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EDITORIAL

The Journal of Current Medical Diagnosis and Research, an official journal of Ayursundra Super Speciality Hospital, is one of the very few journals published from a private hospital with an aim to maintain a high publication standards. The journal strives to publish as many articles from the medical community as possible. However, it has been observed that in spite of the huge amount of good quality clinical work being done in the country, the number of manuscripts from Indian authors is few.

Most research involves a team effort, and some junior members of the team who may be playing a substantial or lead role may not be aware of the ethical issues involved in human research though all researchers are equally abide by ethical principles. Authors must know that irrespective of where or how a research is done, patients confidentiality, right to anonymity, and privacy is of paramount importance. During the whole course of the conduct of the research till its final submission for possible publication, the authors must ensure that any information which reveals patient identity such as name and hospital number is avoided.

Authors must also understand that patients consent, given for medical management or surgery by the caregiver, should not be assumed to be blanket consent. A separate informed consent must always be taken if the researchers plan to use patient data for research purposes. Moreover, approval from an Institutional Review Board, Ethics Committee, Departmental Board of Study, or an equivalent authority of competence is mandatory for any human research.

Most reputed journals including the current journal have a robust system of peer review. Peer review ensures that only the highest quality research is published, and errors and oversights corrected. Following the review process, most authors would be asked to do a major or minor revision of their work. As an ethical rule authors must take the reviewers comments positively and must answer the concerns raised systematically and within the stipulated time frame. Sometimes, queries might be raised by the editorial team even after a manuscript has been accepted and authors must again respond to them quickly. Once a manuscript is published, queries may be raised by the readers formally in the form of letter to editors of informal communication. Again it is the authors' moral duty to respond to these questions. All records and data pertaining to the study must be maintained for a sufficient period of time so that they can be produced anytime for scrutiny if desired by readers, other researchers, agencies, or editorial teams.

If all members of a research team follow these words in letter and spirit, a number of ethical issues which crop up during the conduct and publication of medical research, especially involving human participants, would be taken care of automatically.

It is our hope that readers find this journal to be a worthwhile addition to office libraries, to medical reference areas and to the pockets of their lab coats. We always welcome your feedback. Please contact us at anytime at wecare@ayursundra.in with questions you may have or ideas you would like to contribute.

Dr. Partha P. Kalita

CONTENTS

ATRIAL FIBRILLATION AND ANEMIA

Dr Aditya Bhaskar, MD, DM CARDIOLOGY
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Atrial fibrillation (AF) is one of the most common form of cardiac arrhythmia in adults. Its prevalence increases with age and reaches up to 12% in those older than 80 years [1]. Patients with AF often have multiple cardiovascular comorbidities like chronic heart failure, valvular heart disease, hypertension, stroke, and diabetes mellitus. AF is independently associated with increased risks of ischemic stroke, hospitalization for heart failure, and mortality [2]. Cardiovascular comorbidities and thromboembolic events significantly increase the mortality rate and treatment cost of AF.

Anemia, which is a risk factor for cardiovascular disease, is independently associated with an increased mortality rate in patients with chronic heart failure, left ventricular hypertrophy, chronic kidney disease, diabetes mellitus, and acute coronary syndrome.[3,4,5]

Coexistence of anemia with AF not only makes the management of AF further complicated it also increases the morbidity and mortality associated with it.

PREVALENCE OF ANEMIA IN ATRIAL FIBRILLATION-

Prevalence of anemia in atrial fibrillation range from 12-30% in different studies. In Randomized Evaluation of Long-Term Anticoagulation Therapy (RE-LY) trial database 12% of the AF patients were anemic at baseline.[6] Anemia was defined as a hemoglobin concentration of <12 g/dL for women and <13 g/dL for men, according to the definition of the WHO. During the course of the study, average hemoglobin levels gradually decreased and the proportion of patients with anemia increased accordingly . Clinical factors associated with anemia in AF patients were higher age, diabetes, chronic heart failure, chronic kidney disease and the use of aspirin. During the course of the study, patients with anemia spent more time below the therapeutic range than those without anemia, whereas the times above the therapeutic range were comparable.

On the other hand in AFCAS (Atrial Fibrillation undergoing Coronary Artery Stenting) registry 30% AF patients were anemic.[7] Similar to RE-LY trial database anemic patients were older; more likely to have diabetes mellitus, hypertension, history of heart failure and chronic renal impairment.

ANEMIA AND NEW ONSET AF-

Anemia is an independent risk factor for new-onset AF. Anemia causes decreased oxygencarryingcapacity, which is compensated byincreasing the heart rate and stroke volume. This, in turn leads to cardiac overloadwith an increase in myocardial mass as well asincreased neurohormonal and sympathetic activity.It also leads to increased levels of vasopressin and endothelin, all ofwhich can cause arrhythmogenic remodeling susceptible toAF.[8] Because patients with anemia often have co-morbidities, it isdifficult to clearly provide true risk of anemia alone for AFonset, and anemia may act as a synergic factor with other conditions for AF onset.

CLINICAL CHARACTERISTICS OF ANEMIA WITH AF-

The Fushimi AF registry which represents the clinical profile of real-world AF patients found that AF patients with anemia areusually olderthan those without anemia and they are more likely to have various co-morbidities like heart failure, coronary artery disease, peripheral artery disease, chronic kidney disease, and history of major bleeding.[9] However no significant difference in the prevalence of hypertension or diabetes was found between anemic and non-anemic AF patients. Anemic AF patients have greater CHADS2 score and CHA2DS2-VASc score and higher prevalence of previous stroke .Patients receiving the prescription of oral anticoagulants are less in anemic patients and the vast majority of them receive warfarin .The study concluded that AF with anemia is associated with higher prevalence of stroke as well as bleeding, irrespective of the presence of other comorbidities.

Anemia as a predictor of thromboembolic events in AF-

Anemia has been identified as a predictor of thromboembolic events in AF. Retrospective analysis of the RE-LY trial database found that anemia was present in 12% of the population at baseline, and the presence of anemia was associated with a higher risk of thromboembolic cardiovascular events.[6] Increased incidence of thromboembolic events in anemic patients reflect the increased rates of discontinuation of oral anticoagulants (OACs)and suboptimal warfarin dosing in these patients. Multiple studies have shown that OACs produce a net clinical benefit in AF patients at risk of stroke, irrespective of their risk of bleeding.[10,11] current guidelines therefore recommend OAC in all patients of AF who are at increased risk of stroke irrespective of their bleeding risk.[12]

Anemia as a predictor of bleeding complication in AF-

The presence of anemia at baseline was associated with a 2.2-fold increase in the incidence of major bleeding in RE-LY trial database.[6]Anemia remained a predictor of bleeding complications, even after adjustment for the HAS-BLED score. Anemia at baseline also predicts temporary and permanent discontinuation of the OACs. A significant interaction for age and bleeding complications was also noted in RE-LY database. Although anemia predicted bleeding complications in both age groups, the strength of the association was consistently higher in younger patients.[6] So in anemic patients, more intense monitoring of anticoagulation is appropriate to reduce the bleeding and thromboembolic events.

Anemia as a predictor of mortality in AF-

Several studies have investigated the relationship between anemia and mortality in patients with AF. In a study by Sharma *et al.* hematocrit level was an independent predictor of all-cause mortality in elderly patients with AF.[13] In AFCAS registry by Puurunen *et al.it was* revealed that, AF patients who undergo percutaneous coronary intervention, major adverse cardiac, cerebrovascular, and bleeding events are more likely in those with anemia compared to those without anemia.[7] At 1-year follow-up of AFCAS registry it was revealed that anemia was an independent predictor of all-cause mortality in AF patients.

Anemia affects the outcome of AF patients by multiple mechanisms. First Anemia leads to increased cardiac workload leading to adverse cardiac remodelling. Second it further exacerbates the ischemia in patients with pre-existing coronary artery disease.

Impact of anaemia on AF patients undergoing PCI-

In AFCAS registry anemia was an independent predictor of all-cause mortality in multivariate analysis.[7] The higher rate of all-cause mortality might be related to the higher risk profile in anemic patients, as well as the underlying disease causing anemia. The rate of definite or probable stent thrombosis was also significantly

higher in anemic patients. As patients with anemia more often present with ACS as compared to those without anemia, it may contribute to higher incidence of stent thrombosis. In addition presence of anemia may also influence the choice of antithrombotic medication and a bleeding event could lead to the interruption of antithrombotic therapy, and thus predisposes the patient to a higher risk of stent thrombosis. Interestingly in a study by Toma C et al, anemia was the only independent predictor of high residual platelet reactivity on clopidogrel in a series of patients undergoing PCI.[14]

Anticoagulation in presence of anemia- A double edged sword-

Anemia is a strong predictor of bleeding in anticoagulated patients with AF, and it is included in various bleeding risk algorithms.[6] On the other hand, anemia is also a strong predictor of thromboembolic events, including stroke. In this way, anemia identifies patients who are at increased risk of thromboembolism who could benefit most from treatment with OACs, but who are also more prone to bleed.[6] Together, these findings suggest that, in anemic patients, it is close monitoring of anticoagulants that is needed, rather than dose adjustment or discontinuation.

So instead of discontinuation of OACs, the occurrence of anemia should prompt a detailed diagnostic assessment of the etiology of anemia and it should be treated accordingly. Other precipitating causes of bleeding, such as concomitant antiplatelet or anti-inflammatory drugs or uncontrolled hypertension should be managed.

A more reassuring fact is that resolution of anemia is associated with partial normalization of prognosis. Anemia is, therefore, a modifiable risk factor, which suggests that prevention or treatment of anemia could improve the prognosis in patients with AF.

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MOVEMENT DISORDER EMERGENCY IN PARKINSON'S DISEASE- A REVIEW

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ABSTRACT : Parkinson's Disease is common neurodegenerative disorder with motor and non- motor symptoms and complications . Acute or sub-acute onset movement disorders are common in Parkinson's Disease during the course of disease process and treatment. Failure to accurately diagnose and manage the patient may result in significant morbidity or even mortality. In this review, author covered situations with relevant Movement disorder emergencies related to Parkinson's disease.

Movement disorders are characterized by involuntary movements and/orloss of control or efficiency in voluntary movement . Patients with Parkinson's disease, most cases are followed and treated in outpatient care settings. A movement disorder emergency is defined as a neurological disorder evolving acutely or sub-acutely, in which the clinical presentation is dominated by movement disorder and in which failure to accurately diagnose and manage the patient may result in significant morbidity or even mortality.[1]

Major movement Disorder emergencies in Parkinson's Disease :

1. **THE SUPER OFF PHENOMENON AND MOTOR FLUCTUATIONS:** Motor fluctuations (MF) develop in 70% of Parkinson's disease patients treated with levodopa for nine years or longer and in nearly all patients with early onset PD after less than ten years of treatment.[2] The definition of MF is closely related to the definition of ON and OFF periods. An ON period is the time of levodopa responsiveness, with adequate control of motor symptoms, while an OFF period is the time when parkinsonian symptoms are significantly present. Both usually correlate directly or indirectly with levodopa plasma level, however the mechanisms that lead to MF are complex and involve the reduction of striatal synthesis and storage of dopamine from exogenous levodopa and sub-sensitization of postsynaptic dopaminergic receptors.[2]

   The encountered phenomenon in management of Parkinson's Disease :

C. THE "WEARING OFF"PHENOMENON:

It describes the presence of end dose OFF periods, mainly due to the loss of striatal dopamine storage capacity and short levodopa half-life. [2]

B. ON –OFF PHENOMENON AND UNPREDICTABLE OFF: It describe the occurrence of sudden and disabling OFF periods with no clear relationship with timing of levodopa intake. The "delayed ON" (increased time latencies from dose intake to the start-up of an ON period) and "no-ON" phenomenon (complete failure of a levodopa dose to exert an ON response) are typically related to impaired absorption of oral levodopa.[2]

In rare instances, MF can represent a MD emergency, especially in the case of frequent and distressful ON-OFF periods.

Typically, management involves various combined approaches, such as administration of small multiple

daily doses of levodopa, controlled release, dispersible and soluble levodopa formulations, subcutaneous dopamine agonists, MAO-B and COMT inhibitors, and surgical approaches. Administration of crushed levodopa tablets may also be helpful in certain circumstances[2]

C. **THE SUPER OFF PHENOMENON**: It is a rare complication found in patients with PD defined as an increase in motor disability scores in comparison to basal (OFF period) motor scores described in those experiencing MF.[3] As the denomination implies, it means that patients affected by this phenomenon have the sensation of being more symptomatic from most parkinsonian motor standpoints than their usual OFF levodopa dose status. The super OFF state can be divided into a **beginning-of-dose** and an **end-of-dose inhibitory effect**, depending on whether it occurs before or after levodopa has reached a therapeutic plasma concentration.[4] The phenomenon is often very distressing and disabling, lasting from minutes to hours. The pathophysiology of super OFF episodes is not entirely clear, but is probably linked either to presynaptic inhibition of firing and release of dopaminergic neurons, or an effect mediated by the preferential stimulation of a subpopulation of postsynaptic DA receptors that have an inhibitory action on motor activity caused by low levels of levodopa in the rising or declining phase of the plasma concentration curve.[3]

2. **PARKINSONISM HYPERPYREXIA SYNDROME (PHS)**: It is also known as neuroleptic malignant-like syndrome, is a rare complication of PD, characterized by hyperthermia, autonomic dysfunction, altered consciousness, severe rigidity and elevated serum creatine kinase (CK) levels.[6] PHS may be triggered by infections, reduction in dopaminergic drug dosage, hot weather or dehydration. Potentially life-threatening complications include deep venous thrombosis and pulmonary embolism, aspiration pneumonia and renal failure. Treatment is based on intravenous fluids infusion, antithermics and use of levodopa and dopamine agonists. Occasionally, dantrolene may be necessary.

3. **DYSKINESIA-HYPERPYREXIA SYNDROME** : it consists of severe dyskinesias (dyskinetic status), leading to muscle exhaustion, rhabdomyolysis, hyperthermia and confusion.[7] This complication shares some of the clinical characteristics of PHS, but differs in those dyskinesias – instead of rigidity, Dyskinesia dominates the clinical picture. Additionally, the dyskinesia-hyperpyrexia syndrome, contrarily to PHS, should be treated by reducing the dosage of dopaminergic drugs, particularly dopamine agonists.

4. **ACUTE ONSET PARKINSONISM** : Acute severe de novo parkinsonism is an uncommon form of parkinsonism that occurs over hours or days. The most frequent cause is exposure to dopamine-blocking agents, such as neuroleptics and anti-emetics, however other rare causes have been described, including hypoxic-ischemic encephalopathy, intoxications (organophosphate pesticide, carbon monoxide and drugs used for cancer therapy, as cytosine arabinoside, cyclophosphamide and amphotericin) and infections (viruses-similar to Von Economo's encephalitis lethargic, Japanese B encephalitis and neurocysticercosis).

   Additional medical conditions, as urinary or respiratory infections and metabolic disturbances (hypothyroidism a), or even due to a concomitant neurological disease, for example, subdural hematomas, compressive spinal cord lesion or brain tumor can cause acute set back in clinical status of parkinson'sdisease patient .

   Treatment of both de novo and worsening of PD should be focused on resolution of the etiologic process and occasionally symptomatic treatment of parkinsonism using dopaminergic agents.[8]

5. **NEW ONSET PSYCHOSIS** : The acute occurrence of psychosis is frequently associated and triggered by the same agents used to treat the motor symptoms of PD (levodopa, dopamine agonists, anticholinergics, amantadine, COMT and MAO-B inhibitors), however other comorbidities, such as infections (urinary and respiratory tract), metabolic or neurological disturbances, may play a role.The manifestations typically include visual hallucinations, persecutory delusions, confusion and psychomotor agitation. After treatment of

potential comorbidities, management recommendations begin with the stepwise withdrawal of potentially associated drugs, starting with anticholinergics, followed by MAO-B inhibitors, dopamine agonists, amantadine and COMT inhibitors. Frequently, the introduction of a second generation antipsychotic,as clozapine or quetiapine, is necessary. Other options include risperidone or olanzapine, with a significant risk of motor symptoms worsening.[8].

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AMEBIC COLITIS: AN OLD SAGA RETOLD THROUGH THE LENS OF A GASTROENTEROLOGIST'S COLONOSCOPE

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INTRODUCTION:

Entameba histolytica isa parasite which is found globally. Most of its morbidity and mortality occurs in the Indian subcontinent, Africa, Central and South America. It is estimated that E histolytica causes up to 50 million symptomatic cases and up to 10000 deaths annually. The primary organ of involvement is the large bowel whereas extra-colonic disease occurs as a consequence of haematogenous spread from the bowel.

The definitive diagnosis of invasive amebiasis remains the demonstration of the trophozoite form in the biopsy specimens or in the stool specimens.

Bloody diarrhea and abdominal pain are dominant symptoms. However non bloody diarrheais not a well-recognized clinical presentation.

AIM:

To study the sites of involvement of colon in invasive colonic amebiasis and identify any pattern(s) or features on colonoscopy which could be considered as characteristic/pathognomonic of the disease.

To evaluate if the pattern of distribution of colonic lesions in invasive colonic amebiasis was related to the clinical presentation

METHODS AND MATERIALS:

The study was conducted from November 2019 till December 2020. Patients who underwent colonoscopy during the study period and were diagnosed as confirmed amebiasis were retrospectively evaluated regarding the clinical presentation and distribution of lesions

Inclusion criteria:

Patients who had Colonic ulcers and ulcer biopsy showing trophozoites of E histolytica

The data collected included the presenting symptoms ulcer location, number, approximate size and the ulcer morphology.

Statistics: Fischer's exact test was used to analyse the difference between discrete variables in the two groups

RESULTS:

A total of 34 cases were included in the study. 31 patients underwent colonoscopy and 3 had limited sigmoidoscopy.

