atrophy (MSA), Corticobasal degeneration (CBD) and Dementia with Lewy bodies (DLB). The reversible causes of dementia as described above comprised 30 % (9)patients. The reported frequency of dementia due to potentially reversible causes in the worldwide varies from 0 to 23%[6-9]. Commonest among these causes are alcohol related Dementia, surgical brain lesions such as Normal Pressure Hydrocephalus [NPH] and chronic subdural hematomas, metabolic disorders such as hypothyroidism, hypoparathyroidism, vitamin B12 deficiency and central nervous system (CNS) infections such as neurosyphilis as depicted in Table 3. In study done by Srikanth et al,18 % (24 patients) were diagnosed to have reversible causes in which 11 patients had neuroinfections, normal pressure hydrocephalus in 8 patients and vitamin B12 deficiency in 5 patients [33].In our study, 3 patients had leutic related which was diagnosed by applying the criteria by Pope et al as shown in Table 4[34]. 2 patients had hypothyroid related Dementia. In the study done by Das et al, which had focussed on young onset dementia, none of the patients had leutic dementia. 2 patients had hypothyroidism in their study [19] .There were one patient each of vitamin B12 deficiency, gluten related Dementia, Anti thyroid peroxidase (TPO) antibodies related Dementia and neurocysticercosis in our study. In the study done by Das et al, 3 patients of neurocysticercosis were present but none had gluten related, Anti TPO antibodies related Dementia and vitamin B12 deficiency related Dementia.

Table 3 : Comparison of reversible etiologies in different studies from the present study

Etiology of reversible dementias	**Present Study**	**SK das**	**Srikanth**
B12 deficiency	1	-	5
Normal Pressure Hydrocephalus (NPH)	-	-	6
Neurosyphilis	3	-	5
Neurocysticercosis	1	3	1
HIV dementia	-	-	1
Cryptococcal meningitis	-	-	1
Tubercular meningitis	-	-	1
Hypothyroid	2	2	-
Cns vasculitis	-	2	-
Multiple sclerosis	-	2	-
Anti TPO, Antibody	1	-	-
Gluten related	1	-	-
Total patients of Reversible cases	**9**	**10**	**24**
Percentage of Reversible cases	**30%**	**2.79%**	**18.6%**
Total Patients	**30**	**379**	**129**

Table 4[36] : Diagnostic criteria for Leutic infection Definitive diagnostic (requires 1 or 2 and 3)

1. Identification T.pallidum in csf or CNS tissue by microscopic examination or animal inoculation or by PCR
2. A reactive serum treponemal test
3. A reactive VDRL-CSF test on spinal fluid sample

Presumptive diagnosis (requires 1 and 2 or 3)

1. A reactive serum treponemal test
2. Clinical signs of neurosyphilis
3. Elevated CSF protein or leukocyte count in the absence of other known causes

The patients of leutic etiology were treated with Inj. Procaine penicillin 2.4 million units per day for 14 days.

The patient of vitamin B12 deficiency dementia was aged 65yrs and was a strict vegetarian with the MMSE of 22 .He was treated with injectable methylcobalamin as per hospital protocol.After 11 months patients MMSE had improved to 25.

In the study by Srikanth S, 5 patients had vitamin B12 deficiency but none had gluten related Dementia and neurocysticercosis[33]. On follow up after 1 year ,the patients of reversible dementia had improvement in the mean MMSE which was statistically significant (p value-0.0141) (Table 5).

There was one patient of gluten related Dementia who came with symptoms of diarrhoea, steatorrhea, and abdominal cramping since 9 months. Neurological features included dementia and sensorimotor axonal peripheral neuropathy.His admission MMSE score was 18 His serum for Anti-gliadin Antibody was positive. IgG anti-gliadin antibodies have been the best diagnostic marker in the neurological diseases [35]

Table 5 : Outcome of patients of the MMSE score in reversible dementia after treatment with average follow up of 1 year

	Before treatment	After treatment
mean MMSE	20.8	24.6
SD	3.56	2.11
N	9	9

The patients of reversible dementia had improvement in the mean MMSE
p value-0.0141(statistically significant)
N=number of cases , SD=Standard Deviation
MMSE— Mini–mental state examination

After being on gluten free diet for 8 months, his MMSE score gradually improved to 22.

Gluten sensitivity as a neurological Illness has described in many studies in one study where the metaanlysis of 35 papers of single or multiple case reports were done from 1964 to 2000, they found a total 83 of patients of celiac disease with neurological involvement. Dementia was found in 6 patients making it to 7.2% [35.]

There was one patient of Hashimotos dementia with elevated Anti-thyroperoxidase (TPO) of 500 IU/ml.He was 55 yr old male antibody Anti TPO was done using Siemens Immulite 1000 Anti-TPO assay (normal <150 IU/ml). He had insidious onset of cognitive dysfunction over 8 months .Patient was in euthyroid state. He was started with pulse therapy of Intravenous Methylprednisolone 1 gm/day for 5 days and was later given oral prednisolone which was gradually tapered over 12 weeks

The prevalence of Hashimotos dementia in many studies have estimated at 2.1 in 100,000 subjects The mean age is between 45 and 55 years, and it occurs five times more frequently in women than in men Patients may be hypothyroid, hyperthyroid, or euthyroid.

CONCLUSION :

While the degenerative causes followed by the vascular causes were responsible for the majority of cases of dementia, we also found that there were a significant number of patients who had reversible causes of dementia in our study making up to 1/3 of our subjects. There was a statistically significant improvement of cognitive functions (MMSE) in some of these patients with the treatment .The high percentage of leutic dementia and the presence of Anti-TPO and gluten related Dementia in our study novel finding when compared worldwide. So, it can be concluded that all the patients presenting with cognitive impairment should be thoroughly evaluated for detection of potentially reversible cause and if detected to be treated adequately.

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Original Article

Intra articular cortisone infiltration improves the outcome of manipulation treatment in Adhesive capsulitis (periarthritis) shoulder joint

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Abstract

Background : Manipulation under Anaesthesia is an effective method of treating Adhesive capsulitis . Cortisone Infiltration inside joint during manipulation minimizes inflammatory reaction so that patient can undergo physiotherapy regime immediately. We aim to study results of intra articular cortisone infiltration in Adhesive capsulitis treated by manipulation under Anaesthesia followed by physiotherapy and compare the same in cases where no infiltration was done.

Materials & Methods : From Aug.2004 to July 2007, 70 patients, aged 40-70 years, who satisfied the definition of Adhesive capsulitis were included in our study. Patients were divided into two groups ,Group A not receiving and Group B receiving intra articular Triamcinolone Injection, prior to manipulation. Manipulation was done under general anaesthesia. Physiotherapy was started from day one .All patients were clinically assessed at 3, 6, and 12 weeks after treatment.

Results and Observations : Highest incidence was seen in 40-49 years of age with female preponderance (69%).Pain levels & ROM were recorded at the start and at subsequent follow up and final results were evaluated by Haggarts classification. Group B showed better results ,both subjectively and objectively, than the Group A, (p value .0146 & .0240 respectively) which was statistically significant at final follow up. There was no complication following manipulation.

Conclusion : Manipulation under Anaesthesia, provides an effective treatment for Adhesive capsulitis of Shoulder with majority of the patients reporting successful end results, provided the post-manipulation pain is taken care of by Intra Articular injection of Triamcinolone which enabled the patient to take part in post manipulation physiotherapy program effectively.

KEY WORDS : *Adhesive capsulitis Manipulation and Cortisone infiltration*

INTRODUCTION :

Adhesive capsulitis **[**Periarthritis] is one of the commonest condition involvingShoulder joint. It is said to be a self limiting disease with three distinct stages of Freezing or painful phase, Frozen or transitional Phase with Pain slowly reducing but increasing stiffness & Thawing Stage with gradual return of shoulder motion[1].

Various modalities of treatment have been advocated from time to time. This includes Conservative treatment in the form of analgesics, systemic or intra-articular steroids, physical therapy, muscle stretching, and distension arthrography. Operative treatments consist of mainly two types : (1) manipulation under general anaesthesia and (2) arthroscopic release of the joint capsule followed by an early rehabilitation protocol[2].

Manipulation under Anaesthesia is one of the very effective methods of treatment in such ailment[3-5]. In Adhesive capsulitis there is thickening and contracture of the capsule which become adherent to the head of the humerus. There are also adhesions between opposed synovial surfaces in the redundant fold of the capsule[6] .Although manipulation results in complete tear of adhesions, there is inflammatory reaction to this tear inside the joint which prevents the patient to undergo physiotherapy immediately because of severe pain. This invites failure in such cases. Infiltration of Cortisone inside the joint during manipulation had been advocated by Thomas B. Quigley. in order to minimize the inflammatory reaction so that the patient could participate in the physiotherapy regime immediately.[7]

We aim to study the results of intra articular cortisone infiltration in Adhesive capsulitis of shoulder joint treated by manipulation under Anaesthesia followed by physiotherapy and compare the same in cases where no infiltration was done.

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MATERIALS AND METHODS :

70 patients with Adhesive Capsulitis, all of which attended Out Patient Department from Aug. 2004 to July 2007 and satisfied the definition of Adhesive capsulitis, as mentioned below, were entered into the study. The patients were examined thoroughly with detailed history and clinical findings. Investigations included Routine Blood Examination including Blood sugar, Routine Examination of Urine, X-ray of Shoulder joint. In doubtful cases, investigations were done to rule out causes of symptoms and signs other than Adhesive capsulitis. Patients were selected as per criteria mentioned below.

Inclusion criteria : (a) Pain in the shoulder for at least four weekswith Sleep disturbance due to night pain and inability to lie on the affected shoulder and Restriction of all active and passive Range of Movement [AROM/PROM], with a reduction of at least 50% or more as compared to the contralateral shoulder (b) failure of adequate conservative treatment in the form of Medications to reduce pain and physiotherapy for at least four weeks.(c) normal x-ray of shoulder.

Exclusion criteria : (a) symptoms and signs arising out from the cervical spine, spinal cord and its roots, brachial plexus and its branches. (b) Symptoms due to Impingement at shoulder due to various causes and other lesions of Rotator Cuff (c) symptoms of generalized arthritis, Traumatic, infective and tumors lesions in and around the shoulder joint(d)symptoms of referred pain (e) Associated Diabetes mellitus (f) associated co morbid conditions that precluded manipulation under General Anaesthesia (g) patients who could not take part in post manipulation exercise programs. (h) recurrence from previous manipulation (i) Bilateral involvement Patients fulfilling the above criteria were included irrespective of their sex.

Abduction and external rotation were recorded with goniometer attached to the upper arm while internal rotation was recorded as the level of vertebral spine reached by the extended thumb.All patients were clinically assessed before treatment and at 3, 6, and 12 weeks after treatment.

Method of treatment : Patients were divided into two groups depending upon the method of treatment given. Group A, patients were treated with manipulation alone. Group B, patients were treated with manipulation along with intra-articular cortisone infiltration. Just before induction of anaesthesia for manipulation, 1ml containing 40mg Triamcinolone Acetonide mixed with 4ml of 2% lignocaine was injected locally into the shoulder joint through the posterior approach.

Patients were allotted in either groups of treatment on an alternate basis while the first pair was selected by lottery.Both the groups were assigned to physiotherapy from day one.

Method of manipulation : With the patient supine in the OT table and under general anaesthesia, with utmost gentleness, manipulation was done in pure gleno-humeral abduction, fixing the scapula with one hand and abducting the humerus with the other. The humerus was gently but firmly abducted. A tearing noise was felt and heard and a sudden release of resistance occurred, followed by a free range of abduction. As a rule external rotation would found to be restored to normal. Occasionally, further gentle manipulation of external rotation was done to regain rest of External rotation if necessary. Then manipulation was done to regain internal rotation.

After treatment : as the patient regained consciousness, ice packs were applied locally. Position of the limb after manipulation was abduction and external rotation. Medications included injectable analgesics in the same evening, oral analgesic and anti-inflammatory drugs from the next day for 2 weeks, and antioxidants.

Physiotherapy from day one included, shoulder ladder, shoulder wheel, pulley, pendulum exercise, infrared heating; all daily for six days in a week for 3 weeks at physiotherapy clinic. After 3 weeks patient was instructed to continue shoulder exercises like pendulum, circumduction, wall crawling, both internal and external rotation exercises, at home. Further follow up was done at 6 & 12 weeks

Pain levels & ROM were recorded at the start and at subsequent follow up and evaluated finally according to Haggarts classification [Table 1] that included both subjective and objective evaluation[8] which was later endorsed by Parker et al in 1989[9]

Table 1 : Haggart's classification of results.

Excellent	Full range of motion with no pain
Good	Occasional discomfort but full range of motion
Fair	Recurrent mild shoulder discomfort with decreased range of motion but able to function
Poor	Decreased range of motion, constant pain and unable to resume activity.

RESULTS AND OBSERVATIONS :

Adhesive capsulitis of Shoulder comprised an incidence of 3.73% of all cases attending OPD. Highest incidence (46%) was seen in 40-49 years age group followed by 41% in 50-59 years.(Table 2).

Table 2 : Showing age of the patients

Age Group	Number	Percentage
40-49	32	45.72
50-59	29	41.42
>60	09	12.86
Total	70	100%

Females showed an increased incidence of involvement (69%). (Table 3)

Table 3 : Showing Sex of the patients

Sex	No of patients	Percentage
Female	48	68.57%
Male	22	31.43%
Total	70	100%

Left sided Adhesive capsulitis was more common (58%)

Commonest mode of onset was spontaneous (84%). Other mode of onset was trivial trauma like sprain following fall or lifting weight but without immediate period of inactivity of the shoulder and without any evidence of fractures or rotator cuff injury .

Between the two groups, Group B [with intra articular infiltration] showed better results both subjectively and objectively than the Group A [without infiltration].(Table 4)

Table 4 : Showing Subjective and Objective end results

Result	Subjective end result		Objective end result	
	Group A	Group B	Group A	Group B
Excellent	00(00.00%)	19(54.27%)	05(14.29%)	24(69.00%)
Good	15(42.85%)	07(20.00%)	17(48.57%)	07(19.00%)
Fair	11(31.42%)	09(25.73%)	11(31.43%)	04(12.00%)
Poor	09(25.73%)	00(00.00%)	02(5.71%)	00(00.00%)
Total	35(100%)	35(100%)	35(100%)	35(100%)

There was 75% Excellent to Good Subjective End result in Group B as compared to 43% in Group A which was statistically significant (p = .0146 /p=< .05). There was 88% Excellent to Good Objective End result in Group B as compared to 63% in Group A which was statistically significant (p=.0240 /p=< .05).

We did not encounter any intra operative complications in our cases.No recurrence of symptoms could be seen in any of the two groups .

DISCUSSION :

Adhesive capsulitis is a complex shoulder condition of unknown etiology and is a clinical diagnosis manifested by pain and stiffness around the shoulder joint.[10]

Duplay first reported a case in 1872 [11] and Codman defined it as "Frozen Shoulder"[12], characterized by slow onset pain near the insertion of deltoid, inability to sleep on the affected side, painful and restricted elevation and external rotation and a normal radiological appearance. Neviaser started defining it to be Adhesive capsulitis He found fibrosis, inflammation, and capsular contraction in glenohumeral joint causing adhesions to the capsule and the anatomic neck of the humerus which was the basis for pain and a restriction of motion in the joint[5].

Even with the advent of arthroscopy this clinical entity has remained as a specific one, distinctly different from other conditions which produce pain and stiffness of the shoulder joint, like lesions of rotator-cuff, bicipital tendon, subacromial space, cartilage etc.

Mode of onset in adhesive capsulitis was reported to be spontaneous in majority of the studies [13].The Commonest mode of onset in our cases was also was spontaneous (84%). Other mode of onset was trivial trauma like sprain following fall or lifting weight but without immediate period of inactivity of the shoulder and without any evidence of fractures or rotator cuff injury .

The Highest incidence (46%) was seen in 40-49 years age group followed by 42% in 50-59 years in our series. Although the typical age group in such condition was reported to be in 5th to 7th decade,[14],a recent study has highlighted that it can occur in younger age group of 40-60 years[10] .

Although females showed an increased incidence of involvement (69%) in our series, which was comparable to other studies[15], the end results were not influenced by the sex of the patients.

In our series, Left sided Adhesive capsulitis was more common (58%). But Laterality of involvement did not influence the end result as maintained by other workers also[16].

Manipulation under Anaesthesia seems to be a logical method of treatment in such condition from patho anatomical point of view. When the arm is by the side of the body there is a redundant fold of capsule and synovium. As the arm is elevated from the side in

abduction and the head of the humerus rotates, the "redundant fold" of the capsule and synovium expand like the bellows of concertina to accommodate the head[17]. In Adhesive capsulitisthere is thickening and contracture of the capsule which become adherent to the head of the humerus. There are also adhesions between the opposed synovial surfaces in the redundant fold. Both these facts were confirmed by Neviaser, during operation in 10 such patients in 1945[6]. This had also been proved by cinearthrography. During manipulation adhesions were torn as demonstrated in post-manipulation cinearthrography[17].

