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Original Article

Somatization Pattern of Underlying Anxiety and Depression of Wives of Men Working or Living Abroad from Sylhet, Bangladesh: a Cross-Sectional Study

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Abstract:

Background: Wives of men working or living abroad are at a higher risk of developing anxiety and depression. Many of these women present to medical facilities with somatization symptoms rather than the typical features of mental health disorders. This study aims to explore the pattern of somatization among this specific group of women.

Material and method: The study initially included 213 women from Sylhet, Bangladesh, whose husbands were working or living abroad. These women presented to the Medicine inpatient department of Sylhet Women's Medical College. After symptom-specific investigations, 197 participants were selected who had no organic diseases but exhibited anxiety, depression, or both, based on the Mini-Mental State Examination (MMSE) and Hamilton scores. A descriptive analysis was conducted to evaluate the pattern of their somatization symptoms.

Results: Out of the 197 participants, 103 (52%) had anxiety, 88 (45%) had depression, and 6 (3%) experienced both anxiety and depression. The most common somatization symptom was tension-type headache (27.9%), followed by fibromyalgia (22.33%), sleep disturbances (12.18%), and non-ulcer dyspepsia (9.1%). Cardiac-related somatization symptoms included palpitations (8.1%), shortness of breath (5.58%), and chest pain (4%). Additionally, 11 participants (5.8%) exhibited multiple somatization features, such as a combination of headache, body ache, chest pain, and palpitations.

Conclusion: Women whose husbands live or work abroad are particularly vulnerable to mental health challenges, with somatization often obscuring the diagnosis. This presents significant diagnostic and therapeutic challenges for healthcare professionals.

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Key words: anxiety, depression, somatization, husbands living abroad, wives of migrant workers of Sylhet.

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Introduction:

Since the early 20th century, people from Sylhet have been migrating abroad in search of employment. This trend has been more prevalent among men than women.

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In most cases, men migrate alone, leaving their families behind. This separation often leads to significant emotional strain for both the men and their families.[1] A recent study focusing on the wives of men living abroad from Sylhet revealed a notably high prevalence of anxiety and depression among these women.[2]Somatization, the expression of psychological distress through physical symptoms, is a common phenomenon often linked to underlying mental health conditions such as anxiety and depression.[3] This pattern is particularly evident in populations facing unique stressors, such as individuals living in socially or economically challenging circumstances.[4] Interestingly, a significant

number of patients with somatization tend to seek care in medical inpatient and outpatient facilities rather than psychiatric services. They often present with physical symptoms such as breathlessness, headaches, palpitations, chest pain, abdominal pain, and similar complaints, but without an underlying organic cause.[5,6]The wives of men working or living abroad experience a distinct set of challenges, including social isolation, increased household responsibilities, and financial or emotional uncertainties.[7] These factors can exacerbate psychological distress, often manifesting as somatic complaints. Sylhet, a region in Bangladesh with a significant number of men working overseas, offers a unique demographic for examining this phenomenon.[1]The social and cultural context of this area may influence the mental health and coping mechanisms of women left behind, making them susceptible to unrecognized anxiety and depression.[5,7] However, their mental health challenges are often overlooked, as physical symptoms tend to dominate the clinical presentation in such cases. This cross-sectional study aims to explore the somatization patterns of underlying anxiety and depression among the wives of men working or living abroad from Sylhet. By identifying the prevalence and nature of these symptoms, the study seeks to highlight the mental health burden in this population and provide insights for targeted interventions. Understanding this association is crucial for improving mental health care access and quality for these women, contributing to their overall well-being.

Materials and Method:

Study design: This cross-sectional observational study was conducted in the Medicine inpatient department of Sylhet Women's Medical College from July 2021 to December 2022. The participants were individuals who sought medical care at the facility from various regions of the Sylhet division.

Ethical clearance: Approval was obtained from the Institutional Review Board (IRB).

The study population: The study population comprised 289 wives of men working or living abroad from Sylhet, Bangladesh, who were admitted to the Medicine inpatient department with various medical symptoms (Figure 1). Of these, 38 women were excluded after investigations revealed underlying organic diseases. Among the remaining 251 women initially selected, another 38 were excluded for the following reasons: refusal to provide consent for enrollment (n=10), presence of chronic debilitating diseases (n=11), or a diagnosis of anxiety or depression before marriage (n=17). This left 213 women eligible for the study.

These 213 participants underwent evaluations using the Mini-Mental State Examination (MMSE) and Hamilton scoring for anxiety and depression. Sixteen women did not fulfil anxiety or depression scores and were subsequently excluded. The final study population, therefore, consisted of 197 women whose husbands were working or living abroad and who exhibited varying degrees of anxiety and/or depression.

Consent: Informed consent was obtained from all participants at the initial enrollment.

Diagnostic aids: Detailed history, physical examination findings, and investigation results were documented using pre-structured data sheets. Investigations were planned according to the presenting somatic features (Table 1) to exclude organic causes. Women presenting with body aches but normal investigation results were diagnosed with fibromyalgia based on specific criteria (Table 2). Irritable bowel syndrome (IBS) was primarily identified using the Rome IV criteria (Table 3). Non-ulcer dyspepsia or functional dyspepsia was diagnosed on the basis of dyspeptic symptoms like upper abdominal pain, bloating, belching etc. but with normal upper gastrointestinal endoscopy. Tension-type headaches were diagnosed based on clinical criteria[8], rather than extensive investigations, which were conducted only in selected cases.

Statistical analysis: All data were collected, recorded, and analyzed using descriptive analysis in Microsoft Excel 2013.