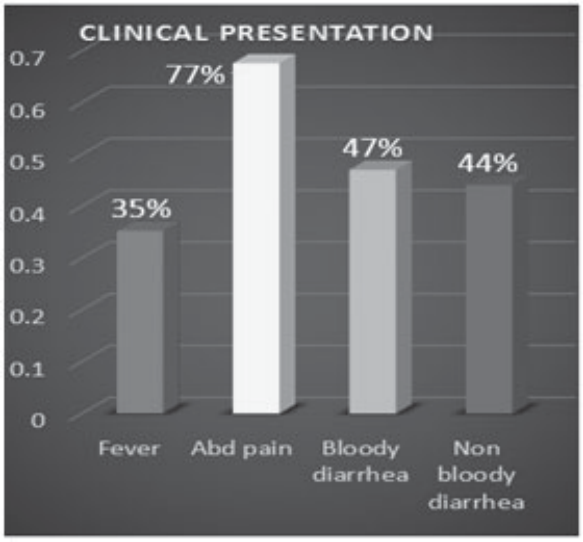

Fig: 1 Clinical presentation of Amebic Colitis

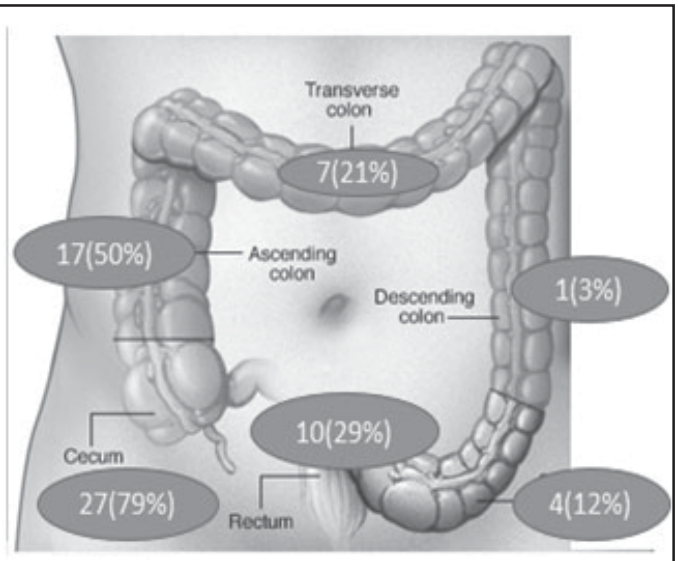

Fig: 2 Colonic Distribution of Amebic Ulcers

The median age was 50 years (9-82) and 61.8% were males. Fever abdominal pain and non-bloody and bloody diarrhea were present in 12 (35%) 23(77%), 15(44%) and 16(47%) respectively.(Figure 1)

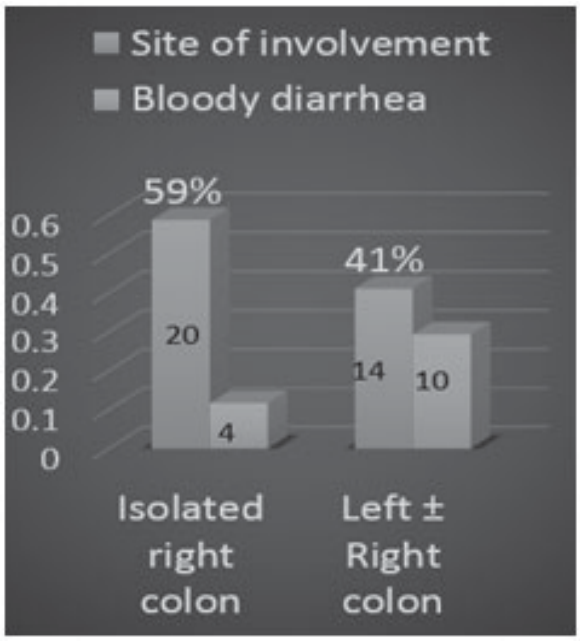

Fig: 3 Presentation related to site

Site	No(%)	Bloody diarrhea	P value
Isolated right colon	20(59)	4(20%)	0.009
Isolated Left colon ± right colon	14(41)	3(71%)	

Fig: 4 Statistical significance of Presentation related to site

Of the 31 patients who had colonoscopy 27 showed cecal involvement. 17, 7 and 10 patients had concomitant ascending colon, transverse colon and rectal involvement respectively. 4 patients had sigmoid and 1 had descending colon involvement. (Figure 2)

Isolated Cecal involvement was seen in 20(59%) of cases whereas 14(41%) patients had lesions of left sided colon with or without involvement of the cecum. Only 4 of 20 patients with isolated cecal involvement had

bloody diarrhea as compared to 10 of 14 patients with left sided disease. (Figure 3) Association of bloody diarrhea with left sided lesions was found to be significant (P= 0.009). (Figure 4)

The ulcers showed clustering of lesions in 33/34 cases. (Figure 5 , 6, 7)Most ulcers 26/34 were less than 1.5cm in size. The ulcers looked like poached eggs in 10/34 cases. (Figure 8)

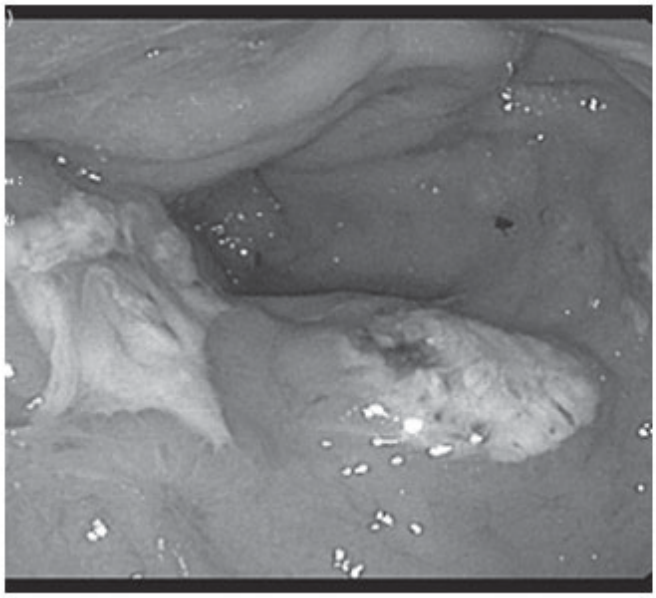

Fig: 5 Amebic ulcer – Multiple large with extensive exudate

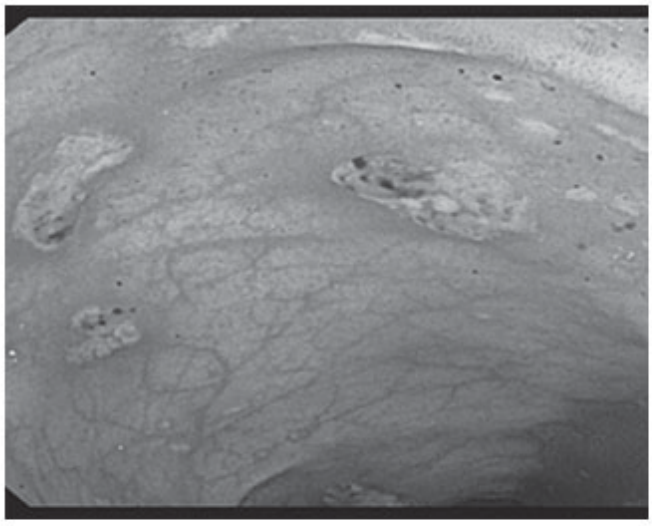

Fig: 6 Amebic ulcer – Clustering of lesions

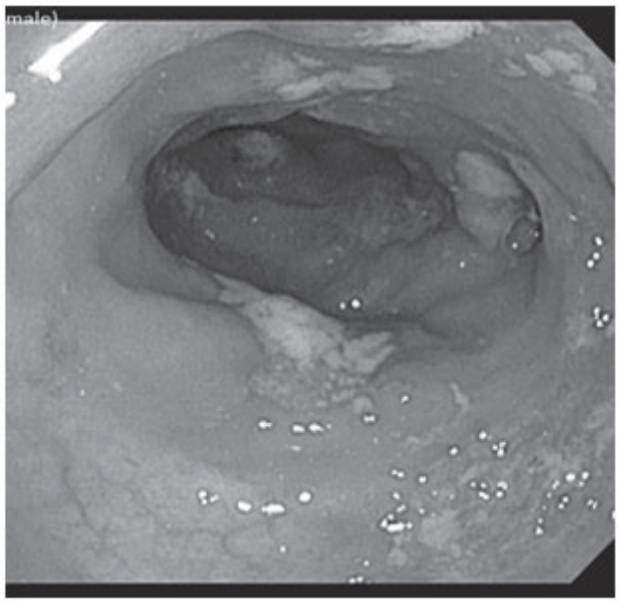

Fig: 7 Amebic ulcer – Clustering of lesions

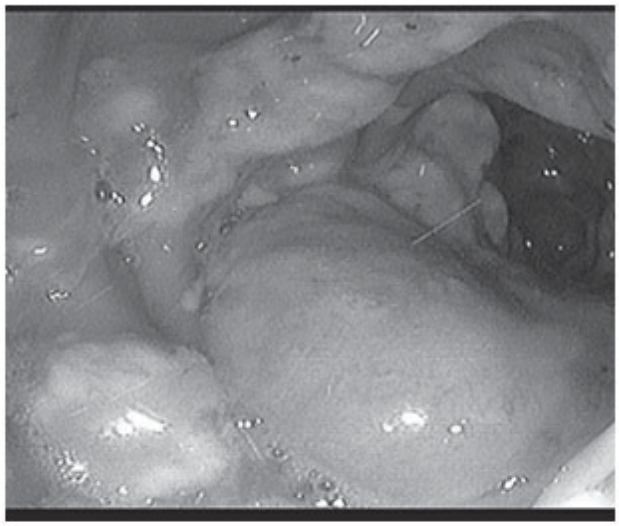

Fig: 8 Amebic ulcer-Poached egg appearance

CONCLUSION:

Most common presentation was abdominal pain (77%).Bloody diarrhea (47%) was more commonly seen than non-bloody diarrhea (44%).

Cecum was the most common site of involvement. Rectal or left sided lesions were more likely to present as bloody diarrhea as compared to patients with isolated cecal lesions which mostly presented as non-bloody diarrhea.

The unusual combination of rectal and right colonic involvement with sparing in between also seems characteristic. The appearance of poached eggs to be almost pathognomonic of invasive colonic amebiasis.

ACUTE AORTIC DISSECTION PRESENTING AS ACUTE ISCHEMIC STROKE IN ER

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ABSTRACT

Introduction. Some cases with aortic dissection (AD) could present with various complaints other than pain, especially neurological and cardiovascular manifestations. AD involving the carotid arteries could be associated with many clinical presentations, ranging from stroke to nonspecific headache**.**

Case Report. A 55-year-old man was admitted in our emergency department with drowsiness, unilateral weakness and mild chest discomfort which started within the previous 3 hours and progressed with deterioration of speech. On arrival, he was disoriented and uncooperative. CT brain plain was consistent with acute ischemia in the cerebral hemisphere.Later on, clinical examination by senior consultants in ER revealed asymmetry of pulses.This inturn raised suspicion about aortic pathology which was confirmed by CT aortogram.

1. INTRODUCTION

Acute aortic dissection is one of the most dramatic cardiovascular emergencies unless promptly recognised and treated. Classical acute aortic dissection (AD) has been described as presenting with sudden, severe back or chest pain characterized as ripping or tearing in nature [1, 2]. However, not all ADs present with classic symptoms, and establishing the diagnosis can be difficult when the classic pattern of pain is absent [3]. Aortic dissection is not diagnosed on its initial presentation in 15–43% of cases [4, 5]. Many cases with AD were reported to present with various complaints other than pain, especially neurological and cardiovascular manifestations. Aortic dissection involving the carotid arteries is reported to be associated with many clinical presentations, ranging from stroke to nonspecific headache [1, 6, 7].

The most important point in acute ischemic stroke (AIS) treatment is that it provides reperfusion with an early fibrinolytic therapy[8, 9]. If any contraindication defined in the guidelines does not exist intravenous fibrinolytic therapy should be started immediately in early hours of acute ischemic stroke. One of the absolute contraindications in the guideline is aortic dissection. This case report is aimed at emphasizing the importance of ruling out diagnosis of aortic dissection before fibrinolytic treatment in acute ischemic stroke.

2. CASE REPORT

In this case, a male patient, aged 55 years, with vertigo which started within the last 3 hours and progressed with weakness of arms and altered sensorium following speech disorder, has been brought to the emergency service. His general condition was impaired; the vital status of the patient who was in altered sensorium was evaluated (BP: 116/60 mmHg; pulse: 76 /min; respiration: 26/min, Capillary blood sugar :354 mg/dl). Auscultation of heart and ECG revealed a regular rate and rhythm without any murmurs, and peripheral arterial pulses were feeble. Emergent chest X-raydid not reveal any abnormality.His brain computed tomography was suggestive of acute cerebral infarct. Fibrinolytic therapy had been planned since symptoms started within three hours. Even though there was not any suspicion of aortic dissection in anamnesis and physical examination,

senior consultant in ER was given a call to review the patient before fibrinolysis.On detailed cardiovascular examination, asymmetry of arterial pulses was detected. Trop I was negative. Thereafter CT aortogramwas done and it was consistent withascending aortic dissection which has been expanding from aortic arch to proximal bilateral carotid arteries (Figure 2). Based on these findings the treatment had been changed and cardio thoracic surgical invention was planned.

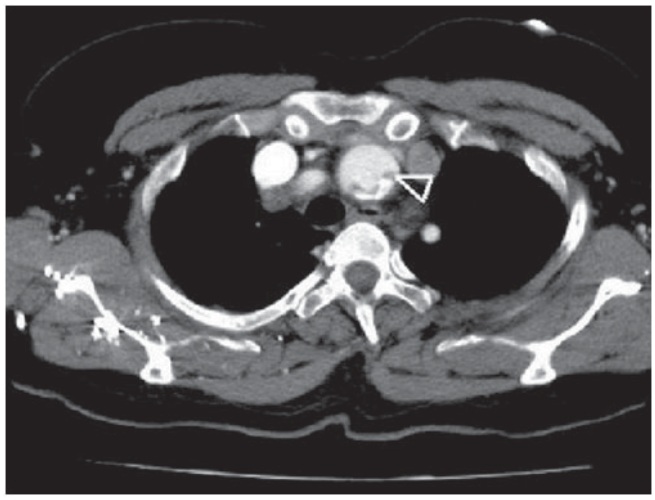

Figure 2

CT scan with contrast enhancement disclosed dissection

3. DISCUSSION

AD is a dramatic medical emergency with a high mortality rate (1% to 2% per hour for 24 hours). AD is most commonly seen in hypertensive people between 50 and 70 years of age and occurs more often in men than in women. Primary neurological presentation is rare, and potentially lethal treatments like fibrinolysis may be initiated, especially in patients presenting with stroke and aphasia [10, 11]. In addition to migrating chest pain (85% of patients) and/or back pain (46%), additional signs such as pulse deficit (30%), hypotension (21%), pericardial effusion (29%), aortic regurgitation (30%), abnormal ECG (69%), and elevated D-dimer may point towards aortic dissection. Side branch involvement of the supra-aortic vessels with dissection of the common carotid or subclavian artery occurs in 15% to 41% of cases [12, 13]. Unrecognized patients with AD would be exposed to lethal complications of fibrinolytic therapy. Common delay in diagnosis of AD which results in higher mortality and narrow therapeutic time window of stroke reflects great diagnostic challenge to emergency clinicians [14].

In our patient risk factors for AD, hypertension and age, which are typical were present. The mediastinum was not enlarged on portable chest X-ray. Additionally, there were no abnormal ECG findings. At first glance AD was not considered because of the lack of signs.

Bedside echocardiography can be performed easily without time delay or transport of the patient, and we consider it a helpful complementary tool for the current diagnostic workup. Emergency physicians are capable of performing basic cardiac scans with focused training.

Stroke is one of the major causes of mortality worldwide. AD may present with predominant neurological symptoms of acute ischemic stroke (AIS) without the typical appearance of chest pain, hypotension, and absent peripheral pulses [11, 15, 16]. Guidelines and similar articles should warn colleagues not to administer fibrinolytics without careful evaluation of signs and symptoms of AD in patients with AIS. This advice was provided in the American Stroke Association Stroke Council's 2010 guidelines for the diagnosis and management

of patients with thoracic aortic disease, as well as in the 2003 guidelines for early management of patients with AIS [17]. Intravenous thrombolysis is the only approved treatment for AIS within 4-5 hours from symptom onset. Systematic investigations of the underlying mechanism of cerebral ischemia have rarely been performed. Because of the narrow time window, the underlying stroke pathogenesis may not be investigated, and therefore careful selection of appropriate candidates may not be performed [18, 19]. Arterial dissection accounts for up to 20% of AISs [20]. Carotid artery dissection (CAD) has been associated with AD, reported in as many as 41% of AD cases [21]. Because of its association with aortic dissection, early recognition of CAD might affect the decision regarding thrombolysis for AIS [22].

Upon suspicion of pulse asymmetry, CT aortogram of aortic root revealed type A AD which involved proximal parts of common carotid arteries.

Intravenous fibrinolytic therapy for AIS is now generally accepted [23]. The US Food and Drug Administration (FDA) approved the use of intravenous rtPA in 1996, partly on the basis of the results of the National Institute of Neurological Disorders and Stroke rtPA Stroke Study, in which 624 patients with AIS were treated with placebo or rtPA within 3 hours of symptom onset, with approximately one half treated within 90 minutes [24]. However, more recent and globally crucial documents regarding stroke therapy do not mention thrombolysis as a medication error in AIS caused by AD [23, 25]. The same is true for recommendations for imaging in patients with AIS published in 2009 [26].

AIS secondary to unrecognized AD may lead to not only inappropriate thrombolysis, but also further worsening of the catastrophe. Colleagues should be repeatedly warned, especially in guidelines, to exclude this possibility quickly [17].

4. CONCLUSION

Thorough clinical examination(cardiovascular) along with bedside echocardiography has a great importance in stroke patients who have aortic dissection risk factors and who are planned to be treated by fibrinolysis treatment. AD, which is one of the reasons of AIS, is a diagnosis which should necessarily be excluded in order to apply fibrinolytic therapy. In patients who are admitted to the emergency department with the altered sensorium and stroke, Aortic dissection should always be kept in mind before initiating fibrinolysis therapy.

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MANAGEMENT OF SUICIDAL CUT THROAT INJURY WITH SUPRAGLOTTIC TRACHEAL AND INTERNAL JUGULAR TRANSECTION: A CASE REPORT

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ABSTRACT

Suicidal or Homicidal Cut throat injuries are one of the emergency conditions managed by Emergency Physicians. Urgent interventions are needed to salvage such patients. If not treated in time, they may lead to death. Immediate resuscitation by securing the airway and prompt control of bleeding along with replacement of volume loss is of utmost importance in preventing complications.

We present a case of suicidal cut throat injury with supraglottic transaction of the trachea and right internal jugular vein. Patient was successfully managed with prompt airway management with endotracheal intubation and emergency room resuscitation and later by tracheal and vascular repair in the Operation theatre. Such presentation of suicidal cut throat injuries is rare and in terms of its mode and outcome is rare and hence is reported.

INTRODUCTION:

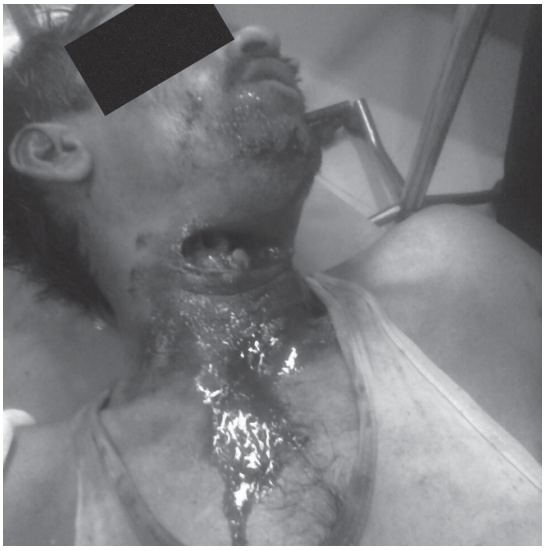
Cut throat injuries are uncommon mode of committing suicides in Indian subcontinent. These injuries are usually associated with multiple hesitation marks [1]. We hereby present a case of suicidal cut throat injury in a patient with history of severe depression and history of multiple attempts of self-harm in the past. Patient was successfully managed with prompt securing of the airway by endotracheal intubation at the Emergency Department and emergency room resuscitation and later by tracheal and vascular repair in the Operation theatre.