An optimally performed manipulation under anaesthesia, done as a primary treatment of Adhesive capsulitis of the Shoulder had given a quick and effective result in majority of our patients. The better results in infiltration group was due to suppression of post-manipulation pain, so that the patient could actively participate in the physiotherapy regime immediately after manipulation. An intra-articular infiltration of triamcinolone acetonide at the time of manipulation adequately suppresses this post-manipulation pain through its non-specific anti-inflammatory action, thereby assisting the patient in maintaining the free range of movement that had been achieved by manipulation.Although JormaKivimaki et al in a study of 24 patients, found no difference of results in patients with Intra articular Cortisone infiltration than with no infiltration before manipulation under anaesthesia.[18] , majority of studies had shown significant improvement in pain and ROM including ADL in the infiltration group than the non infiltration group[7,13,19].

It was also found to be a safe procedure provided the surgeon could pay careful attention to the details of the manipulative procedure. The manipulating hand was placed at a point as near to the shoulder as possible rather than at the elbow or forearm, so that it helped in getting effective leverage with less possibility of fracturing the humerus. Also fixing the scapula restricted any amount of motion at the scapula and clavicle and allowing pure gleno-humeral motion to occur. Complications occurring during manipulation were reported by various workers like Fracture Proximal Neck Humerus, Dislocation of shoulder, labralTears, Brachial plexus injuries, etc.[10] We did not encounter such complications in our series. Gentleness and meticulously following the sequence of manipulation, could be the reason for absence of complications in our series.

CONCLUSION :

Manipulation under Anaesthesia, provides an effective treatment for Adhesive Capsulitis Shoulder with majority of the patients reporting successful end results, provided the post-manipulation pain is taken care of, by intra articular cortisone infiltration, thereby reducing the period of morbidity.

The risk of manipulation is certainly present, but so far as our experience is concerned, they should not be over-emphasized or used to detract from a very useful procedure. A low complication rate can be expected if the surgeon pays careful attention to the details of proper manipulation.

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Original Article

Urinary Tract Infection In Elderly— A Clinical & Microbiological Study

(A Hospital Based Observational Study)

A K Sen*, R M Doley, P K Dutta, D Das***, R R Gogoi******

Abstract

Introduction: Urinary Tract Infection (UTI) remains the second most common community acquired and main cause for nosocomial infection among elderly. In contrast to the *E. Coli* infection in general population other gram negative bacilli and *Enterococci* often being isolated in elderly patient with UTI.

Material and Methods: Our study included 60 elderly patients with UTI. Along with clinical evaluation, routine examination of urine and bacterial culture and sensitivity were done using MacConkey's agar media.

Results and Observations: Among 60 patients 41 were female. Increase in frequency was the most common presenting symptom. Fever was the cardinal manifestation. Atypical presentation like mental confusion was present in 8.33% of the cases. *E. Coli* was the commonest isolated organism followed by *Pseudomonas* and *Enterococcus* with varying degree of antimicrobial sensitivity.

Conclusion: A high index of suspicion is necessary for diagnosis of UTI in elderly as they may present with atypical manifestation. In addition to *E. Coli*, *Pseudomonas* and *Enterococcus* infections are common in elderly. Urine examination for microbial susceptibility for antibiotics should be carried out in UTI in elderly patients.

KEY WORDS : *Urinary Tract Infection (UTI), Elderly, Micro-organism, Antimicrobial Sensitivity.*

INTRODUCTION :

Urinary Tract Infection (UTI) remains the second most common community acquired and main cause for nosocomial infection among elderly[1]. Depending on residential status and presence of an underlying disease, the prevalence of UTI in elderly men and women can range from 5% to 35% and 15% to 50% respectively[2]. In contrast to general population where 90% cases *Escherichia coli* is responsible for UTI, *Escherichia coli* accounts for 60% to 70% of cases of UTI in elderly with other Gram negative bacilli and *Enterococci* often being isolated in elderly UTI[3]. UTI is much more common in women than in men due to anatomic and physiological reasons[4]. UTI in elderly may sometimes present with atypical manifestation. The presenting symptoms and the organism causing UTI in elderly is different from young adults. A thorough knowledge of symptoms and etiological agents with their antimicrobial sensitivity is essential for adequate treatment of UTI in elderly. The study was conducted in Assam Medical College & Hospital to know the clinical profile of UTI in elderly and to find out the etiological cause and drug susceptibility.

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MATERIALS AND METHODS :

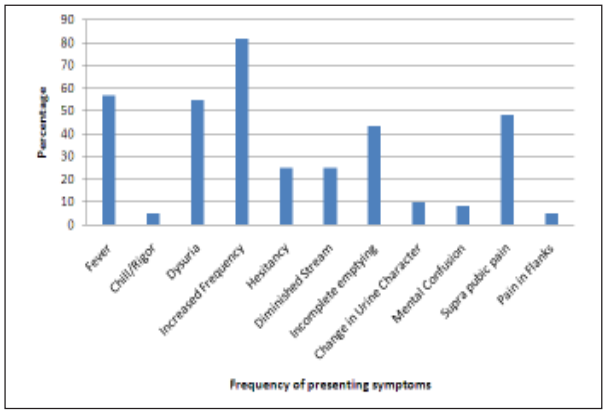
Sixty elderly patients of 60 years and above with symptoms of UTI and examination of urine showing significant pyuria (pus cell ≥5 per HPF under microscopic examination) or / and bacteriuria were included in the study. Clean catch midstream urine was collected (catheter collection and suprapubic collection was done in selected cases) and cultured and bacterial sensitivity tests was done using MacConkey's agar media. Patients with growth of multiple species of bacteria in urine culture or Colony count $<10^3$/ml have been excluded.

RESULTS AND OBSERVATIONS :

A total of 60 elderly patients 60 years of age

and above, with Urinary Tract Infection were included in the study. Out of total 60 elderly patients with UTI, 19 (31.67%) were males and 41 (68.33%) were females. Urinary Tract Infection was more prevalent in 60-69 years age group (50%), followed by 70-79 years age group (41.67%). Increased frequency was most common (81.67%) presenting symptom. Fever is a cardinal manifestation and is present in 56.67% of the cases. Hesitancy and diminished stream was present in 25% of the patients.

Figure 1: Diagram Showing Frequency of Different Symptoms



Atypical presentation like mental confusion was present in 8.33% of the patients.

Different co-morbid conditions were present like Hypertension (75%), Anemia (43.33%), C.O.P.D. (35%), Cerebrovascular disease (11.67%), Cardiac disease (13.33%) and renal disease (5%).

Various risk factors like diabetes mellitus was present in 8.3% of the total no. of case, benign prostatic

Table1: Number of Patients with Different Co-morbid Conditions

Co-morbid Condition	Male	Female	Total	Percentage
Hypertension	13	32	45	75.00%
Cerebrovascular disease	05	02	07	11.67%
Anemia	07	19	26	43.33%
Renal disease	01	02	03	5.00%
Cardiac disease	05	03	08	13.33%
C.O.P.D.	04	17	21	35.00%

hyperplasia and female genital prolapse was present in total 28 (46.66%) cases. Urinary catheter was present in 7 (11.67%) patients.

More than half of the elderly UTI was complicated (56.67%). Next to pyruria, bacteruria was present in

Table 2 : Number of Patients with Various Risk Factors

Risk factor	Sex		Total	Percentage
	M	F		
Diabetes Mellitus	03	02	05	8.33%
Benign Prostatic Hyperplasia	17	00	17	28.33%
Urinary Catheterisation	05	02	07	11.67%
Female Genital Prolapse	00	11	11	18.33%

75.04% of the cases followed by haematuria (8.33%), glycosuria (8.33%) and proteinuria (6.67%).

E.Coli was the most common organism isolated in 53.33% of elderly UTI followed by *Pseudomonas* (16.77%), *Klebsiella* (10%). *Staph. aureas* and *Enterococcus* were the gram +ve bacteria isolated in 8.33% and 11.67% respectively of the UTI in elderly.

All the organisms were sensitive to Imepenum and Pipercillin and Tazobactam. *E.Coli* showed resistance in 18.75% of the cases to 1st and 2nd generation

Table 3 : Showing Various Isolated Organisms from Urine Culture

Sex	E.Coil	Klebsiella	Pseudomonus	Enterococcus	Staph. aureus
Male	06	03	07	01	02
Female	26	03	03	06	03
Total	**32**	**06**	**10**	**07**	**05**
Percentage	53.33	10	16.67	11.67	8.33

cephalosporins. *Klebsiella sp*. showed resistance to fluoroquinolones in 28.75% of the cases, whereas the resistance to 1st and 2nd generation cephalosporins was in 16.67% of the cases.

Pseudomonas were resistant to fluoroquinolones upto 20% of the cases, whereas the resistance to 1st and 2nd generation cephalosporins was in 10% of the cases. *Staphylococcus* were resistant to fluoroquinolones upto 40% of the cases, whereas the resistance to 1st and 2nd generation cephalosporins was in 20% of the cases. *Entercoccus* were resistant to fluoroquinolones upto 42.86% of the cases, whereas the resistance to 1st and 2nd generation cephalosporins was in 14.29 % of the cases.

DISCUSSION :

The study showed increased prevalence of UTI in females than male (2:1). This finding of female preponderance in elderly UTI corroborates with the findings of different studies in India and abroad[5,6,7]. About 5% of elderly patients presented with atypical symptoms

like mental confusion. Thomas *et al*[8] showed up to 30% elderly patients with UTI may present in atypical manner. The study showed 8.3% patients have diabetes mellitus as a risk factor of development of UTI. Other studies like Bahl *et al*[9] and Sathe *et al*[10] showed 30% association with diabetes mellitus. Bori M. *et al*[11] showed both benign prostatic hyperplasia and female genital prolapse results in residual urine which is a risk factor for development of UTI in elderly. These findings are in conformity of our findings of presence of benign prosthetic hyperplasia and obstructive uropathy in 46.66% of the UTIs in elderly. Urinary catheterisation is associated with development of UTI. Catheter associated UTI is the most common among the hospital acquired infections Kothari A *et al*[12] reported incidence of catheter associated UTI in 30%.

Table4: Showing the Antimicrobial Sensitivity Pattern of Isolated Organisms

Isolated Organisms		Antimicrobial agents							
		Norfloxacin	Ciprofloxacin	Cephalaxin	Cefuroxime	Ceftriaxone	Ceftazidin	Piper+ Tazobactum	Imepenum
Escherichia coli	S	28(87.5%)	30(93.75%)	20(62.5%)	28(87.5%)	30(93.75%)	32 (100%)	32(100%)	32(100%)
	R	4(12.5%)	2(6.25%)	12(37.5%)	4(12.5%)	2(6.25%)	0(0%)	0(0%)	0(0%)
Klebsiella sp.	S	4(66.67%)	5(83.33%)	5(83.33%)	5(83.33%)	5(83.33%)	5(83.33%)	5(83.33%)	6(100.00%)
	R	2(33.33%)	1(16.67%)	1(16.67%)	1(16.67%)	1(16.67%)	1(16.67%)	1(16.67%)	0(0%)
Pseudomonas aeruginosa	S	8(80.00%)	9(90.00%)	8(80.00%)	9(90.00%)	9(90.00%)	9(90.00%)	9(90.00%)	10(100%)
	R	2(20.00%)	1(10.00%)	2(20.00%)	1(10.00%)	1(10.00%)	1(10.00%)	1(10.00%)	0(0.00%)
Staphylococcus aureus	S	4(80.00%)	3(60.00%)	4(80.00%)	4(80.00%)	5(100%)	5(100%)	5(100%)	5(100%)
	R	1(20.00%)	2(40.00%)	1(20.00%)	1(20.00%)	0(0.00%)	0(0.00%)	0(0.00%)	0(0.00%)
Enterococcus S	S	4(57.14%)	6(85.71%)	6(85.71%)	6(85.71%)	6(85.71%)	7(100%)	7(100%)	7(100%)
	R	3(42.86%)	1(14.29%)	1(14.29%)	1(14.29%)	1(14.29%)	0(0.00%)	0(0.00%)	0(0.00%)

E.coli was the most common organism grown in 32 (53.33%) numbers of urine samples, *Pseudomonas* was grown in 10 (16.67%) of urine samples, *Enterococcus* grown in 7 (11.67%) of urine samples, *Klebsiella* grown in 6 (10%) of urine samples and *Staphylococcus aureus* was grown in 5 (8.33%) of the urine samples. Though *E. coli* is responsible for 90% of UTI in adult, the incidence is somewhat less in elderly where as *Pseudomonas* and *Klebsiella* infections are more. A Shailini et.al[6] found *E.Coli* in 54.9% in elderly patients with community acquired U.T.I. followed by *Klebseilla* (21.2%) *Pseudomonas* (11.2%), *Enterococcus* (11.2%). Maunir et.al[5] also found *E.Coli* in 31.03%.

Upto 20% of *E.Coli* shows resistance to 1st and 2nd generation of cephalosporins and 12.5% of *E.Coli* shows resistance to 3rd generation of cephalosporins. Upto 20% of *Pseudomonas* are resistance to fluoroquinolones and 1st generation cephalosporins and 10% of *Pseudomonas* shows resistance to 3rd generation cephalosporins. Among *Klebsiella sp.* 28.57% shows resistance to fluoroquinolones and 16.67% of *Klebsiella sp.* shows resistance to cephalosporins.

CONCLUSION :

A high index of suspicion is necessary for the diagnosis of UTI in the elderly as it is one of the commonest infections and it may sometimes present in atypical manner. *E. Coli* was the most common causative organism followed by *Pseudomonas* and *Enterococcus*. All the organisms showed varying degrees of resistance to fluoroquinolones and cephalosporins. Hence urine examination for microbial susceptibility to antibiotics should be carried out in UTI in elderly patients.

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Original Article

Clinico-epidemiological study of Japanese Encephalitis in Tripura

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Abstract

Introduction: JEV is a arthropod borne virus common in Asia-pacific region. This disease affect many rice growing states of India.It is the first time we have recorded 15 new cases of JEV from Tripura. Though there is no sex preponderance in JEV patients in Tripura, but, disease was found in higher age groups. Case fatality rate was 13.13% and neurological sequel was found in 2 persons. The state may be affected by JEV extremely in future and needs epidemiological follow-up.

Materials and Methods: This is a single centre prospective study among the patients presenting with AES at the Medicine and Pediatric wards of Agartala Govt. Medical College and GBP hospital during the Period of 1st June,2013 to 31st August,2013 and confirmation done by MAC-ELISA Test from both serum and CSF or only serum sample of patient.

Results and Observations: There were a total of fifty-five (55) Acute Encephalitis like Syndrome (AES) out of which fifteen (15) had JE confirmed by serology. Symptomatically fever (100%) and headache(100%) were the two most common symptom of patients ; followed by altered sensorium (80.66%) and vomiting (60%). Incidence pattern of JE showed that , confirmation of the cases were observed first-time during the month of June (5 cases) and peak incidence occurred in the month of July(10cases). Out of the total fifteen patients, two died during study period and the case fatality rate being 13.13%. Two patients developed CNS sequel.

Conclusion: Japanese Encephalitis is a serious public health problem with significant mortality . Early diagnosis and treatment can reduce complications. Future epidemiological study can ensure the need of vaccination in a endemic area for proper prevention of infection in Tripura.

KEY WORDS: *Japanese Encephalitis Virus (JEV), Arthropod borne disease, AES(Acute encephalitis syndrome).*

INTRODUCTION :

Japanese encephalitis virus (JEV), arthropod borne pathogen infections can be found throughout the temperate and tropical zones of Asia. In endemic areas annual incidence ranges from 10-100 per 100000 populations[1]. The virus was isolated for the first time in the world from a post mortem human brain in Japan in 1933AD, although descriptive accounts of the disease date back to late 1800AD. In 1954, it was shown that virus could also infect pigs, bovines, dogs and sheep. Basically, JE is a zoonotic disease maintaining JEV in nature by bird- mosquito- bird and pig- mosquito- pig cycles . Pigs are amplifiers of JEV. Bats can also carry the virus for longer period of time. Human beings are only incidental hosts forming a dead end[2]. A growing number of cases of Japanese Encephalitis have been seen in horses in China and humans in India, Nepal, Philippines, Sri Lanka, Northern Thailand, Vietnam, and Myanmar[2]. JEV is the most common documented cause of viral encephalitis in Cambodia. Rise in population density, deforestation and increasing irrigation of agricultural areas may contribute to the rise in JE incidence each year. JEV infections were occasionally found in Indonesia and Northern Australia but never found in United States[3]. In recent years, JE is rare in Japan due to JE virus vaccination, use of agricultural pesticides and controlled pig farming. The occurrence of JE has also been reported less in Korea, China and Taiwan due to JE virus vaccination of children. Japanese Encephalitis (JE) known "a plague of the Orient" continues to be the most common human epidemic encephalitis in the world[4].

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About one third of the patients die and half of the survivors have residual sequel . The major burden of this disease is in children . Diagnosis depends on a high degree of clinical suspicion and confirmation by serology or culture, cerebrospinal fluid (CSF) analysis, computered tomography and magnetic resonance imaging are also important . In India JEV is prevalent in Chennai, Andhrapradesh, Maharashthra,Goa, Uttarpradesh,Bihar,West Bengal,Assam,Karnataka and Meghalaya[1].There was no record of JEV infection in Tripura until now. In June, 2013, we have first diagnosed fever cases of JEV in Tripura and fifteen(15) recorded cases has been followed up during this period, 1st June,2013 to 31st August,2013. In this study, the clinico-epidemiological features of JEV has been evaluated.

AIMS OF THE STUDY :

This Hospital based prospective study is designed to evaluate the clinico-epidemiological features of JEV in Tripura.

MATERIALS AND METHODS :

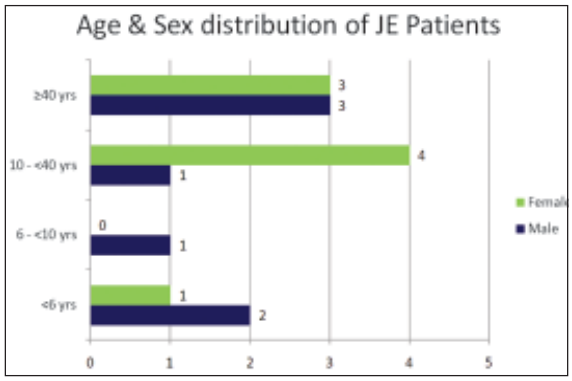
This is a single centre prospective study among the patients presenting with AES at the Medicine and Pediatric wards of Agartala Govt. Medical College and GBP hospital during the Period of 1st June,2013 to 31st August,2013 and confirmation done by MAC-ELISA Test from both serum and CSF or only serum sample of patient.

Inclusion criteria: All patients irrespective of their age presented to the Department of Medicine and Paediatrics with the symptoms of AES (Meningitis, Meningoencephalitis and encephalitis) were included in the study.

RESULTS :

There were a total of fifty-five (55) Acute Encephalitis like Syndrome (AES) out of which fifteen (15) had JE confirmed by serology . Even though a female preponderance was noted in the 10 to <40 years of life, it remained equal in the age groups ≥40 years. Average age of the AES patients was 39.16 years, out of them, 15 patients was diagnosed to have JEV(27.27%).The average age of JEV patient were 44.24 years.

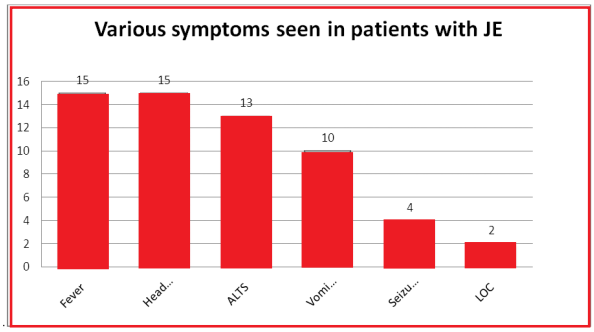
Fig.1: Showing Age and Sex distribution of JE patients.



Symptomatically fever (100%) and headache (100%) were the two most common symptom of patients ; followed by altered sensorium (80.66%) and vomiting (60%). Other symptoms such as loss of consciousness (10.33%) and seizures (20.66%) were also noted. Incidence pattern of JE showed that, confirmation of the cases were observed first-time during the month of June (5 cases) and peak incidence occurred in the month of July (10 cases). No cases reported during August.

Out of the total fifteen patients, two died during study period and the case fatality rate being 13.13%. Two patients developed CNS sequel.

Fig 2: Showing the various symptoms seen in patients with JEV



DISCUSSION :

Tripura is a virgin land for JEV and no case was reported earlier either in children or adult. JE is usually more common among children (2-6 years)[1]. But in our study (in Tripura), JEV infection is more on adults(70.33%) than pediatric age group which signifies new onset of the disease. This epidemiological Shift is of great importance to evaluate. Japanese encephalitis might be a significant public health problem in Tripura in future. JE case is considered to have elevated

temperature over 38.0C along with altered consciousness and is generally confirmed serologically by findings of specific anti JE Ig M in the cerebrospinal fluid[2].

The disease presents with a prodromal stage, an acute encephalitic stage with coma, convulsions and variable deficits and a convalescent stage[2]. The disease primarily affects children between the ages of one and fifteen year's. Of those who contract the disease; approximately, seventy percent either die or are left with a long term neurological disability[5]. JE vaccines existed for a long time, however due to cost and unstable supply; they have not been able to meet the needs of developing country health systems. In view of the high mortality and severe sequel which often leaves behind highly dependent and disabled survivors, the diseases is assuming great importance.

Japanese Encephalitis is demonstrated to be a significant public health problem throughout Asia[4] .The geographical distribution of JE has expanded over the years to include all of Southeast Asia and most of the Indian subcontinent. Epidemics of JE have spread across all of southeast Asia and were reported for the first time in Tripura in 2013, probably as a result of steady rise in population density, deforestation and increasing irrigation of agricultural areas and close proximity of natural and amplifying hosts[5].

This Study showed 50.33% male patients in comparison to 49.67% females. A. Rayamajhi in his study reported 69 % male and 31 % females. Study from South India also showed male to be effected in 58% cases and female sex in 42% case. 20.66% patients in this study were under 10 years of age[7]. Raghava Potula in his study found 71.2% patients less than ten years[7]. A. Rayemajhi et al in their study found 58.4 % patients below nine years of age[8]. Neeru Gupta from North India found 31.25% cases less than five years of age, more than 4/5th of the cases (84%) were between one to twelve years of age[8],wereas only 30% of our patients are less than 12 years of age. This epidemiological shifting is a matter of great concern in our study.

Our study showed that fever and Headache were the two most common symptoms of JE and was found in 100 % cases, the other symptoms seen in this disease were altered sensorium (80.66%), vomiting (60%), seizure (20.66%), and unconsciousness (10.33%). Similar clinical features were reported by other authors.[5,8].

Present study showed most of the cases appeared soon after Monsoon and premonsoon, i.e. during the month of June and July . 100% cases were seen during these two months .No cases reported during the month of August. Study done by Bista B.M showed, upsurge of cases take place after the rainy season (monsoon)[1,9]. They reported that, cases start to appear in the month of April-May and reach its peak during late August to early September and start to decline from October.

During the three months of study, two patients died, hence the mortality rate being 13.13%. Study done at B. P. Koirala Institute of Health Sciences showed 8.3 % mortality rate and report from Indonesia showed, case fatality rate of 10 % . Similarly Akiba T and Kumar R in their studies found case fatality rate of 13.2% and 20-50% respectively[2,5,6] . All these findings are similar to our findings. Besides these, our results are consistent with previous studies done in India and in North-East.

CONCLUSIONS :

Japanese Encephalitis is a serious public health problem with significant mortality. Early diagnosis and treatment can reduce complications. Future epidemiological study can ensure the need of vaccination in a endemic area for proper prevention of infection in Tripura.

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Original Article

Panaromic radiograph : A cost effective tool for early detection of calcified carotid artery atheromas in type 2 diabetic patients

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Abstract

Background: With increasing prevalence of type 2 diabetes, the chances of accelerated carotid artery atherosclerosis and risk of stroke are also increasing. Therefore, a cost-effective tool for screening of atheroma is needed in countries like India where the majority of people are from below poverty line. This study is undertaken to assess the usefulness of panoramic radiograph in the diagnosis of calcified carotid artery atheromas.

Methods and Materials: The panoramic radiographs of 50 diabetic patients (age range, 50 to 80 years; mean age 64.04 years) and 50 controls (age range, 50 to 70 years; mean age 55 years) at outpatient department of Mamata Dental Hospital were evaluated for calcified atheromas. To confirm the calcification and to check the reliability of panoramic radiographs, all the study participants were subjected to 2D colour Doppler and spectral Doppler. The participants were also subjected to estimation of lipid profile to assess the risk. Statistical comparison of the atheroma prevalence rates was done by means of the Fisher exact test or t-test.

Results: The radiographs of the diabetics revealed that 18% had atheromas whereas those of the control demonstrated that 2% had atheromas (a statistically significant difference; p= <0.002). In all these patients, 2D colour Doppler and spectral Doppler confirmed the presence of carotid artery atheromas. The calcification was seen as radio-opacities in the regions 1.5 to 2.5 cms inferio-posterior and lateral to the angle of the mandible on the panoramic radiographs.

Conclusion: Panoramic radiograph for identification of calcified carotid artery atheroma is a sensitive and cost-effective tool in patients with type 2 diabetes.

KEY WORDS : *panoramic radiographs calcified carotid artery, carotid atheromas, atherosclerosis, type 2 diabetes.*

INTRODUCTION :

Type 2 diabetes is a metabolic disorder characterized by dysregulation of carbohydrates, protein and lipid metabolism. It arises from a combination of resistance to insulin's action and insufficient secretion of additonal insulin to compensate for this resistance. The resultant hyperglycemia and associated other risk factors cause premature atherosclerosis of vessels leading to stroke, myocardial infarction and peripheral vascular disease. Approximately 382 million people have type 2 diabetes in the world in 2013.[1] Its prevalence increases with age; nearly one in five people in India older than 65 years of age have the disorder. Previous studies have demonstrated that patients with type 2 diabetes had a high prevalence of calcified carotid artery atheromas visible on panoramic radiographs.[2,3,4] Studies have demonstrated that about 80% of strokes are induced by atherosclerotic plaques, mostly detected in the main carotid artery bifurcates (Bifurcation), which has the highest tensile force.[5,6]

Even though ultrasonography is the gold standard imaging modality in detecting the calcified carotid artery atheromas it is not economical and needs skilled personnels.[7] Therefore there is a need for more economical screening tool for detection of calcified carotid artery atheromas in countries like India where majority of people belong to low socioeconomic group. Panoramic radiograph is simple, economical and easy to interpret. In 1981, Friedlander and Lande et al described the presence of carotid artery calcifications (CAC's) on panaromic radiographs and suggested that these radiographs play an important role in the early diagnosis of CAC's. Therefore panoramic radiographs may help

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us as a screening tool for detection of CAC's.[8,9,10] However, the uses of panoramic radiograph is confined to the field of dentistry only. Therefore we undertook this study to determine the effectiveness of panoramic radiograph in detection of calcified carotid artery atheromas in type 2 diabetic patients.

METHODS AND MATERIALS :

It is a cross-sectional prospective study done in tertiary care hospital (Mamata General Hospital and Mamata Dental Hospital). Both the hospitals are located in the same campus and mainly based on rural and tribal population in and around Khammam. The study population consisted of all diabetic patients attending the Out Patient Department of Mamata Dental Hospital during the study period from March 2013 to August 2013.

The study was approved by the institutional ethics committee and informed consent was obtained from all the study subjects. The following inclusion criteria were used for patient selection:

- A diagnosis of type 2 diabetes mellitus was made if the patient was previously diagnosed as having diabetes and at some time in their disease, other than a time consistent with the "honey moon period", was managed with diet and exercise alone or with oral hypoglycemic agents or based on an initial fasting plasma glucose level of > 125 milligrams/deciliter or higher and or plasma glucose level of 200 mg /dl at two hours after oral glucose tolerance test[1].
- An age of 50 years or older.
- A conventional panoramic radiograph visualizing the area 2.5 centimeters posterior and 2.5 cm inferior to the cortical rim of the midpoint of the mandibular angle.

Exclusion criteria:

- Advanced cerebrovascular disease as evidenced by transient ischemic attack or cerebrovascular accident.
- Advance cardiovascular disease as evidenced by myocardial infarction.
- Any patient who's radiograph is of poor quality (incorrect patient positioning, over exposure or under exposure and processing errors.

The subjects of the control group were from consecutively treated outpatients attending the Mamata Dental Hospital. Inclusion criteria for the control group were:

- Age match (±12 months) to the study population.
- A satisfactory panoramic radiograph.

Exclusion criteria for the control group were:

- A history of diabetes mellitus.
- A history of transient ischemic attack.
- A history of cerebrovascular accident.

All patients were radiographed using a standard dental panoramic radiographic system (sirona orthophos XG 5 panoramic x-ray system). The unit is operated at 10mA, the peak kilo voltage ranging from 60-85 depending on the subject jaw size. The radiographs were examined in ambient light through the use of transmitted light from a standard viewing box for the presence of a calcified carotid artery atheroma. Calcifications which appear as a single or multiple discrete radiopaque nodular masses within the soft tissues of the neck, 1.5cm inferior and 2.5 cm posterior to the cervical rim of the midpoint of the mandibular angle were considered as consistent with the diagnosis.

DATA COLLECTION :

All the subjects (study and control groups) were subjected to detail history, physical examination and panoramic radiograph. The following data were recorded.

- Presence of hypertension requiring medication for control.
- Height.
- Weight.
- Body mass index.
- HbA_1C.
- Total serum cholesterol level.
- Serum low density lipoprotein level (LDL).
- Serum triglyceride level.
- Serum high density lipoprotein (HDL)

The radiographs obtained were examined in ambient light through the use of transmitted light from a standard viewing box for detecting the presence of carotid artery calcifications. The calcifications seen on the radiographs were noted. To confirm these calcifications and to check the reliability of panoramic radiographs in detecting the carotid artery calcifications the patients were subjected to soft tissue ultrasonography, along with 2D color Doppler and spectral Doppler. The ultrasonography was done by an expert radiologist who was unaware about the results of panoramic radiographs. The

ultrasonographic unit used was HDI 1500 Phillips color Doppler with facilities of 2D and duplex Doppler studies. The probe used for the investigation was linear probe with 12-5 MHz frequency, 3 to 5cm depth and 85 grain. The probe was run along the right and left lateral side of the neck and along the common carotid artery. The images were recorded and used for duplex Doppler studies. Color Doppler study was done to check the flow of blood in the carotid arteries and pattern of blood flow in the areas of calcifications. Spectral Doppler studies were done to check the flow pattern in the carotid arteries and the peak systolic volume (PSV), end diastolic volume (EDV), and mean velocity (Vm) were recorded. Age matched controls, free of diabetes were assessed in the similar manner.

Two ml blood was drawn from all the patients for estimation of total serum cholesterol level, serum low density lipoprotein level, and serum triglyceride level. Data analysis was carried out by comparing the propositions of diabetic and control groups through the use of the fisher's exact test. These groups were also compared with respect to each of the following atherosclerosis associated risk factors through the use of t- test:

- HbA_1C.
- Total serum cholesterol.
- Serum LDL
- Serum triglyceride levels.
- Prevalence of hypertension.

RESULTS :

Out of the total 163 diabetic patients, 63 (age < 50 years, n=56; Panoramic radiographs with inadequate positioning, n=7) were excluded as they were not fulfilling the inclusion criteria.

They ranged in age from 50 years to 80 years with a mean age of 64.04 years. The control group consisted of 50 age-matched persons free of systemic disease, with a mean age of 58.44 years (range 50–70 years).

Panoramic radiographs manifesting calcified carotid artery disease were seen in 9 (18%) patients in the study group and in 1(2%) of the control group patients (Table 1). The proportion of patients with calcified carotid artery disease were significantly higher in the study group, as measured by Fisher exact test

Table 1 : Comparison of patients with and without atheromas

Characteristic	Diabetes with atheromas	Diabetes without atheromas
Age in years (mean ±SD)	66.56 ± 4.98	63.48 ± 7.37
Male Sex (%)	44 %	62 %
Prevalence of hypertension (%)	55%	37.5%
Body mass index (mean ± SD)	24.07 ± 4.06	23.88 ± 3.45
HbA_1c level (mean ± SD)	9.25 ± 0.7	9.24 ± 1.4
Total serum cholesterol	186.11 ± 37.45	197.87 ± 33.43
Serum LDL	118.77 ± 34.30	136.41 ± 27.97
Serum triglyceride	173.55 ± 48.03	179.85 ± 56.10
Serum HDL	44.33 ± 10.34	48.53 ± 9.81

(p=<0.002). The diabetics with atheromas on their radiographs had a mean age of 66.5 years (range 60 to 70 years). 6 of the diabetics had unilateral opacities and 3 had bilateral opacities. These patients had an average HbA_1C of 9.25, total average serum cholesterol level of 186.1mg/dl, the average serum LDL level of118.7mg/dl, the average serum HDL levels of 44.3mg/dl and the average serum triglyceride, levels of 173.5mg/dl. Patients had hypertension and required medications for its management.

In the study group 37 patients were treated for their diabetes by oral hypoglycemic agents, 13 patients were receiving both oral hypoglycemic agents and insulin.

One member of the control group had atheroma formation on radiograph. The diabetic study group and the control group were compared with respect to risk factors associated with atherosclerosis by means of the Fisher's exact test or t-test (Table 2).

Table 2: Baseline characteristics of study group & control group

Characteristic	Diabetic group	Control group
Age in years (mean ±SD)	64.04 ± 7.06	58.44 ± 5.91
Male Sex (%)	58%	52%
Prevalence of hypertension (%)	32%	29%
Body mass index (mean ± SD)	23.9 ± 3.5	21.7 ± 3.02
HbA_1c level (mean ± SD)	9.24 ± 1.32	3.8 ± 0.98
Total serum cholesterol	195.76 ± 34.09	159.48 ± 18.94
Serum LDL	133.24 ± 29.63	139.52 ± 11.33
Serum triglyceride	178.72 ± 54.33	134.14 ± 10.46
Serum HDL	47.78 ± 9.94	51.18 ± 9.9

The 2D Colour Doppler and Spectral Doppler done in both the groups (study and control) and confirmed the presence of calcification.