CASE REPORT

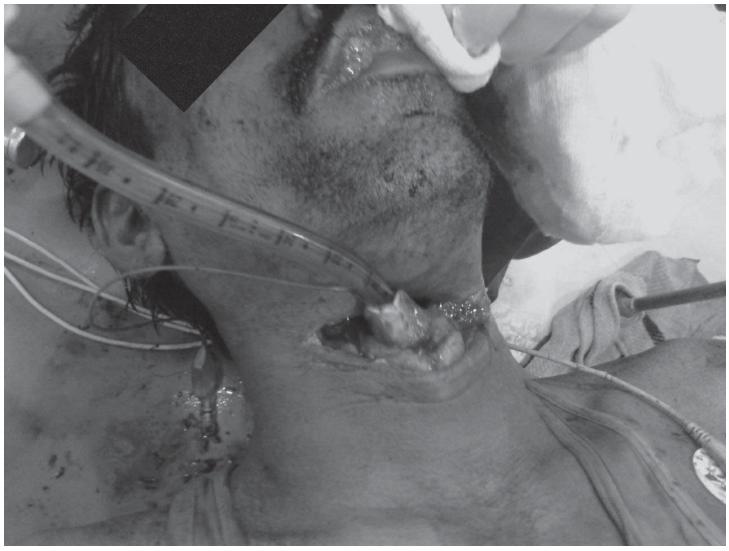
A 45 years old male was admitted to our emergency room in a state of shock and respiratory distress with a deep lacerating wound in his upper 1/3rd of the neck anteriorly after attempting suicide. Patient had a history of being treated for major depression with poor drug compliance and Psychiatry follow up. As per the Police constable, who was an eye witness of the incident of the self-harm, attempt was committed using a hag saw blade which was pulled back and forth by the patient in front of upper part of the neck. His vitals at the time of presentation were: pulse feeble, blood pressure of 70/40 mm Hg, heart rate 160/min and saturation in extremities were not detectable. The patient had aspirated blood and had noisy breathing and was in a gasping state along with torrential bleed from the laceration (Fig 1).

Emergency room resuscitation was started. Airway was secured using a 7.5 size endotracheal tube which was inserted in the distal exposed tracheal end by the of emergency team of doctors (Fig 2). Intravenous crystalloids were started along with intravenous antibiotics, tetanus prophylaxis, pain relief. A right sided femoral central

line was done and blood requisition sent for 6 units of packed red blood cells. The neck laceration was then packed and patient was shifted to the operation theatre after starting the first unit of Packed Cell transfusion.

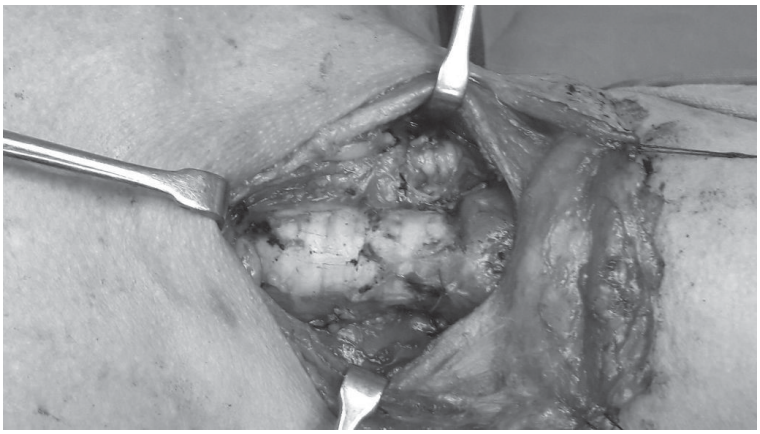


(Fig 1)



(Fig 2)

Emergency neck exploration was started by a team of surgeons under general anaesthesia. Exploration of the neck revealed partial transection of the right internal jugular vein which was clamped and repaired following which haemostasis was achieved. Tracheostomy tube was inserted for better ventilation and tracheal toileting. Wound was thereafter carefully examined which revealed transection of trachea at the level of thyroid cartilage which was then repaired (Fig 3).



(Fig 3)

The patient required admission in ICU for 10 days in his post-operative period owing to a lung infection. However, he responded well to IV antibiotics and was discharged on day 14 of his admission.

DISCUSSION

Injuries following attempts at deliberate self-harm are a common cause of mortality worldwide in the age group of 15-30 years. In the Indian context, incidence is found to be around 38/1,00,000 in this age group. The common modes of suicides in India are consumption of poisonous substance (33.6%), Hanging (31.5%), self-immolation (9.2%) and drowning (6.1%)[1]

Suicidal cut throat injuries with penetrating airway injuries in the neck are relatively uncommon [2]. Mostly suicidal cut throat injuries are very rare. Our patient was a known case of major depression with history of

previous self-harm attempts. As per the eye witness, he had committed suicide by slitting his throat with a hag saw blade on the roadside with many pedestrians. He was brought in to the Emergency Department by a Police personnel who happened to be on duty nearby. There were no hesitation marks seen on the neck which again is an unusual feature of the case and highlights that suicidal cuts may sometimes mimic homicidal cut throats.

Cut throat injuries are usually grievous injuries and carry a high risk of mortality and morbidity due to presence of vital structure in this small part of the body. If the vessels of the neck are injured, patient can rapidly exsanguinate even before reaching the hospital.

Patients with complete tracheal transection can survive for several hours and are salvageable. Hence for securing the airway we did intubation of the patient in the emergency department.

CONCLUSION

Patients with cut throat injuries to neck are challenging in terms of requiring urgent need of securing of the airway and resuscitation along with surgical repair. To deal with such emergencies, requirement of an efficient team of Emergency Physicians trained in airway management and resuscitation is of utmost importance. Such emergencies when are dealt with the concerted efforts of Emergency Physicians, team of Anaesthesiologists and the Surgeons the results are often successful.

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ILEO-SIGMOID KNOTTING IN MID-PREGNANCY COMPLICATED BY INTRA-UTERINE CONTRACEPTIVE DEVICE -A UNIQUE CASE OF ACUTE ABDOMEN.

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INTRODUCTION

Ileo-Sigmoid Knotting is the wrapping of Ileum or Sigmoid Colon around the other one and it's mesentery causing a Double loop obstruction.It is a rare life-threatening condition, with mean mortality rate of 20-100%. Most common cause of death is Shock- due to bowel perforation or gangrene. Herein we report a unique case of Intestinal Obstruction due to ISK, in A Multi Para lady at 16th week of pregnancy complicated by Intra-uterine Contraceptive device who underwent Emergency Surgery.

Unfamiliarity with the disease, no specific blood test for diagnosis, atypical radiograph demonstrating - dilated Sigmoid colon,and multiple small intestinal air-fluid levels, clouded by normal pregnancy complaints make pre-operative diagnosis a rare. However, prompt diagnosis and early surgical intervention significantly improves outcome.

CASE REPORT

A 28year Young Multi -Para at 16 week of pregnancy, presented to the Accident & Emergency Department,Ayursundra Super-Speciality Hospital with severe acute abdominal pain of less than 16 hours of duration, that awakened the patient from sleep. Patient precisely gave the time of onset of pain, after which the pain became constant in nature. Pain was associated with abdominal distension and vomiting. At presentation patient was in severe distress, dehydrated, tachypneic and tachycardia.

USG showed -Free fluid in the pelvis ?Hemoperitoneum. Increased fetal heart rate with decreased fetal movements.

X- ray showed- Disproportionately dilated small bowel loops with multiple air fluid level.

Obstetric history- Gravid 2, Para-1. ‘

Lab Investigations- Total Count -25000/mm, Serum Creatine 1.5

No significant surgical or medical history.

Patient was initially resuscitated with IV fluid, started on Inj. Piperacillin tazobactam and Metronidazole and taken up for laparotomy 5 hours after admission .The peritoneal cavity contained about a litre of foulsmelling dark fluid.Therein, a 360 degree volvolus was encountered with several loops of necrotic small bowel wrapped around a necrosed sigmoid colon.The volvolus was detorsed. Around 100 cm of gangrenous distal ileum upto 5cm proximal to Ileo-caecal junction and 20 cm of gangrenous sigmoid colon was resected. An end ileostomy was performed and colo-colic anastomosis was done.A gynaecological consultation was performed intraoperatively but caesarean section was not considered since it was too early.

Patient was haemo-dynamically stable on Post operative day 1 and clear liquid was started orally. Patient

delivered a dead fetus spontaneously on Post Op day 2.On day 10 patient was discharged and Ileostomy closure was done after 3 months by an Ileo-ascending colon(end-to-end) anastomosis

DISCUSSION

Intestinal obstruction in pregnancy is rare with its incidence varying from 1 in 1500 to 1 in 66,431 deliveries(1). Adhesions are the most common cause of intestinal obstruction in pregnancy, accounting for 58% of cases whilst volvulus is the second most common cause accounting for 24% of intestinal obstructions in pregnant patients (1). ISK is a rare cause of intestinal obstruction and its incidence is not known.(2)

Three factors are responsible for the ileo-sigmoid knot : a long small bowel mesentery and freely mobile small intestine, a long sigmoid colon on a narrow pedicle and finally the ingestion of a high bulky diet in the presence of an empty small bowel. When a semi liquid bulky meal progresses into proximal jejunum it increases the mobility of intestine and the heavier segments of proximal jejunum fall into left lower quadrant. The empty loop of ileum and distal jejunum twist in a clockwise rotation around the base of narrow sigmoid colon.

Further peristalsis forms an ileo-sigmoid knot with two closed loop obstruction, one in the small bowel and other in the sigmoid colon.

Late pregnancy is a predisposing risk factor for ISK due to the obvious displacement of the bowel (3).

In the present case, the patient was in early pregnancy; thus, formation of the ISK in this case was not related to displacement of the bowel resulting from an enlarged uterus. One possible risk factor for ISK in the present case was an unbalanced oral intake owing to morning sickness during the pregnancy, together with the anatomic bowel features mentioned above.

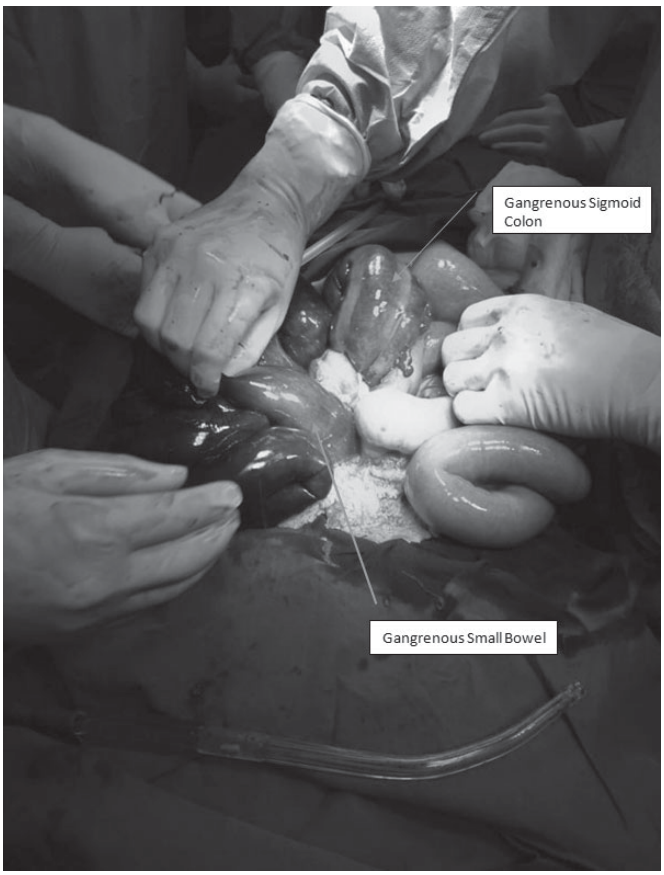
While an increasing number of cases are reported in literature, these are mostly retrospective case reports, thus our knowledge of the evolution of this condition remains limited.The largest reported series in the literature is that of Shepherd(4), in Uganda, with 92 cases in 1967.Atamanalp et al(5) presented a retrospective review of 73 cases in Turkey. more recently Mbanje et al reported a case series of 21 cases at Zimbawe.

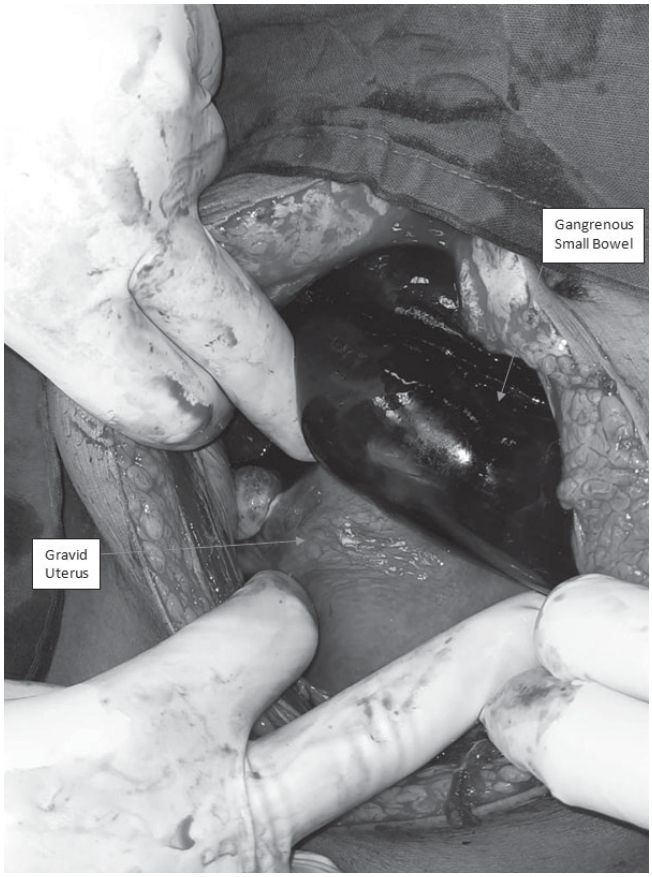
Normal pregnancy complaints may obscure the clinical presentation of ISK in pregnancy. Symptoms include colicky abdominal pain, abdominal distension, constipation and vomiting. If the bowel is gangrenous there is tenderness, guarding and rebound tenderness. The current case presented with the above symptoms of gangrenous bowel

In most instances, the diagnosis is made at laparotomy as there are no recognised pathognomonic symptoms, signs, or radiological features to allow for a definitive preoperative diagnosis.(6)The combination of clinical features of small bowel obstruction with radiological features of large bowel obstruction have been described as contradictory and contributing to diagnostic uncertainty. In the current case the diagnosis was made at laparotomy.

Management of ISK requires a multidisciplinary approach involving general surgeons, obstetricians and neonatologists. Preoperative fluid resuscitation, electrolyte balance correction, intravenous broad spectrum antibiotics and nasogastric decompression are the initial management followed by emergency surgery in all patients Surgical options depend on the intraoperative findings. In non-gangrenous cases untwisting the knot combined with a volvulus preventing procedure such as mesopexy or resection and primary anastomosis is acceptable management. In cases where both the small bowel and sigmoid colon are gangrenous, untying the knot maybe difficult and rupture of the gangrenous bowel may lead to spillage of toxic bowel contents. In our patient we resected the gangrenous small bowel and sigmoid colonan end ileostomy was performed and end-to-end colo-colic anastomosis.

In the diseases leading to bowel necrosis, such asIleo-sigmoid knot, prompt management of thepatient is of vital importance. However, avoidanceof CT radiography in pregnant patients is anobstacle to establish an early diagnosis of thepatients .In the present report, we aimed topresent a rare case of ISK in a 28-year-old, 16-week pregnant woman under the light of relevantliterature, which reminds us the maxim "Acuteabdomen is full of surprise" and which gave us theopportunity to confirm that early treatment saveslife.





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ROLE OF THORACOSCOPY IN UNDIAGNOSED PLEURAL EFFUSION

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ABSTRACT

BACKGROUND:

Differential diagnosis of pleural disease is often a lengthy process fraught with pitfalls. Contrary to thoracocentesis and closed pleural biopsy, thoracoscopy permits biopsy with direct visualization. Medical thoracoscopy increases the diagnostic yield in patients with pleural disease when thoracocentesis and closed pleural biopsy are non diagnostic.

AIMS &OBJECTIVES:

This study investigated diagnostic yield of medical thoracoscopy for undiagnosed pleural effusions.

MATERIALS AND METHODS:

This is a observational study conducted in Department of Pulmonary from September2017 to August 2018 after ethical committee clearance. Twenty patients with undiagnosed pleural effusion after CXR, CT Thorax, Pleural Fluid analysis were selected. Written informed consents were taken from all the patients including attendants. Hemoglobin, total leucocyte count, platelet, BT , CT , PT- INR, Creatinine levels were checked prior to procedure. Medical thoracoscopy was performed and biopsy was taken under direct visualization.

Observation &Results:

Among 41 patients, 32 were male and 9 females. Out of 41 patients, 22 were diagnosed as adenocarcinoma (55%), 6 were diagnosed as Non small cell lung carcinoma (20%), 1 was malignant mesothelioma (5%), 7 were tuberculosis (10%) and 5 diagnosed as nonspecific pleurisy (10%). Mean age of the patients was 55+11.01 years.

Overall complication rate after medical thoracoscopy was low with no reported mortality or major complications.

CONCLUSION:

Medical Thoracoscopy is a valuable diagnostic tool for undiagnosed pleural effusion. Diagnostic yield was 87.8% in this study. It is a simple and safe procedure with low complication rate.

BACKGROUND:

Medical Thoracoscopy is a minimally invasive procedure that allows access to the pleural space using a combination of viewing and working instruments. It also allows for basic diagnostic(Undiagnosed pleural effusion or pleural thickening) and therapeutic procedures(pleurodesis) to be performed safely.

The guidelines from the American College of Chest Physicians (ACCP) recommend that trainees should perform at least 20 procedures in a supervised setting to establish basic competency. Then to maintain competency, dedicated operators should perform at least 10 procedures per year. Medical thoracoscopy/ pleuroscopy must be considered an invasive procedure that the chest physician should use only when other, simpler methods fail to yield a diagnosis or when less invasive therapeutic measures are not available or less promising. Only two diagnoses that are usually established with thoracoscopy are malignancy and tuberculosis.

The 40% of pleural effusions remain undiagnosed after an initial thoracentesis. 20% of pleural effusions remain undiagnosed after pleural biopsy. The sensitivity of medical thoracoscopy in making the diagnosis of malignancy was 94%.Thoracoscopy should also be considered if a patient has a malignant pleural effusion that is loculated.

OBJECTIVE:

1. The aim of the study is to evaluate the diagnostic yield of Thoracoscopy in an undiagnosed pleural effusion.

MATERIALS AND METHODS:

This is a single centered ,observational study conducted in Department of Pulmonary Medicine from 1st September 2017 to 1st September 2018.