DISCUSSION :

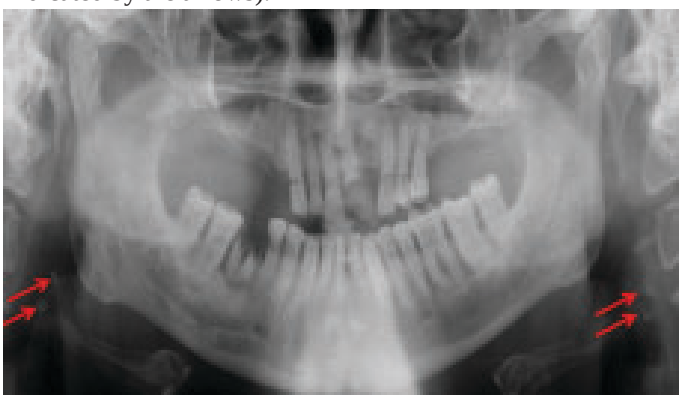
Macrovascular atherosclerosis is a frequent cause of morbidity in patients with type 2 diabetes mellitus.[8, 10, 11] It tends to accumulate within the bifurcation of the common, external and internal carotid arteries, and it

produces symptoms as plaque forms and embolises to the brain[12]. Stroke caused by atherosclerosis at the bifurcation of common carotid artery are two to four times more likely to occur in people with diabetics than in non diabetics. Preventive measures must be taken to diminish its incidence. Changes in the lifestyles, including adequate diet and eating habits, practicing physical activity and a less stressful life are important strategies in the primary prevention of this pathology.

The panoramic radiographs of patients with type II diabetes mellitus showed higher prevalence of calcified atherosclerotic lesions of the carotid artery (18%) than did those of matched control patients without diabetes (2%). The difference in the prevalence rates of atheroma formation between these groups may have occurred because of significantly greater burden among the diabetic group of risk factors (elevated levels of plasma glucose, serum LDLs, and serum triglycerides) known to be associated with the development of atherosclerosis. Panoramic radiographic examinations performed in diabetes have revealed the prevalence of carotid artery calcifications in 24% of the patients treated without insulin and in 36% of diabetics treated with insulin.

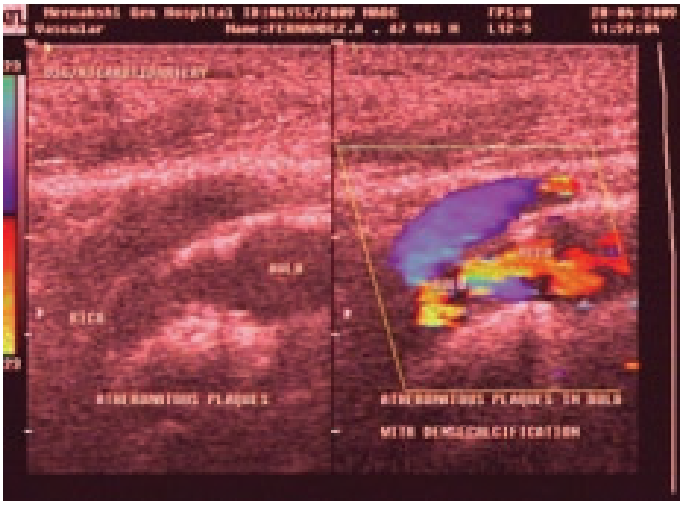
The results of our study demonstrated that accelerated macrovascular atherosclerosis is more common in diabetic group when compared to non diabetic group. In our study nine patients of diabetic group (18%), showed carotid artery calcifications on the panoramic radiographs (P= <0.002). These calcifications were seen as radiopacities in the regions 1.5 to 2.5cm inferio-posterior and lateral to the angle of the mandible on panoramic radiographs (figure 1). The mean age of the patients which showed the calcifications was 66.5 years and the male to female ratio was 5:4. Out of all the cases which showed carotid artery calcifications four cases had hypertension. Both unilateral and bilateral calcifications were detected (6 cases of unilateral calcifications with P value 0.0256 and 3 cases of bilateral calcifications with P value 0.2405).

Figure 1 : OPG shows calcified carotid atheromas (position indicated by the arrows).



All the nine cases of carotid artery calcifications were subjected to duplex ultrasonography (2D Color Doppler and Spectral Doppler), to confirm the carotid artery calcifications and there by checking the reliability of panoramic radiographs. All the nine cases of carotid artery calcifications were detected on duplex ultrasonography and were reconfirmed as the calcifications of carotid artery (figure 2). In our study

Figure 2 : Ultrasound colour doppler showing atheromatous plaque in right carotid artery and bulb.



Duplex ultrasonography demonstrated that most of the calcifications are seen in the bulb area of carotid artery and internal carotid artery. Out of nine cases three cases showed calcifications in carotid bulb area and other three cases in internal carotid artery. Three cases showed calcifications in both carotid bulb and internal carotid artery. Atherosclerotic lesions in the carotid bulb region are a known cause for cerebrovascular accidents. Our study demonstrated that left carotid artery is more commonly affected than right carotid artery. All the positive cases of calcifications were later referred back to general hospital for medical management of diabetes and prevention of stroke.

The result of our study demonstrate that the prevalence of calcified carotid artery atheromas is significantly higher in diabetic patients that the non

diabetics in our population also and can be detected by means of conventional panoramic radiography. Considering the increased morbidity and mortality associated with diabetes, early referral of these patients or identification of an atheroma on the panoramic radiograph may lead to early diagnosis and intervention, and thereby reducing the incidence of fatal and non fatal ischemic stroke.

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Original Article

Seroprevalence and Epidemiological trends of Dengue in Gauhati Medical College & Hospital

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ABSTRACT :

Introduction: Dengue virus infection has emerged as a notable public health problem in recent decades in terms of the mortality and morbidity associated with it [1] .There are currently no licensed vaccines or specific therapeutics, and substantial vector control efforts have not stopped its rapid emergence and global spread[2]. The first dengue epidemic in India occurred in Kolkata during 1963- 1964. Since then the epidemiology of den-gue virus has continued to change [3]. Dengue infection is endemic in both urban and semiurban India.

Materials and Methods: Patient details were collected in prescribed formats and the samples were processed in the Department of Microbiology, Gauhati Medical College & Hospital over a period of two years. NS1Ag ELISA and Dengue IgM MacELISA were done as per the manufacturer's guideline.

Results and Observations : The positivity rate of dengue case was found to be 47% in 2012 and 30% in 2013. Males were more affected than the female population and the commonest age group affected was the young adults. Dengue transmission was found to occur in post-monsoon season between August to December. in 2013, there was early peaking and sustenance of the dengue cases than the previous year. It was observed that the maximum numbers of dengue cases in both the years were found to be from Kamrup (metro) district, followed by Kamrup (rural). This study has provided important insights into the seroprevalence and epidemiological trends of dengue in Assam.

Conclusion: Dengue was hitherto an unknown entity in Assam till a few years back .The experience of dengue was limited as the cases which were diagnosed and treated were from outside the state; however recently it has been noted that the dengue cases were rapidly increasing in the last few years as evidence from the present study. The study also shows male preponderance of the disease and the seasonal variation shows early peaking and sustenance than the previous year. It also brings to light the spread of dengue to newer districts.

KEYWORDS : *Dengue, Seroprevalence, NS1Ag, IgM Mac Elisa*

INTRODUCTION :

Dengue virus infection has emerged as a notable public health problem in recent decades in terms of the mortality and morbidity associated with it [1] .There are currently no licensed vaccines or specific therapeutics, and substantial vector control efforts have not stopped its rapid emergence and global spread[2]. The first dengue epidemic in India occurred in Kolkata during 1963-1964. Since then the epidemiology of den-gue virus has continued to change [3]. Dengue infection is endemic in both urban and semiurban India. Dengue outbreaks have been reported from many states in India, recently these epidemics have become more frequent. In the Northeast India, Dengue activity was first detected in the rural environment of Lohit district of Arunachal Pradesh and Darrang district of Assam[4]. Den-gue outbreaks have since then been reported from newer areas of Assam as documented in various studies[5,6]. The case fatality rate in patients with dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS) can be as high as 44% [7].Hence early and rapid laboratory diagnosis of dengue is crucial. Appropriate clinical management can save the lives of DHF and DSS patients and mortality can be reduced to less than 1%[8]. It is also worthwhile for planning appropriate control strategies.

Diagnosis of recent dengue infection may be achieved by detection of the virus in the patient's blood, either by virus iso-lation in a susceptible cell culture or by identifying the viral RNA by PCR. The development of nested reverse transcription-PCR (RT-PCR) and realtime RT-PCR have significantly re-duced processing

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times; however, these procedures are expensive and technically exacting and laboratory contamination can yield false-positive results**.** As a result, dengue culture and PCR have limited utility in routine clinical use. Serological diagnosis of dengue infection via detection of immunoglobulin M (IgM) by capture ELISA has many advantages: ease of performance in testing, sensitiv-ity in detecting acute phase antibodies and non-requirement of sophisticated equipment [9]. IgM detection by capture ELISA (MAC ELISA) is the most widely used test for detecting dengue infection. However, time to IgM production varies considerably among patients. Some patients have detectable IgM by the third day of symptoms, others do not develop detectable IgM until the eighth day of symptoms. The sensitivity of this test is inadequate before the fifth day of symptoms[10]**.** Because of the complications associated with dengue infection, it would be beneficial to have a diagnosis early in the illness. Commercial assays based on a non-structural dengue NS1 protein in serum have become available. NS1 is a highly conserved glycoprotein, which appears essential for virus viability. During infection, NS1 is found associated with intracellular organelles or is alternatively transported through a secretory pathway to the cell surface. A soluble hexameric form is released in a glycosylation-dependent fashion from infected mammalian cells **[11].** NS1 protein is found circulating in high concentrations in human serum during the acute phase of the disease. Detection of dengue NS1 antigen by ELISA allows detection of infection prior to serocon-version. NS1 antigen can be detected in patient serum 1-9 days after the onset of fever **[11]**. Early diagnosis of dengue allows earlier monitoring, possibly reducing the risk for DHF/DSS.

AIM AND OBJECTIVE :

To analyse the seroprevalence and epidemiological trend of dengue with clinical suspicion among the patients in the year 2012 and 2013 in Assam.

MATERIALS AND METHODS :

Blood samples were collected and processed in the Department of Microbiology, Gauhati Medical College & Hospital. This centre is one of the dengue sentinel surveillance sites in the country under the National Vector Borne Disease Control Program (NVBDCP) of India. Patients attending the various departments of GMCH and those referred from different hospitals and laboratories, with clinical suspicion of dengue were screened over a period of two year from January 2012 to December 2013. The patients presenting with fever of sudden onset with headache, retrobulbar pain, conjunctival injection, pain in back and limbs, lymphadenopathy and maculopapular rash, haemorrhagic manifestations, who had recent travel history and blood picture suggestive of thrombocytopenia were included. Patient demographic details were collected in the prescribed formats.

All the samples of the patients with a history of fever of less than 5 days were tested by NS1Ag ELISA employing Standard Diagnostic Inc kits and the patients with a history of fever beyond 5 days were tested by Dengue IgM MacELISA which was provided by National Institute of Virology, Pune.The tests were performed as per the manufacturer's guideline. The serum samples were stored at 4^0 -8^0 C, but when longer duration of storage was required, it was stored at -20^0 C. The results were recorded properly and sent to the patients and the concerned authorities.

The test results and other epidemiological variables were put in a performa for analysis of the results.

RESULTS AND OBSERVATION :

Out of a total number of 3117 samples tested, 1460 (46.84%) were seropositive for dengue infection in the year 2012,. NS1Ag ELISA was done in 2100 cases, of which 1180 (56.19%)was found to be positive while Dengue IgM MacELISA was done in 1017 cases, of which 280 (27.53%) were found to be positive.
In the year 2013, among 9521 samples tested, 2925 (30%) were found to be positive for dengue infection. NS1Ag ELISA was done in 7553 cases, of which 2510

Table 1 : No. of Dengue ELISA done in the year 2012 & 2013

	2012	2013
No. of sero positive cases / Total ELISA	1460 / 3117 (47%)	2925 / 9521 (30%)
NS1Ag ELISA positive / Total NS1Ag ELISA	1180 / 2100 (56%)	2510 / 7553 (33%)
IgM MAC ELISA positive / Total IgM MAC ELISA	280 / 1017 (28%)	415 / 1968 (21%)

(33%) was positive and Dengue IgM MacELISA was found to be positive among 415 (21%)cases out of a total of 1968 cases for dengue infection.(Table 1)

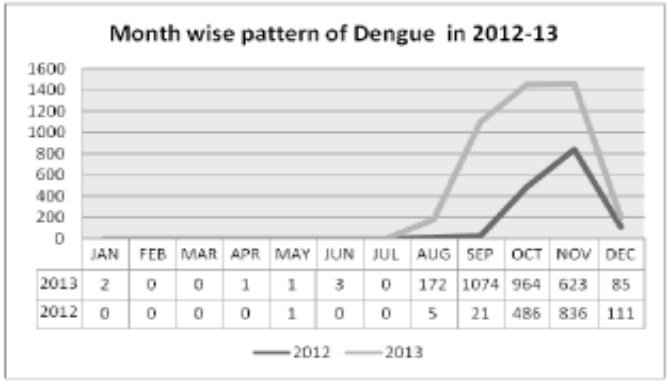
In both the years males patients numbered more than the female patients and the commonest age group affected was between 25 to 34 years followed by 15 to 24 years of age. (Table 2).

Table 2: Age wise distribution of positive Dengue cases from GMCH in 2012&2013

Age Group	2012		2013	
	Male	Female	Male	Female
0-5 yrs	5	2	14	11
6-14 yrs	37	26	120	70
15-24 yrs	182	83	543	265
25- 34 yrs	239	78	631	284
35-44 yrs	144	55	322	189
45-54 yrs	64	27	160	119
55 yrs and above	45	13	129	67

The month-wise distribution of positive cases in the year 2012 were between July to December and maximum cases was found in the month of November. Whereas in 2013, there was early peaking and sustenance of the dengue cases than the previous year. Maximum cases was found in the month of November (Fig-1).

Fig 1: Month wise pattern of Dengue positive cases in 2012 & 2013



When district wise incidence pattern was studied, it was observed in both the years that the maximum number of dengue cases were found to be from Kamrup (metro) district, followed by Kamrup (rural) followed by Nalbari, Lakhimpur,Barpeta. (Table 3.) In the Guwahati city, positive cases were residents along the banks of the Bharalu river. The worst hit areas were Basistha-Beltola-Bhetapara pocket and the Fatasil-Dhirenpara-Jyotikuchi-Lakhara-Garchuk region.

Table 3: District wise distribution of Dengue cases in 2013.

Districts	Dengue cases	
	2012	2013
Baksa	18	20
Barpeta	16	29
Bongaigaon	16	7
Cachar	4	2
Chirang	6	6
Darrang	18	33
Dhemaji		9
Dhubri	10	14
Dibrugarh	2	5
Goalpara	10	3
Golaghat	4	3
Jorhat	4	3
Kokrajhar	N/A	5
Karimganj	4	2
Kamrup (M)	1098	2485
Kamrup (R)	161	121
Karbi Anglong	2	2
Lakhimpur	33	10
Morigaon	3	10
Nagaon	12	20
Nalbari	34	78
Sibsagar	4	3
Sonitpur	4	6
Udalguri	7	7
Cases from Other States	N/A	9

DISCUSSION :

Dengue is one of the major re-emerging viral infections and rapid urbanization and transportation facilities have aggravated the rapid spread to newer areas in Assam. During 1992, dengue virus antibody was detected from Dibrugarh, North Lakhimpur, Dhemaji and Golaghat districts of Assam.After a gap of 12 years, during 2007-2008, dengue cases were reported from Dibrugarh, Sibasagar, Jorhat, Lakhimpur, Tinsukia, Sonitpur and Dhemaji [12]. A comprehensive entomological survey conducted during 2004–2005 in the North eastern (NE) region of India revealed that the region is rich in known dengue vectors, viz. *Aedes* (Stegomyia) *aegypti* and *Aedes* (Stegomyia) *albopictus*[6]. Therefore Assam has experienced an increased number of reported fever cases of unknown origin in recent years.

The study revealed that the sero-positivity of dengue cases was 47%in 2012, and 30% in 2013. It was observed that the number of samples has increase in

2013 as the awareness among the healthcare workers and the physicians has increased and the cases of fever other than dengue has been subjected to testing. Although the NS1Ag Elisa was done in majority of the cases than the IgM Mac Elisa for dengue in both the years as most of the cases came to the hospital during the first 5 days of febrile condition during the post monsoon season. NS1 antigen test is a potentially useful test during early febrile stage. An outstanding point of the test is its high specificity during early infection. In primary infections, immunoglobulin M (IgM) is detected 5 or more days after the onset of illness in the majority of infected individuals .In the present study, it was observed that the maximum cases were in the young adult age group (≥15 years of age) in both the years,similar observations were also found in other studies[13,14].Gender distribution shows a male preponderance in our study . This trend is similar in most dengue endemic countries in Southeast Asia and this finding is in concordance with that of an earlier studies.[13,15,16]. Low incidence among women may occur because they stay at home and are less exposed to infection. The seasonality of dengue cases pertaining to the highest proportion of seropositivity was observed during post monsoon season (September-December). Trends show that there is early peaking of the dengue cases which is more sustained in 2013 than the previous year. The number of cases of dengue fever may continue to swell because of the existing conducive environment for the life cycle of '*Aedes aegypti*[6].

The presence of Dengue virus in Assam is documented in various studies [5,6] from upper Assam only. But the recent outbreak showed the virus has spread to newer districts of Assam. When district wise incidence pattern was studied, it was observed that the maximum number of dengue positive cases were found to be from Kamrup (metro) district, followed by Kamrup (rural), Nalbari, Lakhimpur. When observed the geo-epidemiological pattern in the Guwahati city, from where maximum number of dengue positive cases were found, it was seen that the most number of positive cases were residents along the banks of the Bharalu river. The worst hit areas were Basistha-Beltola-Bhetapara pocket and the Fatasil-Dhirenpara-Jyotikuchi-Lakhara-Garchuk region. Awareness of changing trends in epidemiology of dengue fever is essential for epidemiological surveillance. Developmental activities, especially urban development associated with rapid growth of new townships, have accentuated the problem of vector- borne diseases specially dengue. Solid waste, which is frequently dumped in urban areas, presents an ideal mosquito breeding sources after accumulation of rainwater. The outbreak in Assam during 2012 and 2013 may be the result of many confounding factors including dengue vector mosquito abundance, their activity and behaviour, weather variables like temperature, humidity, precipitation, human activities and movement of people, viral source and their serotypes. The lack of basic knowledge of the community on dengue epidemiology and vector bionomics could be also a major cause of increasing trend of dengue in this highly populated urban environment.

CONCLUSION :

Dengue was hitherto an unknown entity in Assam till a few years back .The experience of dengue was limited as the cases which were diagnosed and treated were from outside the state; however recently it has been noted that the dengue cases were rapidly increasing in the last few years as evidence from the present study. The study also shows male preponderance of the disease and the seasonal variation shows early peaking and sustenance than the previous year. It also brings to light the spread of dengue to newer districts.

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Review Article

Alcoholic Liver Disease – Who are at risk?

A Chowdhury*, U J Deka**

KEY WORDS : *Alcohol, Alcoholic liver disease, Steatosis, Steatohepatitis, Liver cirrhosis,*

Alcohol consumption is a major contributor to the burden of disease in India and the developing world, it is indeed an increasing major public health concern[1,2].

The World Health Organization (WHO) estimates that there are about 2 billion people worldwide who consume alcoholic beverages and 76.3 million with diagnosable alcohol use disorders. The global burden related to alcohol consumption, both in terms of morbidity and mortality, is considerable and increasing in most parts of the world.

Alcohol consumption is also becoming the leading risk factor for disease burden in developing countries.

Within the drinking patterns section four indicators were chosen for the country profiles.

- Rates of abstainers in the population, i.e. people who have not consumed any alcohol in the last 12 months (if other definition of abstainer it has been separately noted).
- Problem drinkers, heavy drinkers or high risk drinkers, as defined in the corresponding source, people drinking regularly at a level where there is a high risk of chronic or acute consequences.
- Heavy episodic or binge drinkers, as defined in the corresponding source, people drinking occasionally at a level where there is a high risk of intoxication and acute consequences.
- Rates of alcohol dependence, either in the general population or some sub-population using some internationally validated instruments such as AUDIT and CAGE, and diagnostic criteria such as those found in the ICD-10 or DSM-IV.

Total alcohol consumption is either derived from official records on consumption or representative population surveys on consumption. In many countries alcohol is available which lie outside the recorded sphere. This is often called unrecorded alcohol. This alcohol mainly stems from the following sources:

- Home production.
- Import by traveller and cross-border shopping
- smuggling, either organized criminal activity or travellers importing amounts which exceed the legal allowance;
- surrogate alcohol intended for industrial, technical or medical purposes;
- tourist consumption i.e. alcoholic beverages consumed during visits to other countries;
- beverages with alcohol content below the legal definition of alcohol.

In many countries there are beverages which either fall outside of the usual beer, wine and spirit categories or which are traditionally produced at the local level, for example in the villages or at home.

India has experienced social and economic changes since the 1990s. Current trends suggest a steady increase in the production and use of alcohol; these are supported by available data from the organized sector in India[3]. However, a large proportion of alcohol produced in India is illicit and its manufacturing is a 'cottage' industry outside the governmental control[3]. Non-commercial alcohol includes traditional beverages brewed using local production (e.g. rice, wheat, potatoes, molasses and sap from palms and trees) and illicit alcohol spiked with chemicals such as battery acid, urea, ammonium chloride

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and pharmaceutical medication.

Morbidity and mortality due to alcoholic liver disease(ALD) are higher in countries with a higher per capita consumption of alcohol.

Japan and India were previously of low prevalence. Recently alcoholic cirrhosis is gradually increasingly.

ALD may take the form of acute involvement (alcoholic hepatitis), chronic liver disease (steatosis, steatohepatitis, fibrosis and cirrhosis) or acute on chronic liver disease(ACLF)

Anyone drinking more than 30-50 g/d for more than 5-10 years is at risk of developing ALD[4].

Disease Spectrum : ALD is classified into alcoholic fatty liver (steatosis), alcoholic hepatitis (AH; steatohepatitis), and alcoholic cirrhosis.

ALD describes alcohol-related liver injury ranging from reversible fatty liver to alcoholic hepatitis (AH), cirrhosis, and hepatocellular carcinoma (HCC). These multiple stages may be present simultaneously in a given patient[5,6].

Fatty liver develops in > 90 % of individuals who drink more than 60 g / day of alcohol[7,8,9], but may also occur in individuals who drink less[10]. About 25% develop alcoholic hepatitis, about 15% develop alcoholic cirrhosis, and about 10% develop hepatocellular carcinoma[9,11]. The relationship between the quantity of alcohol ingested and the development of liver disease is not clearly linear.

The risk of developing cirrhosis increases with the ingestion of >60-80 g/day of alcohol for 10 years or longer in men, and >20 g/day in women[12,13].

Alcoholic steatosis, the earliest manifestation of ALD, is pathologically characterized by microvesicular and macrovesicular fat accumulation within hepatocytes, minimal inflammatory reaction, and no hepatic fibrosis[14].

Simple fatty liver is usually asymptomatic and self-limited, and may be completely reversible with abstinence after about 4 – 6 weeks[15]. The diagnosis is usually incidental. They do not exhibit stigmata of chronic liver disease, such as spider angiomata and palmar erythema. Patients may present with mild elevations of liver enzymes, including gamma-glutamyl transpeptidase(GGT), aspartate aminotransferase (AST), and alanine aminotransferase (ALT) levels[16].

Some studies have suggested that progression to fibrosis and cirrhosis occurs in 5%-15% of patients despite abstinence[17,18]. In one study, continued alcohol use (>40 g/day) was associated with increasing risk of progression to cirrhosis to 30%, and fibrosis or cirrhosis to 37%[19].

Alcoholic hepatitis (AH) represents a spectrum of disease, ranging from mild injury to severe, life-threatening injury, and often presents acutely against a background of chronic liver disease[20,21]. AH is an inflammatory process with predominantly neutrophilic infiltration, characterized by ballooning degeneration of hepatocytes, hepatocyte necrosis, steatosis, and presence of Mallory bodies (homogeneous, eosinophilic cytoplasmic perinuclear inclusions) within hepatocytes[22]. Clinical findings include jaundice, fever, unintentional weight loss, malnutrition, and tender, enlarged liver[23].

AH typically occurs in an individual with long-standing history of alcohol intake although abstinence for several weeks prior to admission is not uncommon. However, clinical presentation after abstinence of more than 3 months should raise suspicion of advanced underlying alcoholic cirrhosis or chronic liver disease[24].

In the short term, the mortality of AH is closely related to the severity of illness on presentation. Mortality varies with the disease severity with about 20% in mild forms, and between 30% and 60% in severe AH[25].

The survival was about 58% in uncomplicated AH, but 35% in AH with cirrhosis in one study on patients with AH with follow up for over 4 years[26]. The probability of developing cirrhosis in patients with AH is approximately 10% to 20% per year, and approximately 70% of patients with AH will ultimately develop cirrhosis[27].

Fibrosis is believed to start in the perivenular area in ALD. Perivenular fibrosis and deposition of fibronectin occurs in 40%-60% of patients who ingest more than 40-80 g/daily for an average of 25 years. Perivenular sclerosis may be a significant and independent risk factor for the progression of alcoholic liver injury to fibrosis or cirrhosis. Progression of ALD culminates in the development of cirrhosis, which is usually micronodular, but may occasionally be mixed micronodular and macronodular[28].

In this same study, progression of AH to cirrhosis

despite abstinence occurred in a higher proportion of women compared to men indicating that women are at a greater risk of progression to ALD[29].

Risk Factors:

In some populations, alcohol consumption is overtly high (>50 g/day). Nevertheless, only a relatively small number of subjects develops ALD and progresses to cirrhosis. Therefore, there must be other factors that influence ALD development and progression.

Factors increasing susceptibility to ALD

- Lifetime intake of alcohol, duration and amount.
- Frequent Binge drinking upon chronic alcohol intake.
- High concentration alcoholic drinks—for example, spirits.
- Drinking multiple different alcoholic beverages.
- Female sex
- Genetic factors, Genetic polymorphisms
- Race
- Obesity, Physical inactivity.
- Iron storage
- Concomitant Hepatitis B, C, hepatotoxic drugs.
- Nutritional deficiencies.

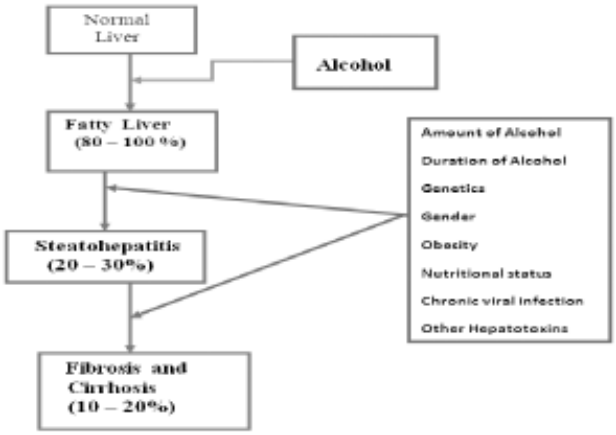

Fig: Progression factors influencing the progression of alcoholic liver disease in alcoholics.

The amount of alcohol ingested (independent of the form in which it is ingested) is the most important risk factor for the development of ALD[30]. The relationship between the quantity of alcohol ingested and the development of liver disease is not clearly linear[31,32]. The risk of cirrhosis or noncirrhotic chronic liver disease increase with a total lifetime alcohol intake of more than 100 kg, or a daily intake of more than 30 g/day[33].

The type of alcohol consumed may influence the risk of developing liver disease. In one study, drinking beer or spirits was more likely to be associated with liver disease than drinking wine[34].

Another factor is the pattern of drinking. Drinking outside of meal times increase the risk of ALD by 2.7-fold compared to those who consumed alcohol only at mealtimes. Binge drinking, defined by some researchers as five drinks for men and four drinks for women in one sitting, has also been shown to increase the risk of ALD and all cause mortality[35].

Women are more sensitive to alcohol-mediated hepatotoxicity and may develop more severe ALD at lower doses and with shorter duration of alcohol intake than men. There are differing blood alcohol levels in women versus men after consumption of equal amounts of alcohol. This gender difference can be explained by differences in the relative amount of gastric alcohol dehydrogenase, a higher proportion of body fat in women, or changes in alcohol absorption with the menstrual cycle[36].

A higher risk of liver injury may be associated with an individual's racial and ethnic heritage. The rates of alcoholic cirrhosis are higher in African-American and Hispanic males compared to Caucasian males and the mortality rates are highest in Hispanic males[37]. These differences do not appear to be related to differences in amounts of alcohol consumed.

Malnutrition plays an important role in determining the outcome of patients with ALD. Mortality increases with the extent of malnutrition, approaching 80% in patients with severe malnutrition (i.e., less than 50% of normal)[38]. Micronutrient abnormalities, such as hepatic vitamin A depletion or depressed vitamin E levels, may also potentially aggravate liver disease.

The presence of long-standing obesity is an independent risk factor for liver disease and cirrhosis in alcoholics. Obesity potentiates the severity of ALD in all its stages, including fatty liver, AH, and cirrhosis[39]. The synergy between obesity and heavy alcohol intake presumably reflects similar mechanisms of disease for both ALD and non-alcoholic fatty liver disease, along

with the direct fibrogenic effects of expanded larger mass of adipose tissue (via high levels of noradrenaline, angiotensin II and leptin, and low levels of adiponectin).

There is higher occurrence of alcoholism in adopted children of alcoholic parents and in monozygotic twins compared to dizygotic twins[40]. Monozyotic twins have a significantly higher prevalence of alcoholic cirrhosis than dizygotic twins. Polymorphisms of genes involved in the metabolism of alcohol (including alcohol dehydrogenase, acetaldehyde dehydrogenase and the cytochrome P450 system), and in those which regulate endotoxin-mediated release of cytokines have been associated with ALD.

Polymorphism of the gene encoding for CD14 has been implicated in the risk of ALD. CD14 molecule is expressed on Kupffer cell which helps in sensitization of these cells to endotoxin. There is a correlation between endotoxin levels and alcohol-induced liver damage. Chronic ethanol exposure amplifies CD14 expression , suggesting sensitization of these cells to stimuli like endotoxin, which may be linked to polymorphism in the promoter region of the CD14 gene.

The polymorphisms of genes encoding for proinflammatory (e.g. TNF-a) and anti-inflammatory (e.g. interleukin IL-10) cytokines are associated with increased risk of developing ALD.

Coexistence of alcohol consumption and chronic hepatitis C virus infection leads to an acceleration of liver injury . From these data it can be concluded that individuals with chronic hepatitis C who drink more than 30 g per day increase their risk of developing fibrosis approximately by 4-fold. However, one study has even quantified the risk of cirrhosis as 30 times greater in patients with hepatitis C who consume alcohol to excess[41,42]. The combination of hepatitis C virus and alcohol predisposes to more advanced liver injury than alcohol alone, with disease at a younger age, more severe histological features, and a decreased survival.

Excess iron in liver has been associated with fibrosis in ALD and increased mortality in alcoholic cirrhosis . Elevated serum iron indices are common in ALD patients than in alcohol misusers without liver disease. However, there is no clear association with the C282Y HFE gene mutation. Some studies have described an association with the H63D mutation. Certainly alcohol and iron can act synergistically to produce oxidative stress and thus potentiate progressive liver damage[43].

CONCLUSION :

In addition to amount of alcohol intake and alcohol consumption patterns, factors such as gender and ethnicity, genetic background, nutritional status, energy metabolism abnormalities, oxidative stress, immunological mechanisms and hepatic co-morbid conditions play important role in the genesis and progression of alcoholic liver injury. Identification of risk factors for the development of ALD may help us to set preventive strategies, and to understand the means through which liver damage arises, thus providing the background for developing therapeutic approaches.

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Case Report

A Case of Male SLE with Discoid Skin Lesions

D Goswami, M P Das[*], A Deuri[**], B Brahma[***], S Das[****], C K Das[****]

ABSTRACT :
A 23yrs old male presented to Gauhati medical college and hospital with generalised discrete skin lesions with central depression and raised hyperpigmented border with adherent scales, hair loss and generalised weakness along with anaemia (Hb%- 8.8), proteinuria(0.52 g/d) and positive ANA & anti Smith and U_1SNRNP. He was diagnosed to be a case of male SLE with discoid skin lesions and put on steroids and intravenous cyclophosphamide and improved.

KEY WORDS : *Systemic lupus erythematosus (SLE), ANA, Anti Smith antibody, Discoid skin lesion.*

A 23 yrs old male, Nirmal Ch. Roy, hailing from Gologgang, Dhubri district, Assam, presented with –

- Skin lesion all over the body. It started 3 months back as papulo-squamous lesion over chest which later spread to back, trunk, upper and lower limbs (mainly extensor surface) within next 8-10 days. Face and lower body was also involved. The lesion gradually enlarged, coalesced to form large non-pruritic, non-tender lesions with scaling and atrophic centre.

There was hair loss in the form of increased loss in combing, during head washing and large amount of hair in the pillow in the morning.

- There is also generalised weakness for last 3 months which was insidious in onset, gradually progressive in nature. He has also on and off fever, occasionally high grade.
- On October 2013, he was treated for skin lesions with inj. Penidure 1.2 million units (2 doses 21 days apart taken) and tab Azithromycin 500mg by a local doctor but the lesions did not improve, rather it has worsened and became darker . The patient had on and off generalised, pruritic skin lesions since childhood which usually used to subside on anti allergic medications.