Inclusion criteria was age above 14 year, 1. All cases of undiagnosed pleural effusion

2. Recurrent malignant loculated pleural effusion(pleurodesis)

EXCLUSION CRITERIA

Patients were excluded for any of the following reasons:

1. Patients having very poor general condition, Hb < 10g/dl, plat count <1.5 lac, elevated INR
2. Lack of pleural space
3. Suspected mesothelioma where the visceral and parietal surfaces are fused
4. Previous pleurodesis
5. Sputum-positive pulmonary tuberculosis
6. Not willing to participate in study
7. Unable to take lateral decubitus

INVESTIGATIONS

1. Haemoglobin, Total Count, Differential leucocytes Counts, ESR, platelet count.
2. Serum Glucose
3. Renal function tests : Serum creatinine, blood urea.
4. Sputum G/S ,C/S, AFB smear, CBNAAT
5. ICTC, viral markers, BT, CT, PT-INR
6. Chest X-RAY,CT Thorax
7. pleural fluid analysis(TC ,DLC ,protein,sugar,LDH,ADA

8. three sample of pleural fluid malignant cytology

RESULTS:

Among 41 patients, 32 were male and 9 females. Out of 41 patients, 22 were diagnosed as adenocarcinoma (55%), 6 were diagnosed as Non small cell lung carcinoma (20%), 1 was malignant mesothelioma (5%), 7 were tuberculosis (10%) and 5 diagnosed as nonspecific pleurisy (10%). Mean age of the patients was 55+11.01 years.

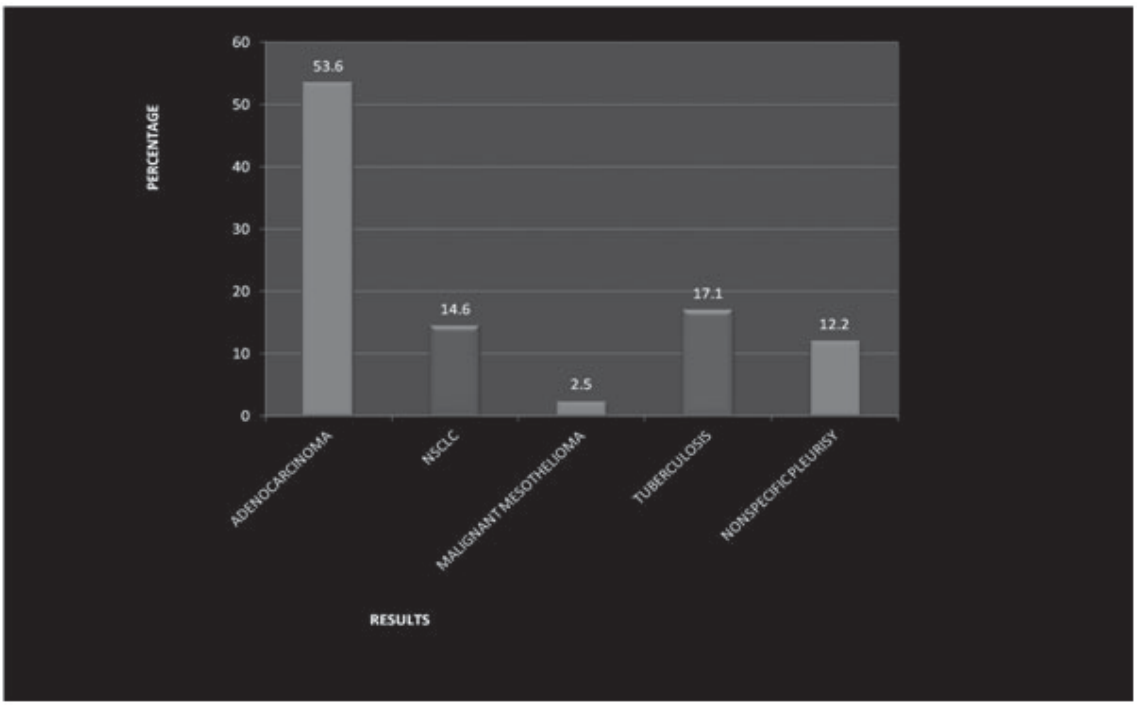
Overall complication rate after medical thoracoscopy was low with no reported mortality or major complicationsTable.1 Showing Demography of patients

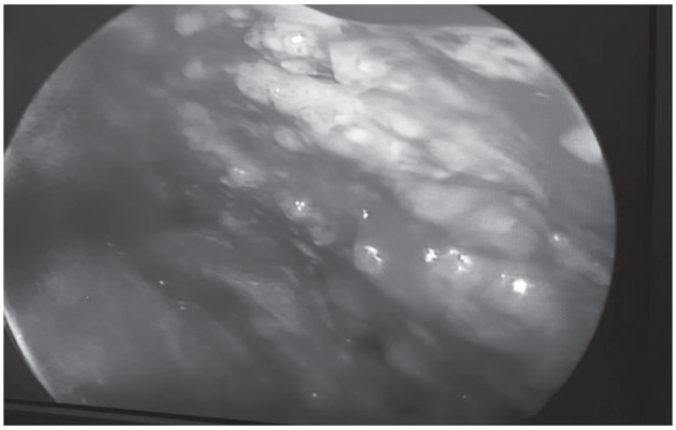
DEMOGRAPHY

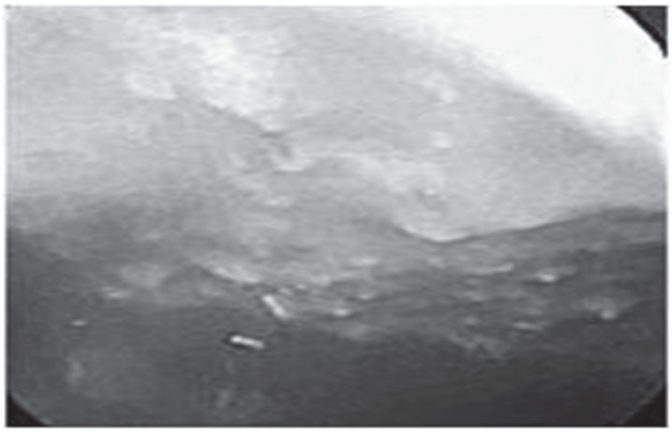
	NO. OF PATIENTS	MEAN AGE (IN YEARS)	STANDARD DEVIATION (SD)
TOTAL	41	57.21	16.43
MALE	32	57.46	6.99
FEMALE	9	56.33	16.70

Sl/no	Patient Particulars	age	Sex	Result
1	BirenSarma	57	M	Adenocarcinoma , EGFR negative
2	Dilipkrhandique	67	M	epitheloid mesothelioma, deposit from a ca.metastatic primary lung adenoca
3	Abdul karim	60	M	NSCLC
4	Riazuddinahmed	47	M	NSCLC
5	Ram bahadur lama	40	M	Adenocarcinoma , EGFR negative
6	Mahatchchaliha	65	M	TB
7	Bhagirathi devi	68	F	MALIGNANT MESOTHELIOMA
8	Abul hussain	58	M	METASTATIC Adenocarcinoma , EGFR negative
9	Sama rabi das	48	F	Adenocarcinoma , EGFR negative
10	Sarifa khatun	42	F	Adenocarcinoma , EGFR negative
11	Sarbeswar barman	70	M	NONSAMLL CELL LUNG CARCINOMA
12	Tanuramkalita	67	M	TB
13	Saiten das	55	M	NSCLC
14	Kamal kantinath	48	M	mesothelioma,pleural deposit from ca, adenoca
15	Simanta barman	36	M	Adenocarcinoma , EGFR negative
16	Labanyachoudhary	65	F	Non specific pleurisy
17	Rupeswarkalita	58	M	Adenocarcinoma , EGFR negative
18	Deben Doley	65	M	Adinocarcinoma negative
19	Pankaj Medhi	39	M	Adenocarcinoma , negative
20	Sambhuchauhan	45	M	Nonspecific pleurisy
21	Kumud Kalita	61	M	CHRONIC INLAMMATION

22	Dhaneswar das	70	M	Fibrinous pleuritis
23	JalaLuddin	40	M	TB
24	Farida Bibi	60	M	ADENOCARCINOMA
25	Moni Das	38	M	ADENOCARCINOMA
26	Milan Bhattacharya	45	M	TB
27	Tarani Kanta Nath	62	M	TB ???
28	Babul Das	45	M	ADENOCA
29	Santi Devi	55	F	TB
30	Ranjita Swargiyari	55	F	ADENOCA
31	Thaneswar Borgohain	70	M	ADENOCA
32	Nandeswar Deka	62	M	METASTATIC CARCINOMA
33	Bhubaneswar Talukdar	60	M	ADENOCARCINOMA
34	Sayeed Rahman	55	M	METASTATIC CARCINOMA
35	Prakhen Bora	55	M	FIBRINOUS PLEURITIS
36	Hiren Das	74	M	METAST CARCINOMA
37	Masooma Khatun	48	F	NSCLC
38	Puna Kalita	50	F	ADENOCA
39	Niralata Gogoi	76	F	NSCLC
40	Bimal Saha	50	M	TB
41	Siba Das	60	M	ADENOCA







DISCUSSION

1. Maturu *et al.* studied patients with undiagnosed exudative pleural effusions over a course of 10 years, Closed Pleural Biopsys were performed without imaging guidance using either the Abrams needle techniques and Medical Thoracoscopy(MT) was performed with either a rigid or semi-rigid thoracoscope, with or without ultrasound guidance for site selection. The diagnostic yield of MT was higher than CPB (93.2% *vs.* 84.5%, P=0.02) with a reported yield of 98.7% when ultrasound visualization was used to guide trochar placement in MT. These findings are similar to our study.
2. Thomas M et al retrospectively studied 407 patients undergoing medical thoracoscopy from from 2008 to 2015 in atar and found that the diagnostic yield of medical thoracoscopy for tuberculous pleural effusion was 91.4%. in all exudative effusion. Similar diagnostic yield was seen in our study as well.
3. Chen RL et al. in a study in china from 2012 to 2013 conducted medical thoracoscopy in 86 patients of undiagnosed pleural effusion. Of the 86 patients, 79 cases of pleural effusions were confirmed by medical thoracoscopic biopsy with a diagnosis rate of 91.9%. In these 79 confirmed patients, 37 had pleura cancer metastasis (43.0%) and 20 had tuberculous pleuritis (23.3%).

   These findings are similar to our study in diagnostic yield and also malignant cause being the most common cause.

4. Abd El Rehim IY et al. in the chest department of Zagazig University Hospitals in the period from October 2014 to October 2015. It included 36 patients with undiagnosed pleural effusion. The diagnostic yield of medical thoracoscopy among the studied patients was 80.6%. Maximum no cases were malignant effusion.

   These findings too correlates to our study.
5. Valsecchi A et al executed thoracoscopy 2752 patients from 1983 to 2013 over 30 year period and reported medical thoracoscopy as a high yield procedure with diagnostic yield of 71% in average. Here also most common cause of undiagnosed pleural effusion was malignant metastatsis.

CONCLUSION

- Medical Thoracoscopy is a valuable diagnostic tool for undiagnosed leural effusion
- Diagnostic yield was 87.8 %. It is a simple and safe procedure with low complication rate

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WILSON S DISEASE-A CASE REPORT

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INTRODUCTION:

Wilsons disease or hepatolenticular degeneration is a rare autosomal recessive genetic disorder caused by mutation in the ATP7B gene of chromosome 13 leading to impaired cu metabolism leading to accumulation of cu in different organs like liver, brain, eyes etc and manifest as liver disease (acute, chronic hepatitis and cirrhosis) and neuropsychiatric symptoms, acute or chronic kidney disease etc.[1]

CASE HISTORY:

A 5 years old female child presented with history of decreased appetite for 1 month and yellowish colouration of whole body for 15 days, and decreased urine output for 7 days and gradual swelling of abdomen and legs and fever on and off. The child received treatment from a local hospital. History of intake of herbal medicine was also present. The child developed altered sensorium for 4 days for which she was referred. History of consanguinity or liver disease was absent in the family. On admission the child was having a GCS of E1M3V1. The child was admitted and put on mechanical ventilator in view of low GCS and treatment started with iv fluids, Inj cefotaxime, inj vitamin k, tab rifaximine, inj Furosemide, Lactulose syrup, infusion mannitol, ursodeoxycholic syrup, tab pyridoxine, enema etc. LFT was deranged (table 1) with a deranged PT and INR. Child was transfused with 2 units of FFP. 1 unit of packed cell was also transfused as hemoglobin was 6.7 mg/dl . Child was worked up for possible etiologies. IGm HAV, HBs ag and anti HCV were negative.Ultrasound abdomen revealed enlarged liver with diffuse increase in echotexture and nodular hypoechogenicity with periportal collaterals consistent with Wilsons disease.There was a compensated state fo portal hypertension.DCT was negative. 24 hr urinary copper was raised (559.29 microgram/dl). Serum ceruloplasmin was decreased (10 mg/dl.) Blood culture and urine cultures were sterile. Slit lamp examination could not be done.Condition of the patient had improved and patient was weaned from ventilator after 3 days The child was statrted. Zinc tab and tab penicillamine, tab propanolol was started. Child gradually improved and was discharged form the hospital after 12 days of stay.At dicharge the child was having a GCS of 15/15 and was on oral feeds and was ambulatory.She is on regular follow up and continuing oral penicillamine and zinc. Her liver echotexture has improved on repeat ultrasounds and the liver function has improved.

Table 1: Liver function tests

Test	Day 1	Day 3	Day 6	After one month	After two months
Bilirubin	34.2	31.4	27.2	7.8	1.8
Bilirubin (direct)	31.5	29.1	26.5	7	1.3
AST	218	138	82	120	91
ALT	40	59	73	115	75
GGT	122	139	127	122	180
LDH	1122	705	460	249	355
Total Protein	5	5.1	5	6.2	6.7
Albumin	2.6	2.8	2.4	2.8	3.8
Globulin	2.4	2.3	2.6	3.4	2.9
PT	40.4	33.6			
INR	3.4	2.8			

DISCUSSION :

Wilson's disease is related to a derangement in copper metabolism, with high levels of liver copper and copper accumulation within the brain substance, especially within the central gray matter. It is transmitted as an autosomal recessive trait, with an incidence of 1 : 40000 to 1: 100,000[2] in homozygous form. Copper first accumulates in the liver; hence the usual presentation during childhood is that of hepatic disease. After the liver storage capacity for copper gets saturated, copper gets redistributed with accumulation in the nervous system, cornea, kidneys and other organs.[3] Most patients present in the second decade of life with a primary hepatic presentation with the remainder of patients presenting during the third and fourth decade with a primarily neurologic or psychiatric presentation[3].

The age of presentation can vary from 4 to 40 years though more than half present before the age of 15 years.The manifestations are more likely to be hepatic in early childhood and neurological in adolescents.[4]Children with WD are usually normal at birth and may remain healthy for a variable period of time; most cases present in the second and third decade of life . [5] Our patient presented with fulminant hepatic failure at 5 years of age. The earlier age of presentation of our patient is in accordance with other studies which show an earlier age of presentation than western countries.[6-8]

Our child presented with hepatitis like features which is the usual mode of presentation in younger children. Diagnostic tests of WD include: (i) high hepatic Cu (> 250 μg/g dry liver), (ii) low ceruloplasmin (Cp) (< 18 mg/dl), (iii) increased non-Cp Cu (free Cu), (iv) increased urinary Cu (> 100 μg in 24 hours increasing to > 250 μg in 24 hours) after test dose of penicillamine, and (v) KF rings as seen by slit lamp microscopy.[4] Our child had a high 24 hours urinary copper and low serum ceruloplasmin.

The drug of choice for Wilsons disease is Penicillamine along with Zinc and Pyridoxine.[4] Our child has shown excellent response to therapy with the above mentioned drugs.

Despite initiation and maintenance of adequate Cu chelation therapy, the outcome is unpredicatable with upto 48% mortality in hospital series(. The types of outcome seen are: (i) Rapid and complete clinical improvement especially of hepatic symptoms with reversal of parenchymal lesions including early cirrhosis; (ii) Initial deterioration particularly of neurological symptoms with eventual improvement but with residual handicap

(speech, hand writing); and (iii) Relentless deterioration and death inspite of therapy as in FHF and hemolysis.[7]

CONCLUSION :

Wilsons disease is an inherited metabolic disorder. It should be suspected in young children with hepatic complication. Since the presentation could be akin to any hepatitis a high index of suspicion is required for diagnosis. Early diagnosis and treatment with proper follow up shows significant reduction in morbidity and mortality. Siblings should be screened to prevent manifestation. Liver transplantation may be required.

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MEMANTINE EXPLORED IN THE TREATMENT OF RESISTANT OCD WITH COMORBID DEPRESSION: A CASE-REPORT

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INTRODUCTION:

Obsessive compulsive disorder is a common psychiatric disorder which is often refractory to treatment. Current options for treatment resistant cases includes switching to an alternative selective serotonin reuptake inhibitor or augmentation with dopamine antagonist or other agents[5]. Evidence from genetic,behavioural and neuroimaging studieshave indicated that disrupted neurotransmission of glutamate within the cortico-striato-thalamo-cortical circuitry is also involved in the pathophysiology[1,2]. One medication that has shown promising results in this regard is Memantine,NMDA receptor antagonist, which provides anti-obsessional benefits[3,4]. This case report provides therapeutic effect of add on memantine in treatment resistant OCD with comorbid depression.

CASE REPORT:

Patient A,36 years female,presented with 15 year history of debilitating ego-dystonic obsessions and past history of three suicidal attempts. She hadrecurrent intrusive thoughts about contamination resulting in compulsive behaviours with her indulging in repeated and continuous activities,be it taking bath or washing vegetables or cleaning house, to decrease the associated anxiety.

She would take 2-3 hours in bathing and devote nearly 6-7 hours in self care and household activities leading to an overall slowness while performing any task. She would divide each act into a number of small steps and spend variable periods of time deciding on whether to continue with the behaviour. She would sometimes even pass stools in her clothes due to her inability to decide whether to go toilet or not. Her obsessional fear of contamination and the resulting slowness also led to strained relationship with her kins.

At the time of presentation,patient was experiencing low mood with decreased pleasure and pessimistic views about future resulting in suicidal ideas. She verbalised inner distress and also reported that she would self starve at times because of having to clean her up multiple times if she defecates.

Routine blood investigations, urine examination, computed tomography of brain and also electroencephalogram conducted did not reveal any abnormality. Patient was a known case of hypothyroidism on Thyroxine 25microgram/day and her Thyroid Function Test showed moderately raised TSH level(12 microIU/ml) and after medicine consultation Thyroxinewas titrated to 75 microgram/day.

The patient was diagnosed to have OCD with secondary slowness and depressive disorderaccording to ICD-10. Subsequent adequate trials with Fluoxetine and Escitalopram were ineffective and add on Risperidone caused galactorrhoea and were discontinued.Addition of Aripiprazole 15mg was considered andECT was started.As no response with fluoxetine(Y-BOCS score=36), Oral Clomipramine started and titrated to 150mg per day.Y-BOCS score came as 35 ,10 weeks later. Sertraline titrated to 200mg/ day, was added along with Clomipramine and Aripiprazole.patient responded inadequately and thereforeSodium Valproate(titrated to

600mg) was given. Psychotherapy including CBT was also initiated (Y-BOCS score=30). At this point adding Memantine was considered, started at 5 mg and titrated to 10 mg within 10 days. Patient A reported initial relief on day 17 and significant reduction in symptoms severity was noted 3 weeks later(Y-BOCS score= 21). On follow up visits there is no clinically significant side effect and improvement is maintained till date.

DISCUSSION:

This case report provides evidence that memantine augmentation is beneficial in resistant OCD and is also well tolerated. Literature in support for the same also exists, exploring the role of memantine in OCD

Ajay K.Bakhla et al[6] conducted an open label trial using fixed dose memantine in few treatment refractory OCD patients and reported a mean Y-BOCS reduction of 44% in the good responders, supporting the role of menamtine in treatment resistant OCD

Aboujaoude E et al[7] conducted an open label augmentation trial of memantine in treatment resistant OCD and almost half the patients had a meaningful improvement in symptoms.

There are case reports describing memantine treatment in OCD. Poyurovsky et al.[8] reported improvement with memantine augmentation in one patient with treatment resistant OCD, while Pasquini and Biondi[3] noted improvement in one OCD patient with checking compulsions

Case control study by Stewart et al[4] reported a mean OCD severity improvement of at least 25%, denoting clinically significant improvement among the case patient.

CONCLUSION:

Further identification of such cases is needed to learn about pathophysiology and develop treatments for this disorder.

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TUMOURS OF THE HEART: A SHORT OVERVIEW

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Neoplastic involvement of the heart can be divided into primary cardiac tumours arising in the heart and secondary cardiac tumours that have metastasised to the heart. Primary cardiac tumours can be further stratified into benign and malignant tumours. Primary cardiac tumours are rare with an incidence of 0.0013 and 0.03% in collected autopsy series[1,2,3]. In adults 50% of benign cardiac tumours are myxomas[4].

PRIMARY BENIGN TUMORS

Myxomas: Cardiac myxomas are benign intra-cavitary neoplasm. They most often occur in women in the third to sixth decade of life. Myxomas are usually sporadic, but at least in 7% cases, they occur as part of an autosomal dominant syndrome referred as the *Carney complex*[5]. The exact incidence in India is not known but these tumours constituted 0.24% of all cardiac operations and constituted 91.7% of all primary tumours operated (n = 72), during a 20 year period at AIIMS, New Delhi. Approximately 75–80% of myxomas are located in the left atrium, 10– 20% in right atrium and 5–10% in both atria or either ventricle. Since cardiac myxoma exhibits rapid growth most patients show advanced symptoms at the time of surgery.[6] Possible risk factors for recurrence of cardiac myxoma include incomplete resection, intracardiac implantation, embolisation, and a reserve of tumour precursor cells in the subendocardium.[7] The risk of embolism is higher for polypoid or multi-lobular shaped tumours than round ones.[8] Therefore, if a polypoid or multi-lobular shaped myxoma is suspected on preoperative echocardiography, urgent surgical intervention is necessary to prevent an embolism. Surgical resection is the mainstay of treatment. The intracardiac mass is excised after establishing cardiopulmonary bypass and cardioplegic arrest. Recent studies on the resection of cardiac myxoma with normal cardiac tissues indicated that there was no difference in recurrence rates between patients who underwent an en bloc resection and resection with only endocardial tissue attached with the tumour.[9] However, the type of resection depends largely on factors such as the location of the tumour and the shape of tumour stalks.

Lipomas

Lipomas are well-encapsulated tumours consisting of mature fat cells that can occur anywhere in the heart, but also are found in the pericardium, subendocardium, sub-epicardium, or intra-atrial septum.[10] Many are asymptomatic and are discovered incidentally on routine chest roentgenogram, echocardiogram, or at surgery or autopsy.[11] Large tumours that produce severe symptoms should be resected.

Lipomatous Hypertrophy of the Interatrial Septum

Nonencapsulated hypertrophy of the fat within the atrial septum is known as lipomatous hypertrophy.[12] This abnormality is more common than cardiac lipoma and is usually encountered in older, obese, or female patients as an incidental finding during a variety of cardiac imaging procedures. Various arrhythmias and conduction disturbances have been attributed to its presence.[13] After the demonstration of a mass by echocardiography, the typical T1 and T2 signal intensity of fat on MRI can usually establish a diagnosis[14] Arrhythmias or heart block are considered by some as an indication for resection, but there is lack of data as to the long-term benefits from resection.[15]

Papillary Fibroelastoma of the Heart Valves

Papillary fibroelastomas are tumours that arise characteristically from the cardiac valves or adjacent endocardium.[16] Valve repair rather than replacement should follow the resection of these benign tumours whenever technically feasible, using conservative margins of resection. Cytomegalovirus has been recovered in these tumours, suggesting the possibility of viral induction of the tumour and chronic viral endocarditis.15

Rhabdomyoma

Rhabdomyoma is the most frequently occurring cardiac tumour in children. It usually manifests during the first few days after birth.[17] it is associated strongly with tuberous sclerosis. Early operation is recommended in patients who do not have tuberous sclerosis before 1 year of age.

Fibroma

Fibromas are the second most common benign cardiac tumour, with over 83% occurring in children.[18] Intracardiac calcification on chest radiographs suggests the diagnosis, which is confirmed by echocardiogram. Surgical excision is successful in some patients, particularly if the tumour is localised, does not involve vital structures, and can be enucleated[19]

Paraganglioma

Cardiac paragangliomas arise from chromaûn cells of the sympathetic nervous system and can produce excess amounts of catecholamines, particularly norepinephrine, but usually are not hormonally active.[20] After the tumour is located, it should be removed, using cardiopulmonary bypass with cardioplegic arrest. Hormonally active patients require preanesthetic alpha and beta blockade, and careful intraoperative and immediate postoperative monitoring. Most tumours are extremely vascular and uncontrolled operative haemorrhage has occurred.[21]

Haemangioma

Haemangiomas of the heart are rare, affect all ages, and can occur anywhere within the heart. The tumours can be resected in asymptomatic patients, and cardiopulmonary bypass is recommended.[22]

PRIMARY MALIGNANT TUMOURS

Sarcomas

Primary cardiac malignancy is uncommon. Sarcomas are cancers that develop from connective tissues (blood vessels, nerves, bones, fat, muscles, and cartilage). Sarcomas develop in the right or left atrium and can block blood flow through the heart. Tumours in the right atrium can spread to the lungs.[23] These tumours are all aggressive, and post-resection chemotherapy are recommended even when R0 resection is achieved. Surgical resection and overall care of these patients is complex. Malignant primary cardiac sarcomas often grow to a larger size until clinical detection. Extensive involvement may therefore make complete resection and reconstruction impossible. Because complete resection is necessary for improved results, cardiac transplantation has been considered in some cases.[24, 25]

Nonsarcomatous Primary Malignant Cardiac Tumors:

Lymphomas

Lymphomas can arise from the heart, although it is rare. Most of these tumours respond to radiation and chemotherapy[26]

SECONDARY METASTATIC TUMORS

Approximately 10% of metastatic tumours eventually reach the heart or pericardium, and almost every type of

malignant tumour has been known to do so.[27] Up to 50% of patients with leukaemia develop cardiac lesions. Other cancers that commonly involve the heart include breast, lung, lymphoma, melanoma, and various sarcomas.[28] Cardiac metastases produce clinical symptoms in only about 10% of aûicted patients. The most common symptom is pericardial effusion or cardiac tamponade. Surgical therapy is generally limited to relief of recurrent pericardial effusions or, occasionally, cardiac tamponade.Surgical therapy is directed at providing symptomatic palliation with minimal patient discomfort and hospital stay.[29]

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VANISHING RENAL STONE: A MYSTIFYING INCIDENT.

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ABSTRACT

Renal stones more than two centimeters diameter are mainly managed with surgical intervention. Here, we present a case where a stone of 2.1cm diameter disappeared after DJ stenting and antibiotic therapy. To our knowledge, only one such case has been reported in literature. The underlying physiology is unclear and the case remains an enigma.

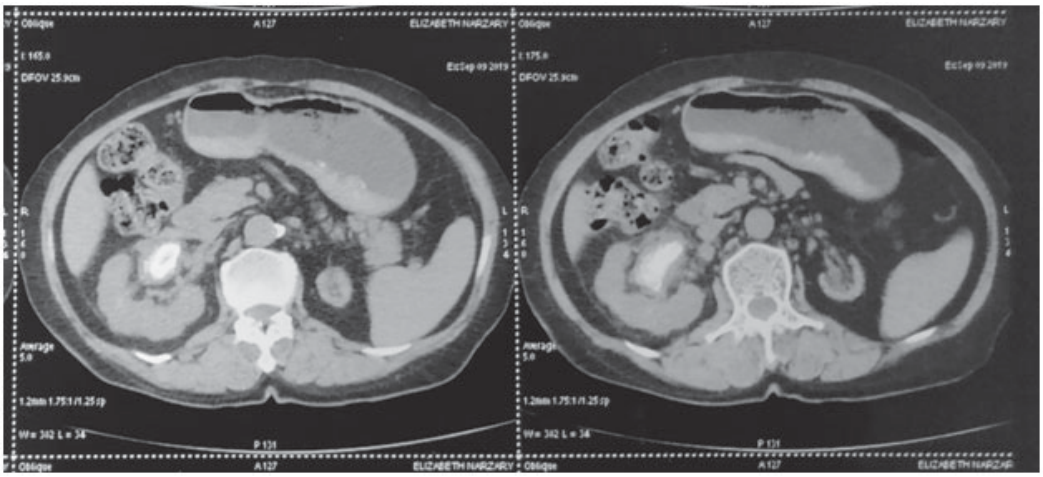
INTRODUCTION

Renal stone is a global disease with incidence up to 12% [1]. The management depends on stone size, location andhardness and a variety of other clinical factors[2]. The management options for stone size more than 1cm includes Extracorporeal shock wave lithotripsy, Retrograde intra-renal surgery and Percutaneous nephrolithotripsy [2]. It is extremely uncommon for renal stone size more than 1 cm to disappear without any intervention.

Here, we report a case where a stone of 2.1 cm size disappeared after DJ stenting and antibiotics.

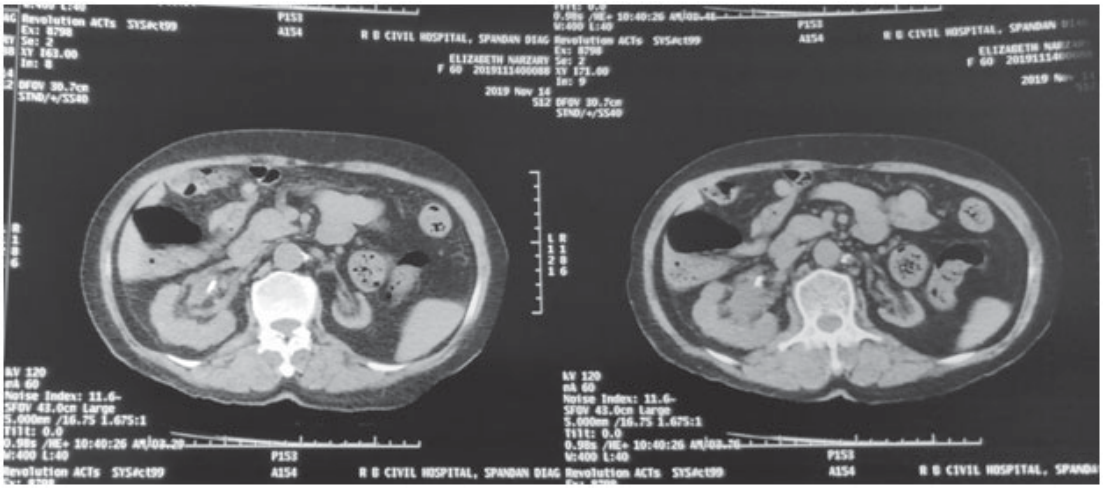
CASE REPORT

A 72 years old woman presented to urology opd with right flank pain, low grade fever, and oliguria for 5 days. She had a history of left renal surgery 26 years back for stone disease. her hemoglobin level was 7.2 g/dl, total leucocyte count as 13000 with 90% neutrophilia. Her serum creatinine level was 9.0 mg/dl with normal serum electrolytes. On initial ultrasound examination, she had a small left kidney and a pelvic calculus in right kidney with signs of pyelonephritis. A non- contrast CT scan was done which showed a 2.1 cm right renal pelvic calculus with 720 HU as shown in picture-1, and other features corroborating with ultrasound exam.



Patient was admitted and started in IV antibiotics. Initially right percutaneous nephrostomy was offered as drainage for which the patient did not give consent after which right sided DJ stenting was done. Increase in urine output and a falling trend on the serum creatinine values was noted. Patient was discharged on post-operative day 3 with a daily urine output of around 2 liters and serum creatinine value of 4.2.

Patient was instructed to return to follow up after 1 week but she returned after 6 weeks. Her creatinine was 1.5 and a non- contrast CT scan was done to reassess the stone. To our surprise, the stone was absent altogether, and only the stent could be visualized, as seen in picture-2.



Patient was later followed up after one more week when she had the same creatinine value. The stent was removed subsequently.

DISCUSSION

Renal stones present in a myriad of clinical situations and the management entails medical and surgical principles considering the holistic clinical scenario. Vanishing renal stone after antibiotics or stenting is rare and have been reported in only one other case report [3]. The exact mechanism is not clear for this phenomenon. However, one hypothesis which can be put is that the stone was an infective soft matrix stone. As it consists of primarily amorphous material [4], antibiotics and effective drainage through DJ stent might have causes dissolution and drainage of stone fragments. Nevertheless, the physiology of it remains unclear and it remains an enigma.

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EPIDEMIOLOGICAL AND CLINICAL PROFILE OF COVID 19 PATIENTS IN A TERTIARY CARE HOSPITAL: A RETROSPECTIVEANALYSIS

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ABSTRACT

BACKGROUND: An unidentified pneumonia outbreak was first observed in Wuhan, the capital of Hubei Province, China, in December 2019. WHO officially named the virus Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2)and the disease, Coronavirus Disease 2019 (COVID-19), and on Mar 11, 2020, declared COVID-19 as pandemic Hence, we aimed to perform a systematic review of the epidemiological and clinical characteristics of COVID-19 patients admitted inatertiarycare hospitalin North East India.

METHODS: We retrospectively analysed 375 cases admitted at Ayursundra Super Speciality Hospital, Guwahati between July 6th to September 10th, 2020.

RESULTS: We included a total of 375 patients with 60.27% being male. Thepredominant symptoms were fever (86.4%), cough (68.53%), and fatigue/myalgia (38.13%) and other symptoms including dyspnea,chest pain, and so rethroat. We also found patients with G Isymptoms like diarrhea (8.53%) and nausea/vomiting (44.53%). Comorbidities were found in 162 (42.2%) patients with the most common being hypertension (23.73%) followed by diabetesmellitus (13.07%). At admission, 56.37% presented with lymphopenia and 36.27% had elevated D-dimers. Severe-criticalpatientswere 5.06% with a median time from on settocritical disease of 8.5 days. 24% of the patients required oxygen therapy. The case fatality rate was 1.6% with median time from on setto death of 16days.

CONCLUSION: Patients with coexisting comorbidities are at higher risk and need more utilization of health careresources. As this virus is spreading globally, all countries have to join hands and prepareat all levels ofhuman resources, infrastructure, and facilities to combat theCOVID-19 disease.

KEYWORDS: COVID19, fever, cough, D-dimer, SARS-CoV2

* * *

I. INTRODUCTION

COVID-19 is an emerging infectious disease that has exerted a tremendous impact on public health andsocioeconomic development. Conûrmed cases have been reported in 196 countries, areas or territories. As of March 25, 2020, 375,498 cases including 16,362 deaths were reported worldwide[1]. Corona virus is an enveloped, non-segmented, positivesense single-stranded RNA virus with genome size ranging from 26 to 32 kilobases (the largest known viral RNA genome) [2]. COVID-19 is mainly transmitted by droplets and contact with contaminated surfaces or objects, showing human-to-human transmission, family aggregation spread, and no socomial infection [3]. It is worth noting that COVID-19 has a diverse clinical presentation, ranging from a symptomatic infection to mild respiratory illness to more severe complications of pneumonia, Acute Respiratory Distress Syndrome requiring intensive care unit (ICU) admission, and mechanical ventilation. We investigated epidemiological and clinical features, disease severity, diagnosis, treatment, clinical outcomes, and follow-up of COVID-19 in a tertiary care centre in North East, with the hope of assisting other large urban centres in planning for the high risk of extensive SARS-CoV2 transmission. The eû ective management of this epidemic highlights the beneût so fearly and aggressive control measures.

II. METHODS

COVID-19 conûrmed patients admitted in the Emergency Department of Ayursundra Super Speciality Hospital from July 6th to September 10th were included. Epidemiological history, clinical manifestations, laboratory test results, and imaging test results were retrospectively collected from medical records. The study was approved by ASHEthics Committee.

DIAGNOSIS AND ADMISSION PROCESS

Real-timer ever setranscriptase polymerasecha in reaction (RT–PCR)assay for conûrming SARS-CoV-2 infection were conducted according to WHO protocols. Patients with positive nucleic acid test results were transferred to ASH for further treatment using negative pressure ambulances.

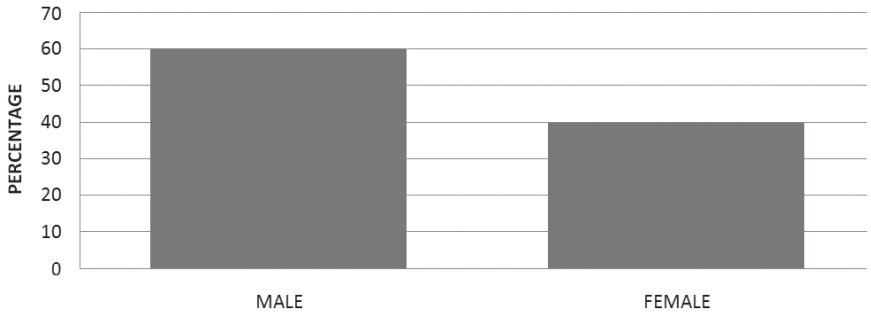
FOLLOW-UP AFTER DISCHARGE

Patients were followed-up at 2-and 4-weeks post-discharge to as certain if any contacts had respiratory symptoms or fever. Blood counts, liver and kidney function, and nucleic acid tests were completed on oro-ornasopharyngeal swabsorinsputum. Chestcomputed tomography (CT) was provided as needed.