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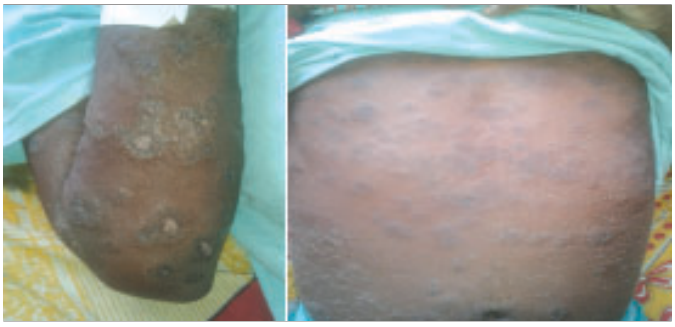
On examination:



- Patient looked sick, pallor and evidence of hair loss was present
- Discrete skin lesions over face, trunk and limbs were present with central depression and raised hyper pigmented border. There were adherent scales to the lesions and on removal of scales, underside shows excrescences. There was scarring.
- On CNS examination, higher functions were intact. All crnial nerves were intact. Motor and sensory system examinations were normal. Fundoscopy was within normal limits.

INVESTIGATIONS:

- Hb-8.8 g/dl, DLC- N-66% L-21% M- 12% E-.7%, PLT-105x10^3 , PBS-normocytic, normochromic anaemia, ESR- 25, CRP- 50
- R/E urine- protein + ,WBC- 8 to 10, RBC-1 to 2.
- 24 hr urinary protein- 520 mg.
- RBS- 103, Creat- 0.78, sodium-136, potassium-

3.8,bilirubin-1.14, AST- 445, ALT- 215.

- HIV- NR, VDRL- NR, Hb_sAg & anti HCV – negative.
- ANA (IFA) + ve, primary dilution 1: 40, primary intensity of IF 4+, speckled pattern, end point titre 1:2560, D_sDNA negative, Anti smith +ve, U_1SNRNP +ve, SSA & SSB negative, anti Histone, anti centromere, SCL 70 & JO 1 are all negative.
- PT-14.8, INR-0.92, APTT-38.1
- USG whole abdomen reveals bilateral renal parenchymal changes(right kidney- 12.5x4.9 cm, left kidney- 12.7x5.8 cm)

DIAGNOSIS:

There were skin lesions which on examination were found to be discrete lesions over face, scalp, trunk and limbs which were erythematous plaques with thick adherent scales. Patient was anaemic(Hb%- 8.8) with proteinuria(0.52 g/d), significant ANA titre and positive Anti smith and U_1SNRNP antibody. He was diagnosed to be a case of male SLE with discoid skin lesions.

TREATMENT:

The rash disappeared completely on infusion of steroid and the patient was also given cyclophosphamide infusion and patient is now presently doing well and under follow up.

DISCUSSION:

SLE is an autoimmune disease in which organs and cells undergo damage initially mediated by tissue binding autoantibodies and immune complexes. 30% patients at diagnosis are women at child bearing age. Male patients with systemic lupus erythematosus (SLE) are thought to be similar to female patients with SLE, but key clinical characteristics may differ. Indian SLE patients too showed differences in clinical manifestations between male and female patients supporting the hypothesis that gender biases exist in clinical expression of the disease.[1] Comparisons were made between male and female patients with SLE in the Hopkins Lupus Cohort. Men were more likely than women to have disability, hypertension, thrombosis, and renal, hematological, and serological manifestations and end organ damage. In general, differences between males and females were more numerous and striking in whites, especially with respect to lupus nephritis, abnormal serologies, and thrombosis.[2] During the follow up a lower incidence of arthritis and malar rash and a higher incidence of other skin complications including discoid lesions and subcutaneous lupus erythematosus was found in the men.[3] The increased frequency of SLE among women may be attributed to differences in the metabolism of sex hormones and/or GnRH. Though less common in men, when it does occur SLE tends to run a more severe course-an important consideration in the diagnosis and follow-up of male patients with SLE.[4]

Lupus dermatitis can be classified as discoid lupus erythematosus (DLE), systemic rash, subacute cutaneous lupus erythematosus (SCLE) or others. Discoid lesions are roughly circular and slightly raised, scaly hyperpigmented erythematous rims and depigmented atrophic centres in which all dermal appendages are permanently destroyed. Lesions can be disfiguring, particularly on the face and scalp. Only 5% of people with DLE have SLE (although one-half have positive ANA); however, among individuals with SLE, as many as 20% have discoid skin lesions.

CONCLUSION:

SLE is an autoimmune disease mainly found in females. Discoid skin lesions in SLE is not a common finding in this part of the world, it is common in African Americans.[5] SLE in Indian males has an earlier age of disease onset, a higher incidence of mucocutaneous and renal involvement and a lower incidence of neuropsychiatric, gastrointestinal and hematological disease in comparison to those published from the developed countries.[6]

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Case Report

Vertebral artery dissection in young male: leading to Opalski syndrome - Case Report

S Choubey*, D K Boruah R Patil, S Mali*****

ABSTRACT :

Vertebral artery dissection is uncommon cause of stroke. Lateral medullary infarction (Wallenberg syndrome) is a relatively common vertebrobasilar vascular syndrome in VAD which is usually not associated with paralysis. However, ipsilateral hemiparesis as part of lateral medullary infarction is known as Opalski's syndrome which is very rare.. We had a young male patient with symptoms typical of vertebral artery dissection and weakness that was specific of this rare syndrome. It was confirmed by specific investigations like MRI brain and CT angiography and treated conservatively with antiplatelet agents.

Vertebral artery dissection is area lead to focal neurologic signs attributable to ischemia of the brainstem or cerebellum ultimately develop in 85% of patients; however, a latent period as long as 3 days between the onset of pain and the development of CNS sequelae is not uncommon. When neurologic dysfunction does occur, patients most commonly report symptoms attributable to lateral medullary dysfunction (ie, Wallenberg syndrome).

Patient history may include, ipsilateral facial dysesthesia (pain and numbness), dysarthria or hoarseness (cranial nerves [CN] IX and X), contralateral loss of pain and temperature sensation in the trunk and limbs, vertigo,nausea and vomiting, diplopia or oscillopsia,dysphagia (CN IX and X), disequilibrium, unilateral hearing loss. Rarely, patients may manifest contralateral weakness or paralysis (Lesion of pyramidal tract in medulla above the level of dicussation).

However, ipsilateral hemiparesis as part of lateral medullary infarction below the level of dicussation is rare, and is known as Opalski's syndrome. Hence the case with vertebral artery dissection with Opalski's syndrome is quite notable case.

INTRODUCTION :

Vertebral artery dissection is a rare condition but is an increasingly recognized cause of stroke in patients younger than 45years. Although its pathophysiology and treatment closely resemble that of its sister condition, carotid artery dissection (CAD), the clinical presentation, etiology, and epidemiological profile of VADs are unique. Lateral medullary infarction (Wallenberg syndrome) is a relatively common vertebrobasilar vascular syndrome in VAD which is usually not associated with paralysis. Combined lateral and medial medullary syndrome leads to contralateral paralysis of arm and leg. However, ipsilateral hemiparesis as part of lateral medullary infarction is known as Opalski's syndrome. Some pathologic and neuroradiologic reports have shown that the lesion is located lower than in Wallenberg syndrome, and the ipsilateral hemiparesis seen in this syndrome is attributed to the involvement of corticospinal fibers caudal to the pyramidal decussation.[1] And also, Opalski's syndrome with cerebellar lesion is rare.

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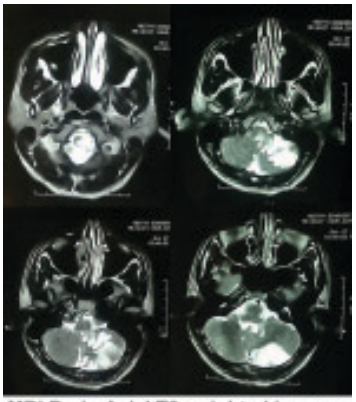
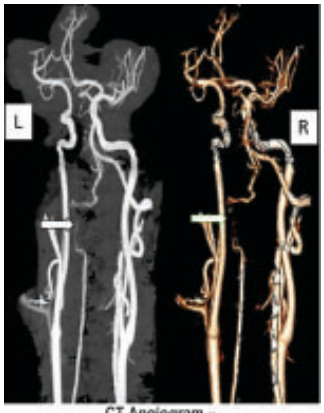
CASE REPORT :

A 32 year old male shopkeeper non-diabetic, non hypertensive came to our hospital with history of severe left sided neck pain and same sided occipital headache followed by weakness of left side of body. He was suffering from that neck pain and headache for 15 days in low intensity. On the day of admission his pain was sharp shooting type and radiating to ward head aggravated by Left lateral neck movement. His weakness had started after 6 to 8 hrs after aggravation of pain and progressed over 1-2 hr to complete immobility of left side of body. He was also complaining of diplopia for 2-3 hr, giddiness, nausea and one vomiting, hiccups, slight difficulty in eating. There was no h/o trauma to spine or any chiropractic neck movement. There was no significant past or family history. He was occasional alcoholic and nonsmoker.



MRI Brain Axial T2 weighted image – Abnormal T2 hyper intense lesion in Lt PICA & AICA territories



CT Angiogram – MIP (Maximum intensity projection) & VRT (Volume reduction technique) images showing Lt vertebral artery dissection

On thorough clinical examination he was conscious and well oriented, his pulse was 70/min, BP was 130/80 mmHg. He was having mild ptosis and constricted pupil in his left eye, and we also noticed decreased sweating on his face after 2-3 days suggesting that he had left sided horners syndrome. Nystagmus was present in left lateral gaze. He had 8 to 11 cranial nerve palsies. He had hypotonia and 0/5 power in left UL and LL. His reflexes were brisk and planar extensor on left side. His pain and temperature sensation over left side of face were decreased. He had posterior column sensory loss on left side body. Only abnormality on right side was decreased pain and temperature sensation. These were features of Brown-Séquard syndrome. His CT brain was normal, MRI brain and upper cervical cord was done on second day showing Infarct in left PICA & AICA territories involving cerebellum, left posterior-lateral portion of medulla and spinal cord up to lower part of C2. CT angiography done on next day showing left vertebral artery dissection in its third part. He was given antiplatelet agents. Patient improved and was able to eat and move his limbs at the time of discharge.

DISCUSSION :

Although a PICA occlusion can be the cause of Wallenberg's (lateral medullary) syndrome, this syndrome is more often caused by an intracranial vertebral artery occlusion. This syndrome produces an ipsilateral Horner's syndrome; loss of pain and temperature sensation in the face; weakness of the palate, pharynx, and vocal cords; and cerebellar ataxia.

Involvement of the ipsilateral posterior spinal artery that arise from the VA or PICA may account for ipsilateral sensory loss from infarction of the ipsilateral dorsal column nuclei as a component of the lateral medullary wedge syndrome.[2]

Contralateral to the lesion, there is hemibody loss of pain and temperature sensation.

The medial medullary (Dejerine) syndrome is less common and may be caused by occlusion of the distal vertebral artery, a branch of the vertebral artery, or the lower basilar artery. Vertebral artery dissection, dolichoectasia of the vertebrobasilar system, and embolism are less common causes of the medial medullary syndrome. The findings with this syndrome include an ipsilateral lower motor neuron paralysis of the tongue and contralateral paralysis of the arm and leg. The face is often spared. In addition, there is contralateral hemibody loss of tactile, vibratory, and position sense. Occlusion of the intracranial vertebral artery can lead to a total unilateral medullary syndrome (of Babinski-Nageotte), a combination of the medial and lateral medullary syndromes.[3]

The Babinski-Nageotte syndrome is caused by hemimedullary infarction and combines the medial medullary and the lateral medullary syndromes.[4]

The submedullary syndrome of Opalski is caused by VA occlusion with extensive infarction of the cervicomedullary junction. The patient has the lateral medullary wedge syndrome plus ipsilateral hemiplegia because of infarction of the pyramidal tract after its decussation.[5]

The lesion also may affect the ipsilateral dorsal column nuclei.

Our paient had symptoms of lateral medullary syndrome that is ipsilateral nystagmus, vertigo, nausea, vomiting due to involvement of inferior cerebellar peduncle and vestibular nuclei.

He had decreased pain and temperature on left side of face due to involvement trigeminal nucleus and tract, decrease pain and temperature sense on right side due to involvement of spinothalamic tract. Left sided Horners syndrome due to involvement of descending sympathetic tract fibres. Dysphasia due to involvement of nucleus ambigus, left sided taste sensation loss due to involvement of nucleus tractus solitaius, left sided posterior column sensation loss due to involvement of

dorsal column nuclei as described above.

Along with that he had left sided hemiplegia due to involvement of lateral corticospinal tract upto cervical second segment (pyramidal tract after its dicussation – cervicomedulary junction.)

CONCLUSION :

Spinal hemiplegia (Brown-Séquard syndrome) due to other causes can be confused with hemiplegia due to cervicomedullary junction infarction but good history of neck pain and clinical examination of cranial nerve involvent will differentiate the diagnosis from vertebral artery dissection. Vertebral artery dissection in young adult is common but extensive involvement of lateral medulla and inferior cerabellum with upper part of spinal cord can occur.

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Case Report

Aortic Regurgitation as a Manifestation of Takayasu's Arteritis

P Dihingia[*], M S Chaliha[**], P Goswami[***], D K Baruah[****], S Kar[*****], G R Pramod, B Agarwal[******]

ABSTRACT :

Mrs JK a 40 yr old female patient presented with palpitations,giddiness, and tingling sensation of left arm and on examination there was radio radial delay with wide pulse pressure difference between arms, and early diastolic and ejection systolic murmurs in the neo aortic and aortic areas and on investigating with echocardiography and colour Doppler severe aortic regurgitation was found with stenosis of the left subclavian arteries which suggested takayasu's arteritis.

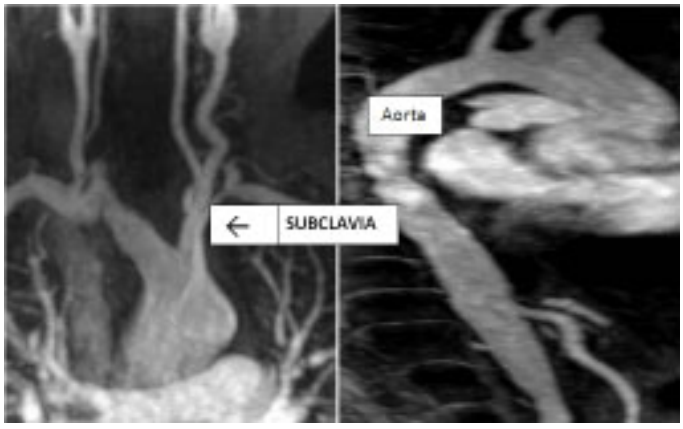
KEY WORDS : *AR: Aortic Regurgitation, TA: Takayasu's Arteritis.*

INTRODUCTION :

TA is a chronic idiopathic vasculitis which mainly involves aorta and its branches, inflammation results in stenosis, occlusion with aneurysms, it is usually most common in young women and adolescent girls[3], the incidence of aortic regurgitation in TA is found to be 13-25% of which AR is now considered an important risk factor for mortatlity in patients with this disease. Here we present a case of aortic regurgitation in TA[1,2].

CASE REPORT :

Mrs JK a 40 yr old female patient presented with palpitation, giddiness and tingling sensation of left arm of the body since 1 year, there was no history of chest pain, pedal oedema, difficulty in breathing, discolouration of arms, no pain abdomen, blurring of vision. Patient is a known hypertensive and diabetic on regular medication, no history of TB and there was no significant past history noted.On examining there was wide pulse pressure difference between arms, and there was radio radial delay.A bruit felt in the bilateral carotids and pulse volume were being equal in both the carotids, pulses were also normal at other peripheral arteries, there was no renal bruit. Respiratory and central nervous system were normal, cardiovascular system examination revealed systolic and early diastolic murmurs over aortic and neo aortic areas with no other significant findings. On investigating patient ESR and CR Pwere slightly raised rest of blood parameters were within normal limit. Doppler sonography of bilateral carotid, vertebral and subclavian arteries showed: Diffuse thickening of aortic arch branches with focal stenosis of left sub clavian artery, MR Angiography revealed features suggestive of aortoarteritis affecting the ascending thoracic to upper abdominal aorta and affecting its major branches, Echo showed severe aortic regurgitation and mild mitral regurgitation with Ejection fraction of 67%.