III. RESULTS

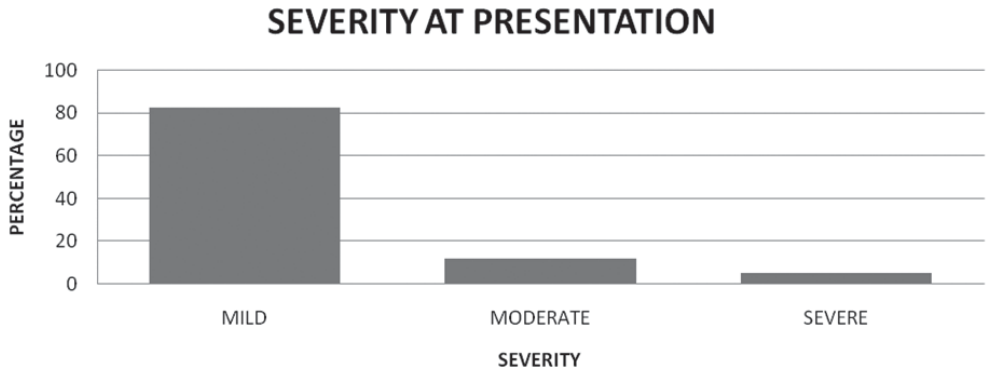
Out of 375 confirmed COVID-19 patients, there was higher percentage of male patients (226 (60.27%)) than female (149(39.73%))

	NUMBEROFPATIENTS	PERCENTAGE
MALE	226	60.27
FEMALE	149	39.73



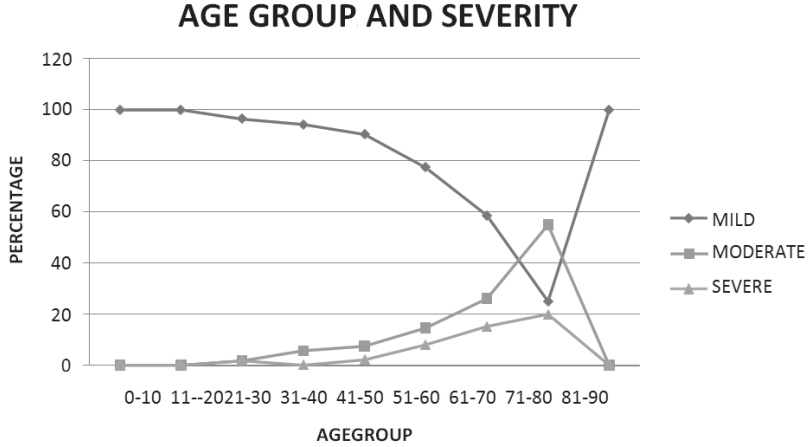
2. Of the confirmed cases, 82.93% were mild (SpO2>94% on room air), 12% were moderate (SpO290-94% on room air) and 5.06% (<90% on room air) were severeat presentation.

SEVERITY	NUMBEROFPATIENTS	PERCENTAGE
MILD	311	82.93
MODERATE	45	12
SEVERE	19	5.06



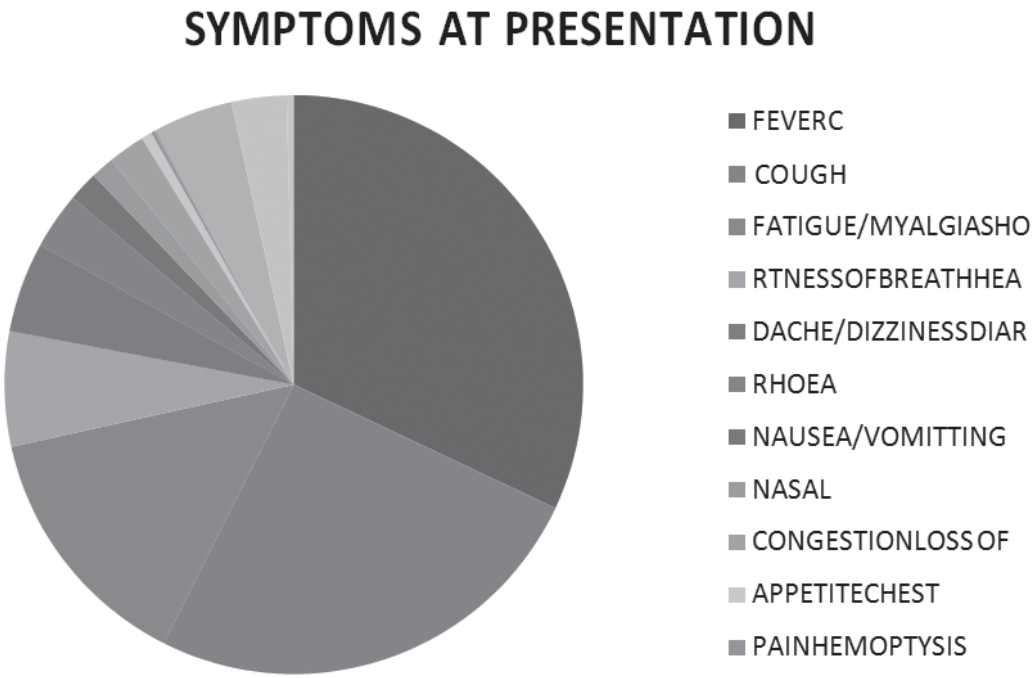
3. Of the total active cases, 94(25.07) were in the age group of 41-50 years followed by 31-40 years(23.47%) and 51-60 years (16.53%) where as maximum severe cases were found in the age group of 71-80 years(20%).

AGE AT PRESENTATION	SEVERITY			TOTAL(%)
	MILD(%)	MODERATE(%)	SEVERE(%)	
0-10	2(100)	0	0	2(0.53)
11-20	3(100)	0	0	3(0.8)
21-30	54(96.43)	1(1.78)	1(1.78)	56(14.93)
31-40	83(94.32)	5(5.68)	0	88(23.47)
41-50	85(90.43)	7(7.45)	2(2.13)	94(25.07)
51-60	48(77.42)	9(14.52)	5(8.06)	62(16.53)
61-70	27(58.69)	12(26.09)	7(15.22)	46(12.27)
71-80	5(25)	11(55)	4(20)	20(5.33)
81-90	4(100)	0	0	4(1.06)



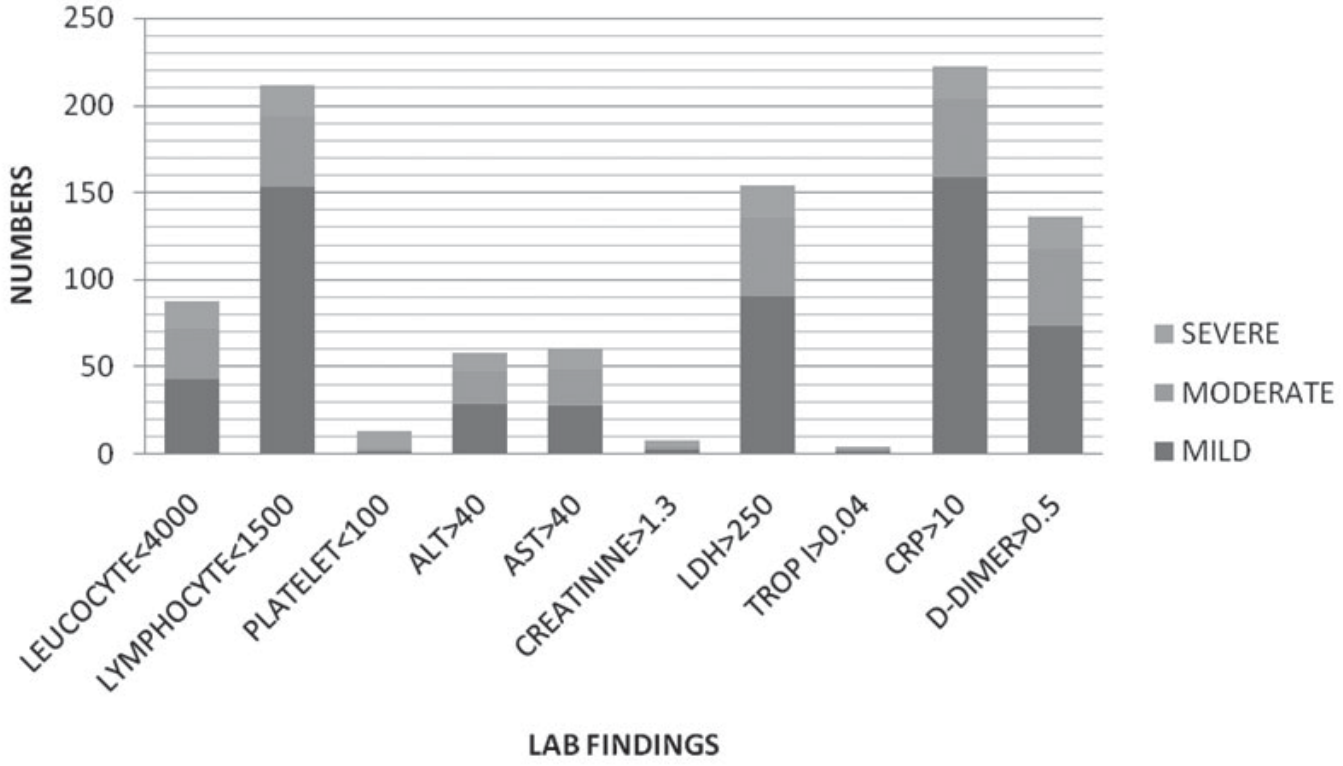
4. In regard to clinical presentation, fever was the most common symptom, seen in 324 (86.4%) patients, followed by cough (257(68.53%)), fatigue/myalgia (143(38.13)) and dyspnea (65(17.33%)). Lesser common symptoms include diarrhea in (32 (8.53%)) patients followed by nausea/vomiting (17(4.53%)), nasal congestion (13(3.46%)), loss of appetite (21(5.6%))

SYMPTOMSATPRESENTATION	NUMBER(%)
FEVER	324(86.4)
COUGH	257(68.53)
FATIGUE/MYALGIA	143(38.13)
SHORTNESSOFBREATH	65(17.33)
HEADACHE/DIZZINESS	51(13.6)
DIARRHOEA	32(8.53)
NAUSEA/VOMITING	17(4.53)
NASALCONGESTION	13(3.46)
LOSSOFAPPETITE	21(5.6)
CHESTPAIN	6(1.6)
HEMOPTYSIS	2(0.53)
IMPAIREDCONSCIOUSNESS	1(0.26)
IMPAIRED SMELL	45(12)
IMPAIRED TASTE	31(8.26)
CONJUNCTIVALCONGESTION	4(1.06)



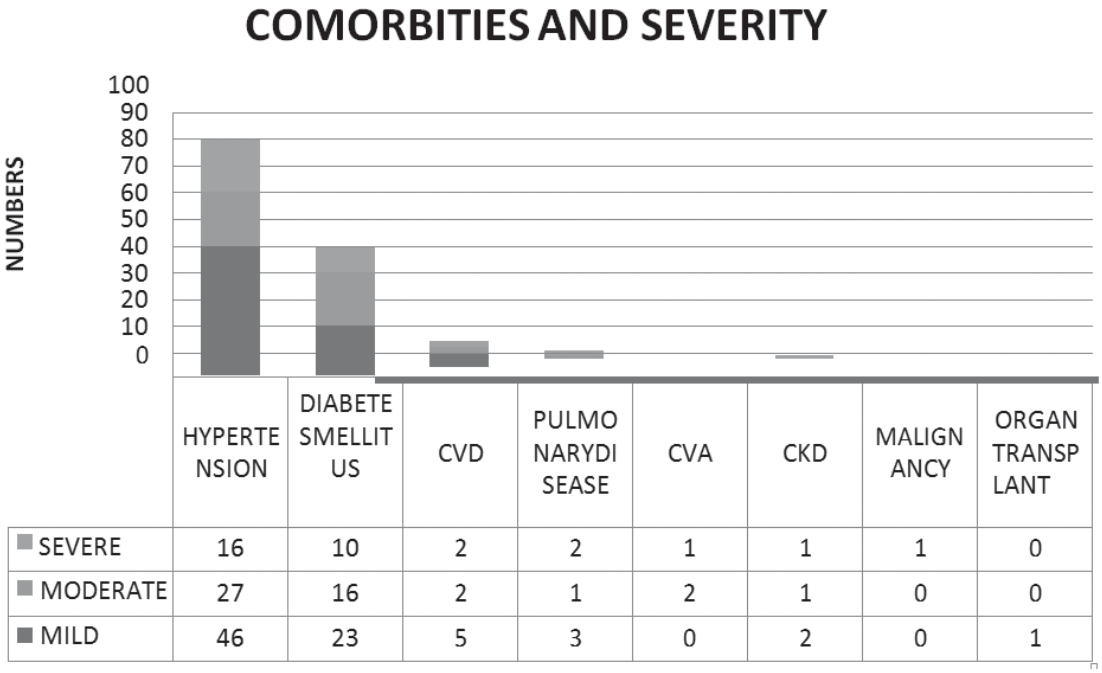
5. At admission, nearly two-thirds (56.27; 211/375) presented with lymphopenia. Over half (559.2%; 222/375) had increased C-reactive protein (CRP); 36.27% (136/375) had elevated D-dimers; a quarter had leukocytopenia (23.2%; 87/375).

LABFINDINDS	SEVERITY			TOTAL(%)
	MILD(%)	MODERATE(%)	SEVERE(%)	
LEUKOCYTE(permm^3)<4000	42(48.27)	29(33.33)	16(18.39)	87(23.2)
LYMPHOCYTE(permm^3)<1500	153(72.51)	40(18.96)	18(8.53)	211(56.27)
PLATELET(10^3permm^3)<100	1(7.69)	2(15.38)	10(76.92)	13(3.47)
Alanineaminotransferase(U/ml)>40	28(48.27)	19(32.76)	11(18.96)	58(15.47)
Aspartateaminotransferase(U/ml)>40	27(45)	21(35)	12(20)	60(16)
Creatinine>1.3	2(28.57)	2(28.57)	3(42.86)	7(1.87)
Serum Lactate Dehydrogenase(U/litre)>250	90(58.44)	45(29.22)	19(12.34)	154(41.07)
TroponinI(ng/ml)>0.04	1(25)	1(25)	2(50)	4(1.07)
C-reactiveprotein(mg/litre)>10	158(71.17)	45(20.27)	19(8.55)	222(59.2)
D-dimer(microgram/ml)>0.5	73(53.68)	44(32.35)	19(13.97)	136(36.27)



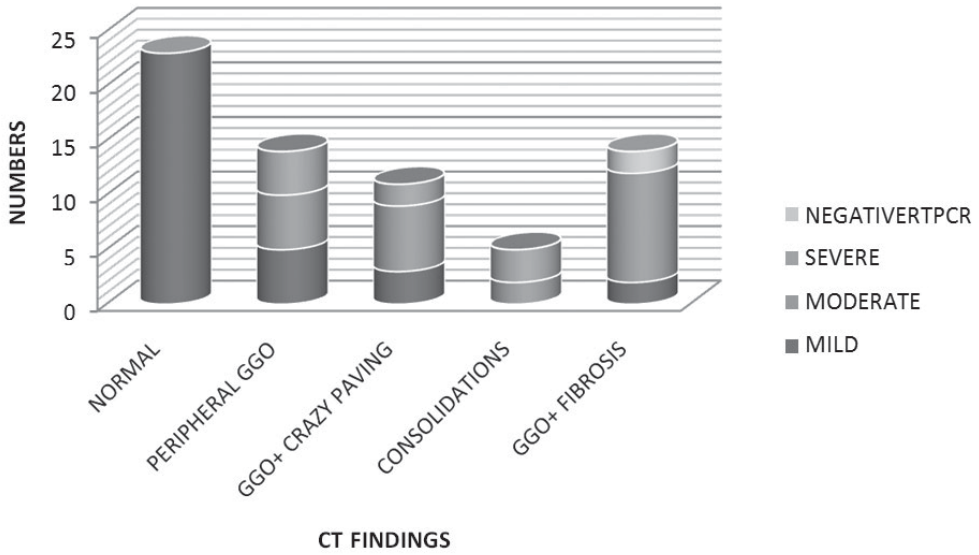
6. Under lying co-morbidities were present in 43.2% (162/375)of cases, included hyper tension (23.73%;89/375), diabetes (13.07%;49/375), and coronary heart disease (2.4%;9/375).

COMORBIDITIES	SEVERITY			TOTAL(%)
	MILD(%)	MODERATE(%)	SEVERE(%)	
Hypertension	46(12.27)	27(7.2)	16(4.27)	89(23.73)
Diabetesmellitus	23(6.13)	16(4.27)	10(2.67)	49(13.07)
Cardiovasculardiseases	5(1.33)	2(0.53)	2(0.53)	9(2.4)
PulmonaryDisease	3(0.8)	1(0.27)	2(0.53)	6(1.6)
CerebrovascularDisease	0	2(0.53)	1(0.27)	3(0.8)
ChronicKidneyDisease	2(0.53)	1(0.27)	1(0.27)	4(1.07)
Malignancy	0	0	1(0.27)	1(0.27)
OrganTransplant	1(0.27)	0	0	1(0.27)



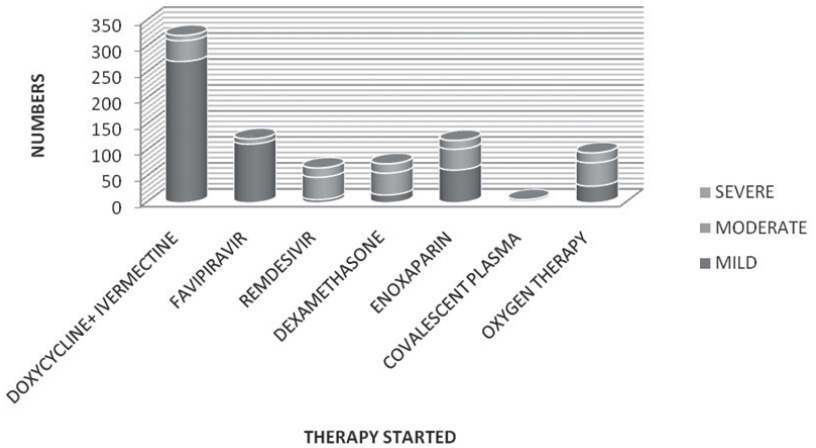
7. Computed tomography (CT) scan was performed on 67 patients (17.87%), mainly revealed ground glass opacities in the majority of the patients. 34.34% patients had normal CT scans. Few patients showed imaging features of pleural effusion and consolidation involving one or multiple lobes. 2(2.98%) patients had negative RTPCR where the CT findings showed GGOand fibrosis typical of COVID pneumonitis.

CT THORAX FINDINGS	SEVERITY			NEGATIVERTPCR
	MILD(%)	MODERATE(%)	SEVERE(%)	
Normal	23(34.34)	0	0	
PeripheralGOO	5(7.46)	5(7.46)	4(5.97)	
GOO+ CrazyPaving	3(4.48)	6(8.95)	2(2.98)	
Consolidations	0	2(2.98)	3(4.48)	
GGO+Fibrosis	2(2.98)	10(14.92)	0	2(2.98)



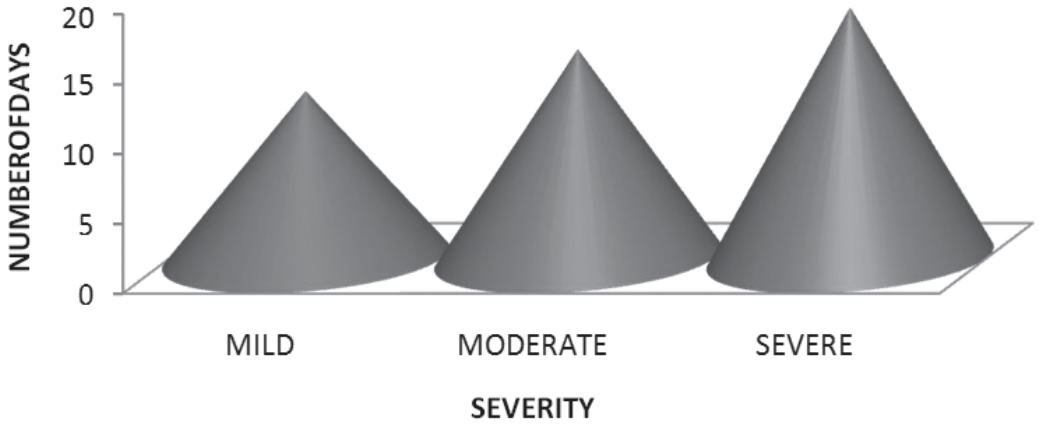
8. A quarter of the admitted patients (24%; 90/375) required oxygen during the disease course with 6.13%(23/375)needing high-flow nasal oxygen (HFNO). Antiviral therapieswere prescribed to 50.67% (190/375) of patients, with treatment consisting of favipiravir and remdesivir. Other prescribed medicationsincluded doxycycline and ivermectin (85.87%; 322/375), anticoagulant drugs i.e. low molecular weightheparin or heparin (32%; 120/375), glucocorticoid i.e. dexamethasone(20%; 75/375). Among mild cases, 190/311(61.74%)did not receive anti viral therapy. Very few patients(1.6%;6/375)were given convalescent plasma therapy

THERAPYSTARTED	SEVERITY			TOTAL(%)
	MILD(%)	MODERATE(%)	SEVERE(%)	
Doxycycline +Ivermectin	272(72.53)	40(10.67)	10(2.67)	322(85.87)
Favipiravir	113(30.13)	10(2.67)	0	123(32.8)
Remdesivir	6(1.6)	43(11.47)	18(4.8)	67(17.87)
Dexamethasone	15(4)	42(11.2)	18(4.8)	75(20)
Enoxaparin	63(16.8)	40(10.67)	17(4.53)	120(32)
CovalescentPlasma	0	2(0.53)	4(1.06)	6(1.6)
OxygenTherapy	32(8.53)	45(12)	19(5.07)	90(24)



9. Median time from illness onsett o negative viral detection was longer in severe and critical patients compared with mild patients.

SEVERITY	AVERAGEDURATIONOFSTAY INHOSPITAL
MILD	12
MODERATE	15
SEVERE	18



10. Six deaths, all caused by acute respiratory distress syndrome (ARDS) or multiple organ dysfunction (MODS), yielded a case fatality rate of 1.6% (6/375) with median time from onset to death of 16 days. Casefatality rate in patients with glucocorticoid therapy was 6.67%, while no patient died among those withoutglucocorticoid therapy.

EXPIRED PATIENTNUMBER	SpO2 AT PRESENTATION	AGE	COMORBIDITIES	THERAPYRECEIVED	DURATION BETWEEN ONSET OFSYMPTOMS TO PRESENATION
1. PK	90	58	DM,HTN,CKD	LMWH,DOXYC YCLINE, MEROPENAM,HFO	8
2. KH	84	59	DM,AKI	LMWH, DEXAMETHASONE, MEROPENAM, MV	2
3. BB	54	62	HTN,MALIGNANY,CVD	DEXAMETHASONE, MEROPENAM,RE MDESIVIR,MV	2
4. JD	52	55	HTN	DEXAMETHASONE, REMDESIVIR,LMWH,BI PAP	1
5. MB	54	70	HTN,HYPOTHYROID	REMDESIVIR,LMWH,DE XAMETHASONE, NIV	3
6. PQ	76	72	NO	REMDESIVIR,DEXA METHASONE, LMWH	1

IV. DISCUSSION

Although most cases presented with fever, 13.6% never developed fever through out their illness. Fever was an important clue for detecting imported cases. Signiûcant reduction of lymphopenia and

CD4+Tlymphopenia as well as ground-glass lesions on CT images appeared in most patients at admission, supporting the three clinical criteria i.e. fever/respiratory symptoms, leukopenia and/orlymphopenia, and typical pulmonary imaging ûndings, for diagnosis of suspected COVID-19 [4]. We also found that CT imaging helped classify disease severity as a larger proportion of scans from critically ill patients revealed bilaterallung involvement compared with mild cases.

Abnormally elevated D-dimer levels were found in over one-third patients. Zhong et al. reported thattheproportionofpatientswithD-dimerover0.5μg/mlweresigniûcantlyhigherinseverecasesandthosewho

met a composite endpoint i.e. admission to an intensive care unit, the use of mechanical ventilation, or death,compared with mild cases [5]. A study by Cao et al. revealed increased odds of hospital-death associated withD-dimerconcentrationover1μg/mlatadmission[6].Theseûndingsmayreûecttheunderlyingimbalanceofthe coagulation system triggered by infection, which warrants continued study. Although most patients in thisstudy presented with mild symptoms (82.93%), one fourth needed oxygen therapies at least once throughout thediseasecourse.ThisresultwassimilartoothestudiesfromHubei province[7].

Although speciûc anti-SARS-CoV-2 drugs are currently unavailable,half of our patients receivedgeneralantiviral agentsontheûrstdayofhospitalization.

Lymphopenia was the most common laboratory abnormality at admission, occurring in nearly 56.27%of cases. Consistent with earlier results, this study found that inûammatory markers, such as CRP, were elevated[8,9]. D-dimer elevation was also an important feature of the disease, seen in over one-third of cases. About aquarter ofour patientswere oûeredanticoagulantdrugs.

The case-fatalityrate was lower (1.6%) than same day national data(2.11%)[10]. Studies have suggested that old age and comorbidities are risk factors for death [11]. In our cohort, 18.66% of patients wereover 65 years old, nearly30%had underlying disease. The main cause of death was ARDS and MODS.

We observe very few cases among children and pregnant women, which was consistent with studiesshowing a typically mild disease course among children and a lack of evidence for higher risks in pregnancy. Moreover,our research may not reûect the transmission and clinical features completely since undetected a symptomatic and mild cases may exist. Other limitations included small numbers of severe and critically illpatients for study and potential lack of representativeness of imported cases if extrapolated to urban communitytransmission.

V. CONCLUSION

Close monitoring of the vitals and lab parameters were carried out and as the disease progressed, our conservative approach changed to more aggressive mode backed by the knowled get h at getting down the viral load early is beneficial to the patient. We learnt that that simple care, assurance and proper communication go along way in building up the patient's confidence and encourage the mtocommunicate their discomfort at the earliest. Moreover, early nutritional supplementation also helps in maintaining the much-needed vital elements.

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CONFLICT OF INTEREST : None

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WINGING OF SCAPULA, AN UNCOMMON PRESENTATION OF A COMMON BONE TUMOUR : A RARE CASE REPORT

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ABSTRACT :

Neuromuscular causes of winging of scapula is well known , but winging and snapping of scapula may rarely be caused by space occupying lesion of the thoracic wall. Although osteochondroma of scapula is rare, it is most common neoplasm of scapula, and osteochondroma of ventral scapula may lead to pseudo-winging, snapping and rib erosion on the same side. Owing to its rarety, we report a case of osteochondroma of ventral scapula in a 18 year old female with complains of difficult scapulothoracic movement (snapping scapula) and pseudo-winging. After initial clinical, radiological investigations, in-toto surgical excisional biopsy was done and diagnosis confirmed histopathologically. In a 2 year follow-up there is no recurrence and symptoms of snapping and pseudo-winging disappeared completely. Pertaining to its asymptomatic nature & rare location, diagnosis of osteochondroma may be missed initially searching for some other neuro-muscular disorders and these cases should be reported to increase awareness.

CASE REPORT :

A 17 year old female presented with winging of left scapula since last 1 year with mild pain at rest and painful terminal restriction of movement with a cracking /grinding sound, with no radiating pain in arm.Her sleep was disturbed since last 2 months and had to lie in prone position for relief. Medial edge of scapula was found to be prominent at rest with normal orientation of superior and inferior angles and no changes on forward flexion of arms against resistance (Fig. 1). Patient was evaluated initially for neuromuscular causes , but etiology could not be ascertained to either of the muscle groups. Than on further examination, a swelling was found below the medial edge of scapula which was missed initially. Xray(Fig. 2) was suggestive of a bony lesion arising from the medial edge of scapula. MRI(fig. 3) confirmed the provisional diagnosis of osteochondroma, a space occupying lesion arising from medial and ventral aspect of scapula.Cartilage cap was less than 2cm.

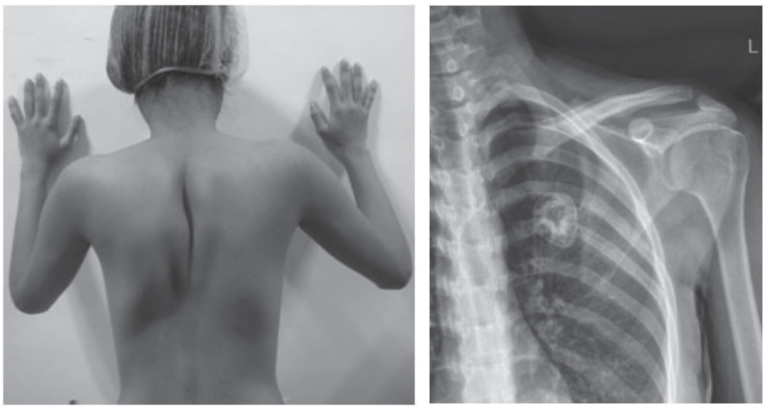


Fig. 2 : Xray showing a bony lesion arising from the medial border of scapula.

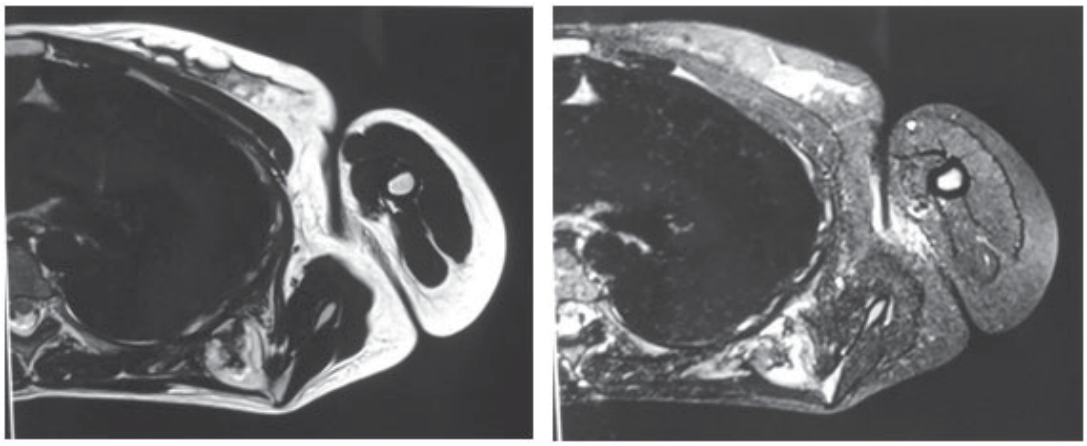


Fig. 3 : MRI images showing a pedunculated osteochondroma arising from medial and ventral border of scapula.

A pre-operative CT scan(fig. 4) was done and a pedunculated osteochondroma was seen arising from ventral aspect of scapula. An excisional biopsy was planned under general anaesthesia. Patient was placed in prone position and an incision was given 2-3 cm medial to the medial border of scapula. Rhomboid muscle was splitted along its fibres and retracted to expose the lesion. A large bursa was covering the lesion and excised (Fig. 5). After excising the bursa, scapula retracted to expose the tumour along its whole length. Meticulous dissection was done to avoid injury to thoracic wall and pleura. Lesion was excised extraperiosteally from its base (Fig. 6).

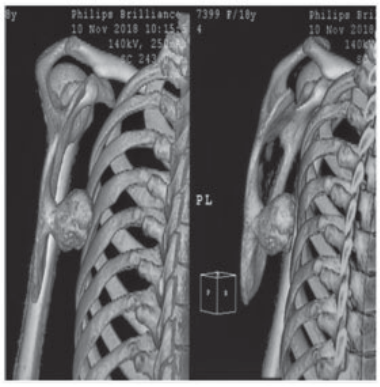


Fig. 4 : CT scan showing the pedunculated osteochondroma lifting the medial edge of scapula off the thoracic wall.

After excision a dead space was created and soft tissue was repaired under draincover to obliterate the dead space.

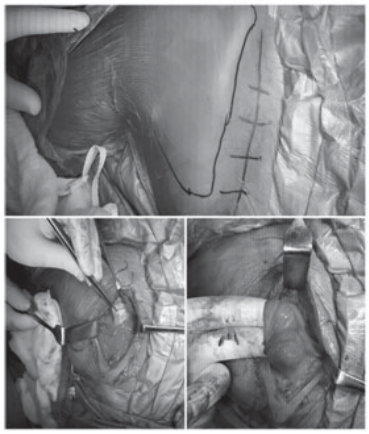
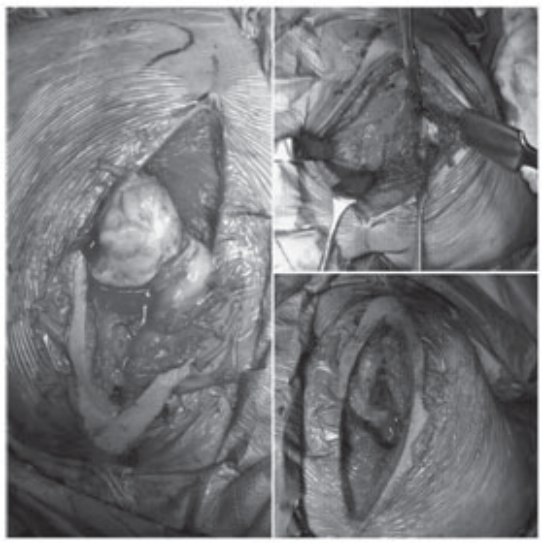


Fig. 5 (A,B,C) : Showing planned surgical incision and bursa.



6A 6B 6C

Fig. 6A: Mushroom shaped lesion, 6B : stalk of the lesion, 6C : after excision.

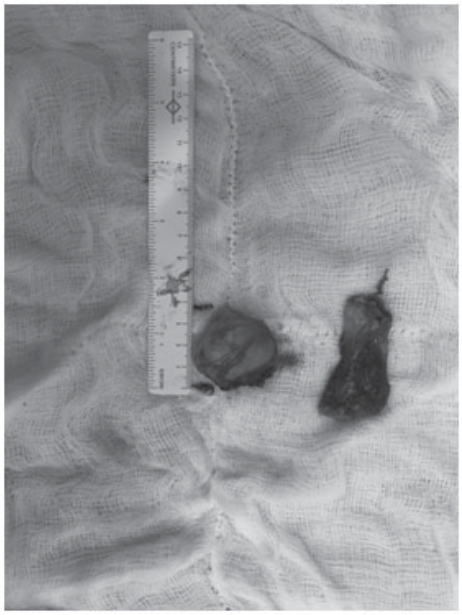


Fig. 7 : Excised lesion with stalk.

After the surgery patient was monitored in ICU and chest xray was done to evaluate any chest injury. Post-operatively diagnosis was confirmed histologically and patient was kept in arm pouch for three weeks and muscle strengthening exercise was started after 2 weeks on stitch removal.

After 2 years of follow up, the patient is doing well without any pain, no signs of recurrence and full ROM of shoulder.

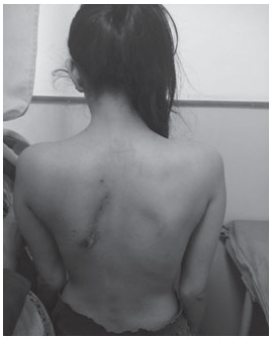


Fig. 9 : Follow-up clinical image, with similar appearing scapula bilaterally.

DISCUSSION :

Winging of scapula is mainly caused by neuromuscular causes and diagnosis is mainly clinical, sometimes supplemented with MRI cervical spine and electro-diagnostic studies. However, pseudo-winging of scapula is caused by a space occupying lesion of either ventral scapula or the rib cage posteriorly. Osteochondroma can appear in any bone which develops by endochondral ossification and mainly appears at metaphyseal region of growing long bones and cease to grow after skeletal maturity . Although osteochondroma is the most common benign bone tumour , it is rare in flat bones and even rarer in scapula. Although osteochondroma is rare in scapula, it is the most common benign bone tumour of scapula. It is mostly asymptomatic initially and causes symptoms later due to its mass effect on near-by structures . Our patient presented with static medial winging, chronic dull pain and terminal restriction of shoulder abduction with scapular snapping due to scapula-thoracic movement. Osteochondroma of scapula should be considered as a possible differential diagnosis in a young patient with these symptoms and in absence of any neuromuscular features . It is difficult to diagnose these cases clinically because of its ventral location and routine chest xray may fail to identify the source of origin of the lesion. CT scan helps to accurately map the boy anatomy of the lesion and MRI can measure the cartilage cap thickness, bursa formation and rib erosion. Complete surgical excisional biopsy should be considered in these cases because of uncertainity of its location, histological diagnosis, cosmetic deformity and possible bursitis and rib erosion. Careful surgical dissection is very important to avoid chest wall injury and soft tissue repair should be done meticulously to avoid dead space formation and possible scapula-thoracic dissociation post-operatively.

REPLANTATION OF SEVERED EXTREMITY

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Replantation refers to the surgical reattachment of a finger, hand, or arm that has been completely cut from a person's body. The goal of replantation surgery is to give the patient back as much use of the injured area as possible. In some cases, replantation is not possible because the part is too damaged.

The patient or family member must decide whether that amount of use justifies the long and difficult operation, time in the hospital, and months or years of rehabilitation.

Anatomic features that are specific to the amputation and not related to the patients health or history include the level and complexity of the injury. More distal injuries have better success in terms of function, but an exception is very distal replants where establishing circulation may be more difficult. Sharp injuries tend to do better than crushing or avulsion injuries. Crush and avulsive injuries tend to involve a wide path of tissue that is irreversibly damaged. Similarly, multi-level injuries do not do as well as single level injures.

Most important deciding factor for surgery is the ischemia time.

Ischemia time refers to the total time the part is lacking circulation and therefore not receiving oxygen. The longer the ischemia time the worse the prognosis. This is particularly true for warm ischemia where oxygen consumption and free radical formation is increase. For this reason parts are kept cool, but not frozen, on ice. Warm ischemia ranges from 6 to 8 hrs whereas cold ischemia ranges from 12 to 24 hrs depending on the level of amputation.

Cooling the amputated part can substantially increase the time that can elapse between injury and surgery. When preparing patients for transfer to a replantation center, emergency medical personnel will package the amputated limb or digits on ice in order to optimize the chance for successful replantation. Please do not forget to inform the Hospital with facilities of replantation so as to keep them on high alert and unnecessary time is not wasted

Transportation of the amputated part is of utmost important in the success of the Replantation.

The amputated part should be cleaned preferably with normal saline or tap water and wrapped with a clean moist towel and put in a plastic bag. This plastic bag inturn goes in another plastic bag containing ice. Care must be taken that ice should not come into direct contact with Ice.The amputation stump which will be bleeding should be given a compression bandage to stop bleeding.

The indications for replanation have broadened over the last decade, as we have gained experience attempting to salvage more complex injuries. The following are indications for replantation:

Amputations in children

Multiple finger and hand amputations

Thumb

Single finger injuries

Ring avulsion injuries

Because the surgery can take many hours, the stresses of anesthesia and vascular changes such as hardening of the arteries may substantially increase the risks of the procedure for the older patient and decrease the chances for replantation success.