Images of MRI Angiography

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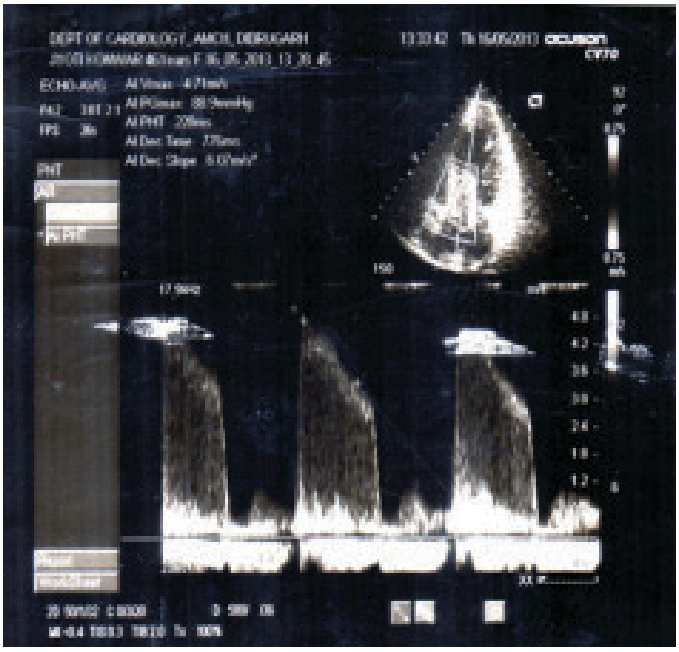

Images of Echocardiography

DISCUSSION :

Takayasu's arteritis is a systemic disorder that affects multiple organs. The diagnosis of TA can be a challenge, especially in its initial phases and symptoms are generally constitutional, including malaise, fever, fatigue, and arthralgia. 'Aortic arch syndrome' is the term given to disease affecting the upper extremities, heart, neck and head[6]. Patients often complain of arm claudication, and on examination absence of peripheral pulses. Hence TA was previously called 'pulseless disease'[7]. Blood pressure varies by more than 10 mmHg between the arms and a bruit may be audible over the artery. Aortic regurgitation due to involment of ascending aorta[6,8,9], pulmonary hypertension, angina, congestive cardiac failure, vertigo, syncope, stroke and visual disturbance may occur. Descending aorta syndrome may cause renovascular hypertension, renal dysfunction, abdominal pain and acute abdominal bleeding or perforation of a viscus from infarction.The finding of hypertension and arterial bruits in young adults necessitates the examination of pulses and blood pressures in different limbs in order to detect asymmetry. Elevated erythrocyte sedimentation rate is a common finding; however, caution is advised, because up to 50% of patients may have active TA disease and a normal sedimentation rate.

Confirmation of TA is best done by angiography or MRI angiography[4,5]. The most common lesion is a smooth, concentric, arterial or aortic narrowing (85%). Irregular narrowing, complete occlusion and fusiform or saccular aneurysms are less commonly seen. Changes may be focal or segmental and are distinguished from arteriosclerosis and fibromuscular dysplasia. Contrast-enhanced magnetic resonance perfusion imaging, ultrasonography and positron emission tomography are new, non-invasive methods of assessment that are likely to replace conventional angiography[10,11].

Renovascular hypertension, coarctation of aorta, severe cerebral ischaemia, and severe aortic regurgitation causing congestive heart failure, or progressive aneurysmal enlargement or dissection may all require prompt surgical treatment.

In TA involvement of aortic root leading to AR is around 13-25% as the patient with this disorder can be managed with steroids[12] or surgical valve replacement therapy, our patient was managed with only oral glucocorticoids as the patient was asymptomatic and patient was adviced for follow up.

CONCLUSION :

TA depending upon the artery involved can present with wide variety of clinical manifestations,for a definitive diagnosis the use of modern day imaging such as CT, MRI and angiography is vital ,The use of steroids is paramount to the acute medical treatment but not curative. Surgery, angioplasty or stenting is only required in a minority of patients.A case of TA with severe AR which is not so common manifestation has been reported in this case study.

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Case Report

Hypokalemic Quadriparesis : A Rare Manifestation of Dengue Fever

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ABSTRACT :

Dengue, also called break-bone fever or dandy fever, is an acute, infectious, mosquito-borne fever that is temporarily incapacitating but rarely fatal[1]. Dengue is caused by one of four viral serotypes, designated DEN-1, DEN-2, DEN-3, and DEN-4[1]. These serotypes are members of the *Flavivirus* genus. *Aedes aegypti* is the vector responsible for transmission of the viral infection[1].

Dengue has a wide clinical spectrum, ranging from an uncomplicated febrile illness to acute fulminant cases with devastating hemorrhages and refractory shock. Hemorrhagic manifestations in dengue range from petechial hemorrhages to the life-threatening gastrointestinal, pulmonary, cerebral or genitourinary hemorrhages.

There are few reports of varied neurological involvement of dengue virus infection which have been increasingly recognized over the last many epidemics. Various neurological involvements reported in dengue virus infection are encephalitis, acute disseminated encephalomyelitis, transverse myelitis, Guillain-Barre syndrome , myositis, and stroke[2,3,4,5,6,7].

The neurological spectrum of dengue patients has been limited because of small number of case reports, paucity of imaging, and neurophysiologic studies. There are only a few isolated case reports and case series documenting acute quadriparesis in dengue fever[2, 6,7]. Here, we report a case of acute reversible quadriparesis due to hypokalemia, secondary to dengue virus infection.

CASE REPORT :

A 34 year old male was admitted to our hospital with history of intermittent high grade fever without chill or rigor for 2 days. There was associated generalized body ache and decreased appetite for 2 days. There was no history of cough or cold, chest pain or pain abdomen, bowel or bladder habit abnormalities. There was no history of similar illness in the past. There was no history of diabetes or hypertension but there was history of jaundice 10 years back. History of any other drug intake could not be elicited.

He was unmarried, non-vegetarian and had no addiction.Family history was insignificant.

On general examination, patient was of moderate built and looked ill but was nourished, conscious and oriented. Pulse was 96/min, BP was 120/80 mm of Hg and he was febrile (oral temperature was 100° F), but there was no pallor, icterus, cyanosis, rash, generalized edema or lymphadenopathy. Cardiovascular, respiratory, abdominal and neurological system examination was normal.

Laboratory examination revealed Hemoglobin of 13 gm%, total leukocyte count 4,400/mm^3 with 44% polymorphs, 54% lymphocytes and 2% monocytes, platelet count 70,000/mm^3, haematocrit: 43.2, random blood glucose 89 mg/dl, CRP 4.5 mg/L. S. Creatinine: 1 mg/dl Liver function tests revealed normal Bilirubin level with SGPT of 128 IU/L. Peripheral blood smear did not reveal malarial parasite. Dengue NS1 antigen test was found positive at our hospital (NS 1 antigen, IgM, IgG using J Mitra Kit) which was confirmed at Gauhati Medical College by positive IgM Dengue antibody test.

2 days after admission to the hospital, he fell down while going to the bathroom and complained of sudden onset and rapidly progressing weakness of all four limbs which involved both the legs initially and gradually progressed to involve both the upper limbs simultaneously within about 4-5 hours. There was no history of loss of

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consciousness, seizure, vomiting or irrelevant talk nor there was any history of recent vigorous exercise, heavy carbohydrate intake, trauma or vaccination. There was no history suggestive of any cranial nerve involvement, bowel or bladder disturbance and neck pain. There was no history suggestive of sensory involvement.

On neurological examination, power in all group of muscles of both upper and lower limbs was grade 2/5 (Medical Research Council Scale) and deep tendon reflexes were absent. Plantar response was flexor in both lower limbs. There were no signs of cranial nerve dysfunction or sensory dysfunction. Cranium and spine was normal and there were no signs of meningeal irritation. ANS examination was normal.Cardiovascular, abdominal and respiratory system examination was normal.

Laboratory tests revealed blood urea 33 mg/dl and serum Creatinine 1.0 mg/dl. His Serum potassium was 2.3 meq/L, serum Sodium 136 meq/L and Serum chloride 115mEq/L. Arterial blood gases revealed pH of 7.40, bicarbonate 21.6 mmol/L. Urine pH was 6.5 (normal). Urine spot sodium was 123mEq/L (Normal-100 to 260mEq/L) and Potassium 22.9mEq/L (Normal-25 to 100mEq/L).There was no proteinuria or glycosuria.Electrocardiogram revealed flattened T waves.Electro-diagnostic studies revealed bilateral symmetrical sensory motor neuropathy of upper and lower limbs.CSF analysis revealed protein of 28 mg/dl (Normal 8-32mg/dl), normal sugar level, and cell count of 2-3/HPF with all lymphocytes. X-ray Cervical spine was normal.

A diagnosis of Dengue fever with thrombocytopenia and acute hypokalemic-quadriparesis was made. Patient was given 40 meq/L of potassium chloride (KCl) infusion in 500ml of Normal saline and ringer lactate 1 liter per day for 2 days. Oral potassium syrup in the dose of 10 ml thrice daily mixed in water was given. Fever was treated with oral paracetamol. In the next 3 to 4 days there was gradual improvement in power and it was grade 5/5 on the 5th day of onset of paralysis with increase in serum potassium to 4.6 meq/L on 7th day. Patient was observed for any bleeding diathesis. Periodic platelet count monitoring did not show deterioration and platelet count reached 1.4 Lakh/ mm^3 on 5th day of admission. Patient was discharged after 10 days.

Differential diagnosis

- Acute flaccid quadriparesis in early phase of myelitis
- Channelopathies
- Acute Guillain–Barré syndrome (GB) syndrome
- Compressive myelopathy in spinal shock.

DISCUSSION :

Dengue is the most rapidly spreading mosquito-borne viral disease in the world. In the last 50 years, incidence has increased 30-fold with increasing geographic expansion to new countries and, in the present decade, from urban to rural settings[8]. Dengue infection is caused by four antigenically distinct dengue virus serotypes (DENV1, DENV2, DENV3 and DENV4)[1]. Dengue infection is usually asymptomatic. Symptomatic dengue was previously classified as classic dengue fever, dengue hemorrhagic fever or dengue shock syndrome. WHO (World Health Organization) suggested a new classification of Dengue infections which included Dengue without warning signs, Dengue with warning signs, and severe dengue[8].

Dengue is generally regarded as non-neurotropic virus. However few case reports indicate neurotropism of dengue virus. Various neurological complications of dengue fever such as encephalitis, encephalopathy, stroke syndromes, myelitis, myositis, acute disseminated encephalitis, Guillain Barré syndrome, mononeuropathies and hypokalemic paralysis are reported[2,3,4,5,6,7].

Acute hypokalemic quadriparesis is a rare manifestation of dengue fever. Hypokalemic paralysis in dengue fever have been reported by few authors[6,7]. Our patient of dengue fever had acute quadriparesis due to hypokalemia which improved with treatment.

Hypokalemic paralysis can occur in other conditions like thyrotoxicosis, following intake of drugs such as diuretics, diarrhea and urinary potassium wasting syndromes (Barter's syndrome, Gitelman's syndrome) and acute tubular necrosis.[9]

Quadriparesis has been reported in other infectious diseases like chikungunya fever[10] and malaria[11]. In our patient peripheral blood smear for malaria parasite was negative. Antibodies to Chikungunya virus were not done as this virus is not seen in this region of India and test kit was not available.

Guillain Barré syndrome and familial periodic

paralysis was considered as differential diagnosis. Presence of sudden onset of areflexic quadriparesis, distal parasthesia, electro physiologic evidence of sensory and motor axonal dysfunction points towards Guillain Barré syndrome especially acute motor sensory axonal neuropathy. However the presence of fever at the time of motor weakness, rapid recovery with parenteral and oral administration of potassium preparation and normal cerebrospinal fluid examination excludes the possibility of Guillain Barré syndrome. Moreover, acute motor sensory axonal neuropathy variant of Guillain Barré syndrome is typically fulminant generally with slow and incomplete recovery and our patient recovered rapidly with potassium supplementation. Absence of family history of episodic motor weakness and this being first episode of motor weakness that the patient suffered, familial periodic paralysis can be excluded.

Electrophysiological study in our patient showed involvement of motor and sensory nerves. Motor nerve involvement is known in hypokalemic paralysis. But sensory nerve dysfunction in hypokalemic paralysis is also reported. With the evidence of dengue serology being positive, documented low serum potassium and excluding the above mentioned differential diagnosis, final diagnosis of hypokalemic quadriparesis secondary to dengue infection was made.

CONCLUSION :

Acute Hypokalemic quadriparesis is an uncommon presentation of dengue fever, not yet widely recognized. Only few cases are reported from different institutes worldwide. The exact mechanism of hypokalemic paresis in dengue remains elusive and is a field awaiting further exploration. Hypokalemic quadriparesis secondary to dengue fever should be considered as differential diagnosis in a case of sudden onset of fever with areflexic quadriparesis.

This case report is to increase the awareness of clinician to a treatable entity

ACKNOWLEDGEMENT :

We owe our sincere thanks to the patient and his relatives for consenting and contributing to this undertaking.

Footnotes : Competing interests: None.

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Case Report

Evans Syndrome as a Forerunner of SLE

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ABSTRACT :

Evans syndrome was originally described in 1951. It is an autoimmune disorder characterized by simultaneous/ sequential development of autoimmune hemolytic anemia (AIHA) and immune thrombocytopenia (ITP) and or immune neutropenia in the absence of an underlying cause[1,2]. Here we report a case who presented and was being treated as idopathic evans syndrome initially but progressed to develop features characteristic of Systemic Lupus Erythematosus.

INTRODUCTION :

SLE (systemic lupus erythematosus) is an autoimmune disease in which the organs and cells undergo damage initially mediated by tissue -binding autoantibodies and immune complexes.SLE is a multigenic disorder ,female sex being permissive for it with evidence of hormonal effects.[3] Among its varied, unpredictable and sometimes unconventional presentation it is seen that 50% SLE cases are associated with Evans syndrome (Secondary Evans syndrome). We are hence reporting a case who is being treated as idiopathic Evans syndrome, and gradually developed serious life threatening complications .During the course of her treatment the patient developed clinical features and some strong laboratory evidence to label her as a case of SLE, fulfilling all the four ACR criterias.

CASE :

A young girl aged 18years was incidentally detected for the first time to have thrombocytopenia when she consulted a physician for menorrhagia. Apart from these there were no history of other illness in the past, no symptoms indicating any other organ system involvement nor any systemic features like fever, malaise etc. Patient was investigated thoroughly and on one ocassion ANA was positive. Then the patient was referred to a rheumatologist who started her on immunosuppressants and steroids which was discontinued later, on advice.

On the second time presentation; she was still having menorrhagia, was severely pale and was in acute LVF(Left Ventricler Failure).

Laboratory investigations revealed ..... Hb -7. 4 gm%,ESR-120 mm/hr, Platelate count-38,000/cumm,Alb +,Cast- granular, cellular cast on routine urine examination, Sputum Culture/sensitivity-Yeast cell with pseudo hyphae, S.LDH-1950 U, Retic count-24%.

Patient was started on diuretics to stabilize acute LVF and later as she stabilized she was put on IVIg to correct the platelet count. However the patient developed cough even though she was on antibiotic coverage for UTI . Chest x-ray and sputum examination revealed candida infection of the lungs. A course of voriconazole was given and it brought on a symptomatic relief, however gradually patient started developing psychosis and a high grade fever. On repeat examination the findings were unremarkable, chest had cleared, urine exam showed no infection , platelet count had rised to near normal, total count was normal so was the differential counts, CRP was within normal limits, but dsDNA was high and C3 level low. At this time patient was started on steroids (methyprednisolone pulse therapy). Patient responded well, fever subsided and psychosis improved.MRI brain revealed multiple arachnoid cyst with non specific atherosclerosis and 24 hours urinary

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protein was 1.2 gm.

The patient was diagnosed as SLE with lupus nephritis and evans syndrome and was discharged with medication and referred to higher centre for renal biopsy (for staging).

DISCUSSION :

The term *lupus* (Latin for "wolf") was first used during the Middle Ages to describe erosive skin lesions that were evocative of a wolf's bite. The "classical" period of lupus starts in 1846 with the Viennese physician von Hebra (1816-1880), who introduced the butterfly metaphor to describe the malar rash.[3]

SLE has a chronic course that is often complicated by exacerbations and flares of varying severity. In the present case she met with the diagnostic criteria by neurological(arteriosclerosis and psychosis), nephrological (granular cast and proteinurea) and ANA and DsDNA positivity and features suggestive of hemolytic anemia. All favoured the diagnosis of SLE. But the usual manifestation like rash, photosensitivity, aphthous ulcer, hair fall, arthritis, serositis were not present unlike the usual presentations. Moreover normaly fungal septicemia in SLE cases are not very common. Many large retrospective studies have elaborated invasive fungal diseases in the form of Cryptococcus[4] followed by histoplasmosis[5] but not candida species. Although the exact prevelance rate of fungal infection are not studied yet but all the patients having fungal infection were taking steroids for their disease process.

Although Evans syndrome was initially described idiopathic but in the long run it has been found to be associated with many diseases such as SLE[6] ,lymphoproliferative disorder[7,8] or primary immunodeficiencies[9]. The prevelance of secondary Evans syndrome (disease associated) is almost 50% and it is predominantly associated with SLE or to a lesser extent common variable immunodeficiency state in younger patients (ie, age younger than 45 years), whereas NHL was the most commonly associated disease in patients aged older than 50 years[10]. But in the present case the patient presented with Evans syndrome without fulfilling the criteria for diagnosing SLE initialy. But during the course of treatment and investigations appeared that the patient was having SLE flare due to infection.

CONCLUSION :

SLE remains largely a clinical diagnosis. The diagnosis of mild SLE at the early stages of the disease may present considerable challenges. Strict adherence to the classification criteria may sometime miss many patients. All patients present with different manifestations of SLE at different point of time. The required four criterias to diagnose SLE may not be present at a time. But high degree of suspicion is required specially in young girls presenting with thrombocytopenia and anemia.

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Case Report

Unusual Presentation of Foreign Body in Airway

K R Sarmah[*], D K Bhattacharjee[**], J Sarma[***], S Choudhury[****]

ABSTRACT:

Foreign body in airways is a common problem in pediatrics age group. Although uncommon in adult, airway foreign body can present with various manifestation and even remain undiagnosed for long time. The established method to remove foreign body from airway is rigid bronchoscopy however in certain cases flexible bronchoscopy can be used with success.

CASE :

A 27 year old male presented with history of cough for two months. There were no other respiratory or systemic complaints. On examination, he had normal general physical examination. His vitals were normal, RR 18/min, BP 110/70 mm Hg. On chest examination there were crackles on the right side of the chest (infra-mammary area). Other systemic examinations like that of the cardiovascular system, abdomen, CNS etc did not show any significant abnormality. Patient's Hb, TLC, DLC and ECG normal. Chest x ray showed right sided bony opacity which was lying along the right main bronchus. Subsequent CT thorax revealed a piece of bone in the right bronchus (Fig1), almost looking like a stent (Fig 2). The size was approximately 47 × 13 mm (Fig 3). On reviewing the history patient revealed that about four month ago he had accidentally ingested a foreign body assuming it to be a piece of chicken bone. He had consulted a local physician he assured him that the chicken piece would enter his stomach and would then be excreted with feces.

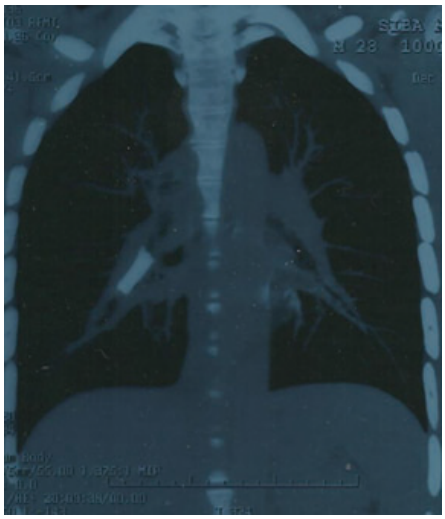

Fig 1: Tomogram showing a bony shadow along right bronchus

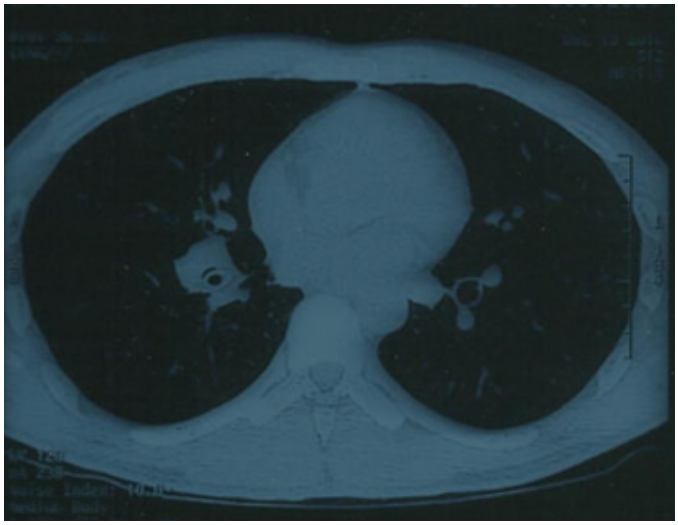

Fig 2: CT thorax showing lodgment of bone as stent inside right bronchus intermedius

Having located the bone piece in the CT scan we decided to perform a rigid bronchoscopy as the bone was distally located and the size was also suitably large enough. Bronchoscopic view showed normal vocal cord, trachea and left bronchial tree. The right bronchial tree was full of secretions, the bone was lodged distal to right intermediate bronchi just distal to right upper lobe bronchus (Fig 4). The edge of the FB was grasped with

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a FB forceps and the bone was removed successfully. Follow up chest x-ray was done which was found to be normal and did not reveal any procedure related complication. This case of foreign body is unique in the sense that the large chicken bone had been lodged in right bronchus intermidus remaining asymptomatic for 2 months and radio-logically found as a stent inside the bronchus.

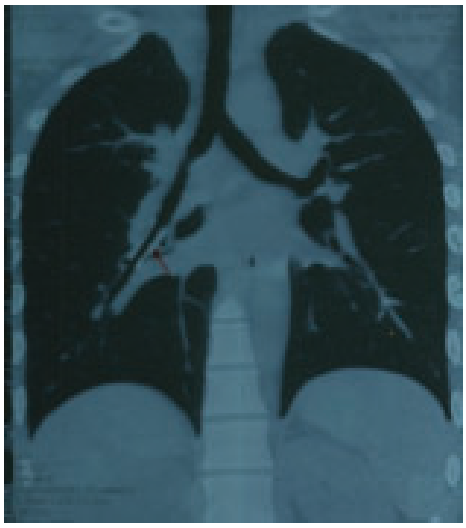

Fig 3: CT thorax, coronal section showed position of the bone

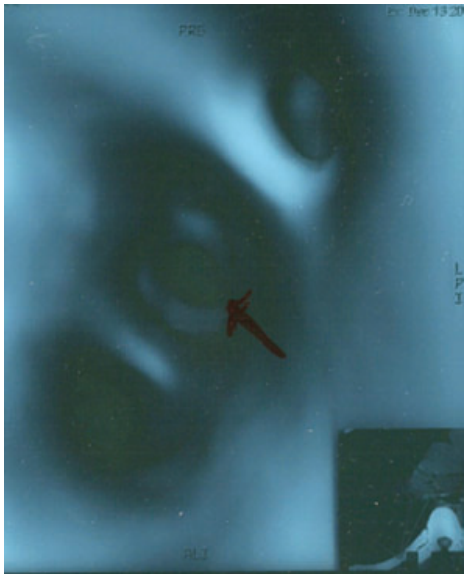

Fig 4: Virtual bronchoscopy showed location of bone in right bronchus intermedius

DISCUSSION : Foreign bodies in airways are most commonly seen in young children usually below 15 years[1] and most of the reported cases are below 2 years[1,2]. In 2000 US has reported 160 death and 17,000 emergency visit of children below 14 years due to foreign body of airways[3]. In adult foreign body of airway is seen less frequently and also there is delay in diagnosis due to lesser symptoms. The Mayo's clinic reported only 60 cases of adult airway foreign body over a period of 33 years[4]. In adults the type of the FB is more variable than children and mostly organic material like food, chicken, fish bone etc[4,5,6]. In case of children the most reported foreign bodies are nuts and seeds[7,8]. Foreign bodies like nail or pin aspiration mostly seen in young or middle-aged adults[4,9]. Certain conditions namely neurologic disorders, loss of consciousness, alcohol or sedative abuse predispose to foreign body aspiration in adult due to loss of pharyngeal muscle co-ordination. Unlike children adult with foreign body usually present as chronic problem[1,10]. Coughing is the commonest symptom and may be seen in 50 to 80 percent of all cases; other symptoms include fever, hemoptysis, chest pain, wheezeing, chocking etc[2,11]. dyspnea is seen in 16 to 25%[1,10] around 1% of patient remain asymptomatic[1]. Due to lack of symptoms and absent history of aspiration FB may remain undiagnosed for long time, even upto 25 years[1]. If there is no typical choking episode, or presence of a radiopaque foreign body in x-ray diagnosis is often overlooked. Radiograph may show unilateral hyperinflation, atelectasis, consolidation, non specific opacities etc[2,11,12,13]. A foreign body may be an incidental diagnosis while doing x-ray[14] or fiberoptic bronchoscopy to evaluate conditions like chronic cough, hemoptysis, non resolving pneumonia etc.

Diagnosis is based on clinical features, radiology and bronchoscopic findings[1]. Flexible bronchoscopy is considered as gold standard for diagnosis of foreign body. Initially rigid bronchoscopy was used for removal of foreign body. With advent of rigid bronchoscope mortality has decreased from 24 to 2%[15]. However in recent times with the advent of better equipments including foreign body removal forceps flexible bronchoscopy has gained popularity[1]. In a case series of 426 patients, success of flexible bronchoscopy was around 86%[4, 5, 16, 17, 18, 19]. However there must be a facility of rigid bronchoscopy if flexible bronchoscopy fails. In our case since the foreign body was impacted like a stent, rigid bronchoscopy was done.

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Case Report

Adult Onset Mumps Presenting with Asymptomatic Pancreatitis- A Rare Presentation

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ABSTRACT :

Introduction: Mumps is an acute, systemic viral infection classically associated with swelling of one or both parotid glands. Inapparent infections are more common in adults. Mumps pancreatitis presents as abdominal pain, occurs in 4% of infections but is difficult to diagnose.

Case: A 27yr old male patient presented with features suggestive of mumps viral infection. On examination, pt was febrile; with tender swelling of bilateral parotid. Rest ENT examination was normal. Chest, cardiac, CNS examination was normal. Abdomen was soft, non tender, no organomegaly and bowel sounds were present. Redness of bilateral scrotum, tenderness and raised temperature on palpation of testis and pain was relieved on lifting the testis .Lab investigations: haemoglobin-14gm%, TC-12400, DLC-Neutrophils 55%, lymphocytes 34%, monocytes 10%, platelets-1.5lacs, ESR-15 AEFH. RE Urine was normal. Blood urea, serum creatinine, electrolytes and liver function tests were normal. Serum amylase, lipase were raised. USG Doppler of b/l scrotum was suggestive of orchitis with inflamed scrotum. USG and CECT abdomen were suggestive of acute pancreatitis.

Conclusion: Mumps pancreatits is a rare but not uncommon clinical manifestation which can be asymptomatic sometimes and should be sought for during routine blood and imaging investigations.

KEY WORDS : *Mumps, pancreatitis, orchitis*

INTRODUCTION :

Mumps is an acute, systemic viral infection classically associated with swelling of one or both parotid glands, caused by a paramyxovirus with a negative-strand nonsegmented RNA. Mumps is endemic worldwide, with epidemics occurring every 3–5 years in unvaccinated populations. The estimated annual global incidence is 100–1000 cases per 100,000 populations in countries without national mumps vaccination programs, where virtually the entire population has been infected by adulthood. Up to half of mumps virus infections are asymptomatic or lead to nonspecific respiratory symptoms. In apparent infections are more common in adults than in children.[1]

Classical tender parotid enlargement, which is bilateral in 75%, follows a prodrome of pyrexia and headache. Meningitis complicates up to 10% of cases. Approximately 25% of post-pubertal males with mumps develop epididymo-orchitis .Rare complications include encephalitis, transient hearing loss, labyrinthitis, electrocardiographic abnormalities, pancreatitis and arthritis.[2] Mumps pancreatitis, which may present as abdominal pain, occurs in 4% of infections but is difficult to diagnose.[1]

Here we present a case of adult onset mumps that also had asymptomatic pancreatitis, which is a rare manifestation of mumps.

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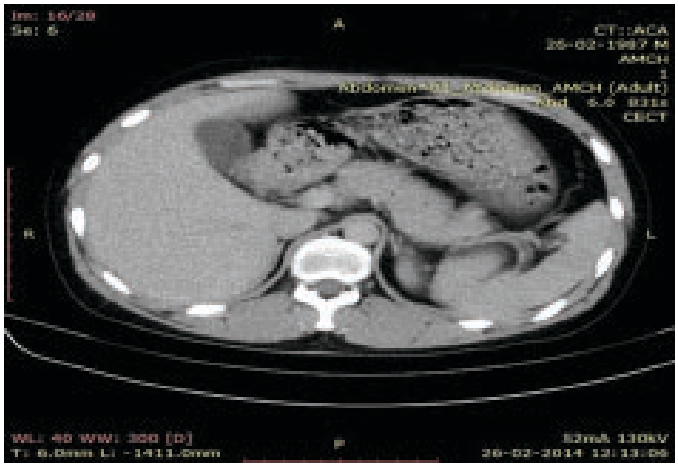
CASE :

A 27yr old married male patient presented with fever, headache, and malaise since 3-4 days; swelling of cheeks since 3days and pain in groin region since 2 days. No history of diabetes, hypertension or tuberculosis. Pt is a non smoker, non alcoholic. Pt was not able to recall the immunization history. On examination, patient was febrile; there was tender swelling of bilateral parotid glands without any discharge or obstruction of parotid ducts. Rest ENT examination was normal. Chest, cardiac examination was normal. Abdomen was soft, non tender, no organomegaly and bowel sounds were present. There was redness of bilateral scrotum and tenderness and raised temperature

on palpation of testis and pain was relieved on lifting the testis. CNS examination was normal. Lab investigations: haemoglobin-14gm%, TC-12400, DLC-Neutrophils 55%,eosinophils 1%,lymphocytes 34%, monocytes 10%, no basophils or atypical cells, platelets-1.5lacs, ESR-15 AEFH. RE Urine was normal. Blood urea-29.5mg/dl, serum creatinine-1.22mg/dl, normal electrolytes and normal liver function tests. Serum amylase-723.7IU/L, Serum lipase-323.5IU/L. USG Doppler of b/l scrotum revealed hyperechoic and thickened scrotal walls with hyperechoic testis suggestive of orchitis with inflamed scrotum. USG abdomen revealed mild bulky pancreas with heterogenous echotexture suggestive of acute pancreatitis. CECT abdomen also revealed the same findings suggestive of acute pancreatitis.



CECT abdomen of patient showing heterogeneous and bulky pancreas body and head suggestive of acute pancreatitis

DISCUSSION:

Mumps, derived from the English verb to mump, meaning to grimace, obtained its name from the most frequently associated physical finding, painful parotid swelling. Mumps has been recognized from the fifth century BC when Hippocrates described the disease as one of swellings behind the ears accompanied by swelling of the testes. However, the first description of neurological involvement was that by Hamilton in the eighteenth century. Transfer of disease from filtered secretions of an affected patient into experimental animals suggested the disease had a viral aetiology.[3]

Mumps is caused by a paramyxovirus with a negative-strand nonsegmented RNA genome. The nucleoprotein, phosphoprotein, and polymerase protein participate in viral replication and, together with genomic RNA, form the ribonucleocapsid. The ribonucleocapsid is surrounded by a host-derived lipid bilayer envelope containing the viral hemagglutinin-neuraminidase (HN) and fusion (F) proteins, which are responsible for cell binding by and entry of the virus and are major targets of virus-neutralizing antibodies. [1]

The incubation period of mumps is 19 days (range, 7–23 days). The virus is transmitted by the respiratory route via droplets, saliva, and fomites. Mumps virus is typically shed from 1 week before to 1 week after symptom onset. Persons are most contagious 1–2 days before onset of clinical symptoms. Classic sites of mumps virus replication include the salivary glands, testes, pancreas, ovaries, mammary glands, and central nervous system.

Up to half of mumps virus infections are asymptomatic or lead to nonspecific respiratory symptoms. Inapparent infections are more common in adults than in children. The prodrome of mumps consists of low-grade fever, malaise, myalgia, headache, and anorexia. Mumps parotitis—acute-onset unilateral or bilateral swelling of the parotid or other salivary glands lasting >2 days without another apparent cause—develops in 70–90% of symptomatic infections, usually within 24 h of prodromal symptoms but sometimes as long as 1 week thereafter. Parotitis is generally bilateral, although the two sides may not be involved synchronously. Epididymo-orchitis is the next most common manifestation of mumps, developing in 15–30% of cases in postpubertal males, with bilateral involvement in 10–30% of those cases. Orchitis, accompanied by fever, typically occurs during the first week of parotitis but can develop up to 6 weeks after parotitis or in its absence. The testis is painful and tender and can be enlarged to several times its normal size; this condition usually resolves within 1 week. Testicular atrophy develops in one-half of affected men. Sterility after mumps is rare, although subfertility is estimated to occur in 13% of cases of unilateral orchitis and in 30–87% of cases of bilateral orchitis. Oophoritis occurs in 5% of women with mumps and may be associated with lower abdominal pain and vomiting but has only rarely been associated with sterility or premature menopause.

Mumps pancreatitis, which may present as abdominal pain, occurs in 4% of infections but is difficult to diagnose because an elevated serum amylase level can be associated with either parotitis or pancreatitis. An etiologic association of mumps virus and juvenile diabetes mellitus remains controversial.[1]

The pancreas is usually involved 3 to 5 days after the salivary glands, and may be the only organ clinically affected. Hemorrhagic pancreatitis was reported to be seen at surgery in a 22-month-old child whose sibling developed the disease 2 days later.[4]

In 1817 mumps virus was implicated for the first time as a cause of acute pancreatitis. Since then the literature contains many reports associating viral infections and pancreatitis. The clinical picture of these patients is characterized by abdominal pain, diarrhea and the outcome of the disease is self limited[5] .

The vast majority of reports does not contain adequate data either for the diagnosis of pancreatitis or for mumps infection. In a prospective study including 116 patients with a definite diagnosis of acute pancreatitis, 3 of them suffered from mumps infection as determined by antibody detection.[6] A few other sporadic cases in the literature show the possible association between acute pancreatitis and mumps-virus. It is very interesting to emphasize that following vaccination of the general population for measles, mumps, rubella (MMR vaccine), only one case of mumps virus associated acute pancreatitis has been described in the literature.[7]

CONCLUSION :

In this case the patient had no symptoms of pain abdomen related to pancreatitis itself. The raised amylase and lipase with radiological findings suggestive of pancreatitis could not be explained by any other common cause of acute pancreatitis and further in the background of the mumps active infection, this was sought to be the probable cause of pancreatitis in this case.

Thus all patients presenting with mumps especially adults should be sought for abdominal symptoms and routine lab investigations should include both amylase and lipase, also a screening USG abdomen with special interest to pancreas can be done, even if the patient is asymptomatic. Though incidence is less but screening is worth considering possibility of pancreatitis.

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