There are a number of steps in the replantation process. First, damaged tissue is carefully removed. Then bone ends are trimmed before they are rejoined. This makes putting together the soft tissue on either side of the wound easier. Arteries, veins, nerves, muscles, and tendons are sewn back together . Areas without skin are covered with skin that has been taken from other areas of the body. Uncovered nerves, tendons, and joints may be covered by a free-tissue transfer, where a piece of tissue is removed from another part of the body, along with its artery and veins.

Patients are watched closely for the first 48 hours. The patient's room is kept very warm after surgery to keep blood vessels dilated and to prevent blood clot formation.

Blood thinning medication is required for up to a week after surgery.

Complete healing of the injury and surgical wounds is only the beginning of a long process of rehabilitation. Therapy and temporary bracing are important to the recovery process. From the beginning, braces are used to protect the newly repaired tendons and allow the patient to move the replanted part. Therapy with limited motion helps keep joints from getting stiff, muscles moving, and scar tissue to a minimum. Even after you have recovered fully, you may find that you cannot do everything you wish to do.

Some patients who have fully recovered from replantation surgery may need surgery later to reach full usage of the part. Some of the most common procedures are:

Tenolysis - frees tendons from scar tissue.

Capsulotomy - releases stiff, locked joints.

Tendon or muscle transfer - moves tendons or muscles to another spot so that they can work in an area that needs the tendon or muscle more.

Nerve grafting - replaces a scarred nerve or a gap in the nerves to improve how the nerve works.

Stay in the flow of life. You have many great gifts. Even with the best medical care, you need to be strong during the course of recovery. Remember that quality of life is directly related to your attitude and expectationsnot just regaining limb use.

A CLINICAL STUDY OF MULTIPLE MYELOMA WITH SPECIAL REFERENCE TO CLINICAL PRESENTATION IN A TERTIARY CARE HOSPITAL.

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ABSTRACT: MultipleMyeloma (MM) is a clonalB-celltumour of differentiated and usually slowly proliferating plasma cells. It accounts for about 1% of all malignant disorders. It has a strong correlation withage and the risk increases with age, peaking at about 60-70 years. In this study we have set out to determine thevarious signs and symptoms of the patients of multiple myeloma at the time of presentation. A total of 52 patients were evaluated from April 2015 to April 2016 in the department of Clinical Haematology, Gauhati Medical College and Hospital. Patients were put on different treatment regimens as per the institutional protocol and followed up for a period of 4 months. As described in literature most of the patients presented withsymptoms of bone pain, anaemia, infection and hyercalcaemia. This study also stages the patients according tothe Revised International Staging System and assesses the short term outcome of different treatment regimens. A greater number of patients presented with advanced stage disease (stage III-44.23%). Fluorescent in situhybridization (FISH) studies were conducted in 19 patients of which 52.93% fall in the low risk group and 21.05% fall in the high risk group. Three drug regimen showed better outcome compared to two drug regimen with increased number of patients demonstrating Stringent complete response (sCR) and Complete response (CR).

KEYWORDS : Multiple myeloma, FISH, Hypercalcaemia, clonal B-cell tumour

I. INTRODUCTION

Multiple myeloma (MM) is the second most common haematological malignancy. [1]. It is a B cellmalignancy characterized by clonal proliferation of plasma cells in the bone marrow and is associated with anincreased level of monoclonal protein in the blood and/or urine[2]. Although the disease remains incurable, outcomes have improved substantially in recent decades as a result of advances in treatment, including high-dose therapy and the availability of novel agents, as well as improvements in supportive care strategies[3]. 34 percent of Multiple Myeloma patients are asymptomatic at presentation with incidental abnormalities on total protein, creatinine, calcium, or hemoglobin laboratory panels[4]. Rare presentations include soft tissue orsolitary bone masses (plasmacytomas), hyperviscosity induced arterial infarctions or venous thrombosis, and concomit antamylo idosis with gastro intestinal symptoms, peripheral neuropathy, orcardiomegaly [5].

The incidence of Multiple Myelomavaries within different geographical ares. Multiple Myelomaoccurs at the highest incidence in African Americans and Pacific Islanders; intermediate in Europeans and North Americans whites; and lowest inpeople from developing countries including Asia. Chinese and Japanese populations have a lower incidence than whites [6]. In India, incidence of Multiple Myeloma varies from 1.2 to1.8 per 100,000.

Many studies have been published describing various aspects of the disease from Europe and the United States over the past 50 years. However, more data is required from India to study the disease course in our country. The earlier studies reported from India suggested low incidence of the disease. However, subsequent

reports in the late nineties have reported an increased incidence of the disease probably reflecting increased awareness, availability of better facilities for diagnosis or truly increased incidence[7].

There has been very few Indian studies which have evaluated patients with Multiple Myeloma. The present study will assess the magnitude of the disease in a tertiary medical centre in North Eastern India and study its clinical profile.

II. MATERIALS AND METHODS

The clinical and biochemical profile of the multiple myeloma patients are studied in this prospective study. Various presentations of the newly diagnosed multiple myelomapatients were recorded, different prognostic markers were evaluated and patients were staged as per the staging criteria. Wherever feasible risk stratification was donebasedon different chromosomal translocations which were assessed by fluorescent insitu hybridization (FISH) studies on bone marrow samples. Patients were put on different treatment regimens asper the institutional protocol and followed up for a period of 4 months. A total of 52 patients were evaluated from April 2015 to April 2016 in the department of Clinical Haematology, Gauhati Medical College and Hospital.

III. RESULTS AND OBSERVATION TABLE 1.

AGE DISTRIBUTION

Age(inYears)	Numberof Patients (n=52)	Percentage(%)
<30	2	3.84
30-39	6	11.54
40-49	10	19.23
50-59	18	34.61
60-69	12	23.08
70+	4	7.69

The mean age was 61.4 years. The peak frequency was in the 6th (34.61%) and 7th (23.08%) decades. Only 8patients (15.38%) were less than 40 years of age, while 4 patients (7.69%) were over the age of 70.

TABLE 2. SEX DISTRIBUTION

Sex	Number of patients(n=35)	Percentage(%)
Male	37	71.15
Female	15	28.85

Of the 52 patients, the rewere 37 males and 15 females, with a male to female ratio of 2.5:1.

TABLE 3. SYMPTOMS AT PRESENTATION

SYMPTOMS	YES (Present)	NO (Absent)	Percentage(%)
Bonepain	45	7	86.54
Anaemia (fatigue, malaise, palpitation, dizziness)	41	11	78.85
Fever	25	27	48.08

Symptoms of hypercalcaemia (polyuria, abdominalpain, nausea, vomiting)	14	38	26.92
Bleeding	5	47	9.61
Symptoms of renal disease (oliguria, facial puffiness, shortness of breath)	12	40	23.08
Spinal cord compression	13	39	25
Symptoms of hyperviscosity (visual disturbances, headache, seizures)	9	43	17.31

The most frequent presenting feature was bone pain, occurring in 86.54% of patients. Other common symptomswerethoserelatedto anaemia(78.85%), infection (48.08%) and hypercalcaemia (26.92%).

TABLE 4. SIGNS AT PRESENTATION

SIGNS	YES (Present)	NO(Absent)	Percentage(%)
Lyticbonelesions	40	12	76.92
Pathologicalfractures	9	43	17.31
Vertebralcompression	19	33	36.54
Plasmacytoma	9	43	17.31
Amyloidosis	2	50	3.85
Anaemia	44	8	84.61
Males Hb <13g/dl	31	6	83.78
Females Hb<12g/dl	13	2	86.66

Anaemia was the dominant clinical sign occurring in 44/52patients (84.61%). Anaemia was commoner infemales (86.66%) compared to males (83.78%). The radiological hallmark of myeloma is the presence of'punched out' lytic bone lesions, which was present in 76.92% of the patients. The most classical site ofinvolvement was the skull. Vertebral compression fractures were seen in 36.54 % of patients . Pathologicalfractures were less common (17.31%). The humeri and femurs were the typical sites of involvement. Amyloidoccurringinassociationwith myelomawasanunusualfinding.

TABLE 5. STAGE OF DISEASE (REVISED INTERNATIONAL STAGING SYSTEM)

R-ISS	NUMBER	Percentage(%)
I	10	19.23
II	19	36.53
III	23	44.23

The majority of patients presented with advanced stage disease (stage III – 44.23%), while early stage disease(stageI) onlyaccountedfor 19.23%ofthe patients.

TABLE 6. RISK STRATIFICATION TESTS

FISHPROBE		NUMBER(n=19)	ISS		
			I	II	III
Del 17p13	POSITIVE	3	0	1	2
	NEGATIVE	16	4	4	8
t(4,14)	POSITIVE	5	1	1	3
	NEGATIVE	14	5	3	6

TABLE 7. RISK STATIFICATION

RISK	NUMBER(n=19)	PERCENTAGE(%)
LOW	10	52.93
INTERMEDIATE	5	26.31
HIGH	4	21.05

FISH studies were conducted in 19 patients of which 52.93% fall in the low risk group and 21.05% fall in the high risk group. Of the 52 patients, 38(73.08%) patients fulfilled transplant eligibility criteria.

TABLE 8. Haemoglobin level:

Haemoglobin	Number of patients(n=52)	Percentage(%)
Hbe>10g/dl	9	17.31
Hb<10 g/dl	43	82.69
Hb<8.5g/dl	38	73.08

TABLE 9.Whitecell count

Whitecell count	Number of patients (n=52)	Percentage (%)
<4.0 $x10^9$/l	3	5.77
4.0-11$x10^9$/l	13	25
>11$x10^9$/l	19	36.54

TABLE 10. Platelet count

Platelet count	Number of patients (n=52)	Percentage (%)
<150$x10^9$/l	7	13.46
>400 xl0^9/l	0	

TABLE 11. ESR

ESR (mm/hr)	Number of patients (n=52)	Percentage (%)
<20	4	7.69
20-100	16	30.77
>100	32	61.54

The mean haemoglobin (Hb) at presentation was 9.38g/dl. In contrast to the low Hb, the white cellcount and platelet count was usually normal. Only 5.77 % of patients presented with a leucopenia and 13.46% with thrombo cytopenia,comparedto84.61% who manifested with anaemia. The ESR was raised above 20mm/hr in the vast majority of patients (92.30%). The mean ESR was 106mm/hr. A markedly elevated ESR(>100mm/hr) was found in almost two thirds of the patients (61.54%).

TABLE 12. ROUTINE BIOCHEMICAL TEST

- **BLOODUREA**

Blood Urea (mg/dl)	Number of patients (n=52)	Percentage(%)
<22	5	9.61
e”22	47	90.38

- **SERUMCREATININE LEVEL**

Serumcreatinine(mg/dl)	Number of patients (n=52)	Percentage(%)
<1.2	15	28.85
1.2-2	14	26.92
>2	23	44.23

- **SERUMCALCIUM (CORRECTED)**

SERUMCALCIUM (CORRECTED)(mg/dl)	Number of patients (n=52)	Percentage(%)
<8.5	3	5.77
8.5-11	32	61.54
>11	17	32.70

- **SERUMURICACID**

SERUM URIC ACID(mg/dl)	Number of patients (n=52)	Percentage(%)
<7.2	22	42.31
>7.2	30	57.69

- **SERUMALKALINEPHOSPHATASE**

ALKALINEPHOSPHATASE(U/L)	Number of patients (n=52)	Percentage(%)
<150	42	80.77
e>150	10	19.23

Hypercalcaemia (>11mg/dl)occurred in 32.70% of patients. Hyperuricaemia was common, being present in almost 2/3 (57.69%) of our patients. An elevated urea of >22mg/dl and creatinine of >1.2mg/dl (after rehydration) was evident in 90.38% and 71.15% respectively. Levels of beta 2 microglobulin >5.5mg/l were found in 61.53% at presentation. In keeping with the non-osteoblastic nature of myelomatous bone disease, the alkaline phosphatase was usually normal (<150U/l) in 80.77%. The LDH level was elevated in 48.08% of

thepatients The mean bone marrow plasma cell representation was 26.7%. 34.61% of the patients had more than 10% plasma cells, while in 38.46% a level of>30% was present.

TABLE 13 : TRANSPLANT ELEGIBILITY AND TREATMENT GIVEN

TRANSPLANT ELIGIBILITY	THERAPYSTARTED		
	BORTEZOMIB+DEXAMETHASONE	BORTEZOMIB+DEXAMETHASONE + THALIDOMIDE/LENALIDOMIDE	BORTEZOMIB+ DEXAMETHASONE+ CYCLOPHOSPHAMIDE
TRANSPLANT ELIGIBLE	6	22	10
TRANSPLANT INELIGIBLE	4	4	6

TABLE 14. BEST RESPONSE TO THERAPY AT THE END OF FOLLOW UP PERIOD(3MONTHS)

THERAPYSTARTED	Stringent complete response (sCR)	COMPLETERESPONSE (CR)	VERYGOOD PARTIALRESPONSE(VGPR)	PARTIALRESP ONSE (PR)	STABLEDISEASE(SD)
BORTEZOMIB+DEXAMETHASONE	0	1	3	5	1
BORTEZOMIB+DEXAMETHASONE+ THALIDOMIDE/LENALIDOMIDE	4	6	13	2	1
BORTEZOMIB+DEXAMETHASONE+CYCLOPHOSPHAMIDE	2	5	8	1	

Accordingly, 10 (19.23%) patients were subjected to two drugregime, VD (BORTEZOMIB+ DEXAMETHASONE), 4 2(80.77%) patients received three drug regime, of which 26(50%) received VTD(BORTEZOMIB+DEXAMETHASONE+THALIDOMIDE/LENALIDOMIDE) and 16 (30.77%) received VCD (BORTEZOMIB+DEXAMETHASONE+CYCLOPHOSPHAMIDE). Shortterm response was assessed at 4 months. 40% patients of two drug regime, VD showed CR (10%) and VGPR (30%) where as 88.46% of VTD regimedemonstrateds CR (15.38%), CR (23.07%) and VGPR (50%). 93.75% of the patients who received VCD regime showeds CR(12.5%), CR(31.25%) and VGPR(50%). Of the 38 transplant eligible patients, 16(30.77%) patients subsequently, underwent successful autologous stem celltransplantation(ASCT).

IV. DISCUSSION

In the present study among patients with Multiple Myeloma, majority of patients were in the age group 50-59 years at the time of diagnosis of their illness constituting 34.61% of total. Advani et al(1978) also reported majority of patients to be in the age group 50-59 years of age[8].

In the present study among Multiple Myeloma patients 71.15% were male and 28.85% were female. The M:F ratio was 2.47: 1. Similar male predominance was reported by Advani et al (1978), National Cancer Registry Programme Statistics, P. Kaur et al (2004)[8,9,10].

In the present study among Multiple Myeloma patients, most common clinical features were bone pain (86.54%) and symptoms related to anaemia (generalized weakness andincreased fatigability) (78.85%). Instudies

by Gupta et al (1995) and Kyle et al (2003), 79% and 58% patients respectively had bone pains at diagnosis[11,12].

In the present study the mean corrected calcium level is 11.08 mg/dl. 32.70% of the Multiple Myeloma patients has corrected serumcalcium level >11mg/dl. Makkaretal (2014) reported serum calcium level of >11mg/dlin41.66% of Multiple Myeloma patients [13].

In the present study a feature common to the Multiple Myeloma patients seen islate stage presentation, with advanced stage disease. Based on the International Staging System (ISS) the majority of patients with Multiple myeloma presented with stage III disease (44.23%) where as patients with stagelandstagell constitute 19.23% and 36.53% of the patients. Similar observation was made by P. Kaur etal 2004) who reported high erincidence of stage lll disease (64.3%) as compared to stage ll (28.5%) and stage l (7.2%)disease. Greippetal (2005) also reported 28%, 33% and 39% of patients presenting with stage l, ll, and lll disease respectively [14]. In the present study Fluorescent in situ hybridization (FISH) to detect adverse translocationt (4,14) and del17p13 was done in 19 patients with Multiple Myeloma and risk stratification was done using combination of ISS and FISH.t (4,14) was found in 5/19(26.31%) patients and del17p13 was found in 3/19(15.79%) patients with Multiple Myeloma. Accordingly, majority (42.10%) of Multiple Myeloma patients were having low risk at presentation. Intermediate risk was present in 36.84% of patients and high risk in 21.05% of the patients. Similar observations were made in various other studies. In the study done by Avet-Loiseau Hetal (2013) patients with low, intermediate and high risk disease constituted 51%, 29% and 20% of total respectively[15]. Boyd K Detal (2012) reported high, intermediate and low risk in 38%, 48% and 14% of their patients respectively [16].

Neben K etal(2010) observed 42%, 44% and 14% of their patients had low, inter mediate and high risk disease [17].

In the present study response to therapy was assessed after 4 cycles of induction chemo therapy. In the patients receiving VD, 10% patients achieved CR, 30% patients achieved VGPR and 50% patients achieved PR. In the three drug regime, patients receiving VTD achieved sCR/CR, VGPR and PR in 30.77%, 46.15% and 15.38% of the Multiple Myeloma patients. Patients receiving VCD achieved sCR/CR, VGPR and PR in 18.75%, 31.25% and 31.25% of the Multiple Myeloma patients. Moreau P et al (2011) observed that patients receiving 4cycles of VD or VTD, the complete response (CR) rate was the same in both groups (13% in the VTDD arm, 12% in the VD arm)[18]. However, the CR plus very good partial response (VGPR) rate was significantly higher in the VTD arm as compared to VD group. (49% vs 36%).Rosinol et al (2012) reported that in patientsreceiving VTD regime, 35%, 60% and25%of patients achievedsCR/CR, VGPR and PR respectively[19]. Inthe study done by Moreau et al (2011) patients on VTD regime, 31%, 49% and 39% patients achieved sCR/CR, VGPR and PR respectively [20].

V. CONCLUSION

In the present study an attempt has been made to study the clinical spectrum and response to treatment with short term follow up in patients diagnosed to have Multiple Myeloma. Among a total of 52 patients, thepeak incidence was seen in the 5th and the 6th decades of life.A slight male predominance was seen. Presenting complaints in majority of Multiple Myeloma patients were bone pain, increased fatigability, fever, polyuria,lower limb paresthesias and oliguria. Nearly all patients were anaemic and lytic bone lesions were evident inthree fourth of the patients. Majority of the Multiple Myeloma patients presented with advanced disease and nearly half of the patients had renal impairmentat the time of presentation. In those patients in whom Fluorescent in situ hybridization was done, half were having low risk at presentation. Majority of the patients were transplant

eligible at the time of presentation. All the patients were managed with bortezomib based regimen. Most patients were treated with three drug regimen consisting of bortezomib, dexamethasone and either thalidomide or cyclophophamide while 11.54% of the patients received two drug regimen containing bortezomib and dexamethasone. Stringent complete response (sCR), Complete Response (CR) and Very Good Partial Response (VGPR) was better achieved with three drug regimen as compared to two drug regimen and that too in those receiving bortezomib, dexamethasone and thalidomide.

Since this was a hospital based study with a small sample size, a larger study recruiting more patients with a longer duration of follow up is necessary for a better understanding of the disease and to arrive at adefinite conclusion.

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