Results:

Of the finally selected 197 participants 103 (52%) had anxiety, 88 (45%) had depression and, 6 (3%) had both anxiety and depression (Figure 2). The ratio between anxiety and depression among participants was 1.2:1. The age of participants (average 32 years) ranged between 19-50 years (Figure 3). They were from

different districts of Sylhet division and both from rural and urban areas. The majority of them (39%) had their husbands working in the KSA. Severity assessment of anxiety and depression by Hamilton scoring (Figure 4) revealed that moderate symptoms were common among participating women with anxiety (53.39%) and depression (53.4%). The commonest somatization symptom among the participants was tension type headache (27.9%), followed by fibromyalgia (22.33%), sleep disturbance (12.18%) and, non-ulcer dyspepsia (9.1%). Somatization with cardiac symptoms were present in 35 participants (17.77%). They had palpitation (8.1%), shortness of breath (5.58%) or, chest pain (4%). At least 11(5.8%) participants exhibited multiple somatization features with variable combinations of headache, body ache, abdominal pain, chest pain, and, palpitation (Figure 5). Unexplained restlessness and generalized weakness with normal test findings were found in 4 and 3 participants respectively. Of another three participants, one had functional dysphagia with normal endoscopy, one had globus pharyngeus and one had functional tremor.

Figure 1: Flow chart of selection of study population

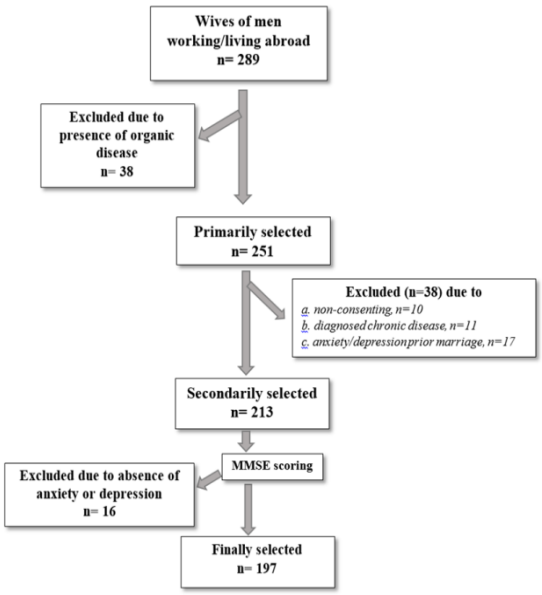


Table 1: Investigations for different somatic features

	Somatic feature	Investigation
1.	Chest pain	ECG, chest X-ray, Troponin I, Echocardiography 2D and M-mode
2.	Breathlessness	ECG, chest X-ray, Troponin I, Echocardiography 2D and M-mode
3.	Palpitation	Complete blood counts, ECG, chest X-ray, serum TSH
4.	Headache	CT scan head, other relevant
5.	Body ache	Complete blood counts, CRP, RA test, ANA, anti-CCP antibody, serum vitamin D, other relevant
6.	Dyspepsia	USG whole abdomen, endoscopy of upper gastro-intestinal tract
7.	Abdominal pain	USG whole abdomen, serum lipase, endoscopy of upper gastro-intestinal tract, CT scan of abdomen, other relevant
8.	Vomiting	USG whole abdomen, serum creatinine, serum ALT, serum electrolyte, endoscopy of upper gastro-intestinal tract, other relevant
9.	Diarrhea	USG whole abdomen, serum TSH, serum electrolyte, stool routine examination, colonoscopy, other relevant
10.	Constipation	USG whole abdomen, serum TSH, colonoscopy, other relevant

Table 2: Fibromyalgia diagnostic criteria[9]

Serial	Pain site
1	Left upper region, including shoulder, arm or jaw
2	Right upper region, including shoulder, arm or jaw
3	Left lower region, including hip, buttock or leg
4	Right lower region, including hip, buttock or leg
5	Axial region, which includes neck, back, chest or abdomen
Diagnosis: Widespread pain throughout the body for at least three months. The pain must be present in at least four of the above five areas	

Table 3: Rome IV criteria for irritable bowel syndrome[10]

Serial	Pain site
1	Recurrent abdominal pain or discomfort
2	Associated features:
	i) Pain and discomfort related to defecation
	ii) A change in the frequency of defecation
	iii) A change in stool consistency
Diagnosis: Recurrent abdominal pain on an average at least one day per week in the last three months with at least two associated features	

Figure 2: Prevalence of anxiety and depression among participants

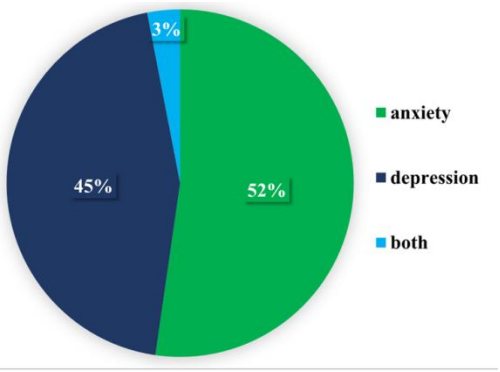


Figure 3: Age distribution of participants with somatization (n=197)

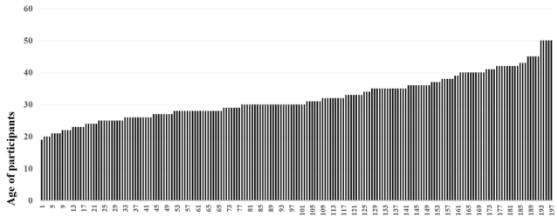


Figure 4: Severity of anxiety and depression among participants

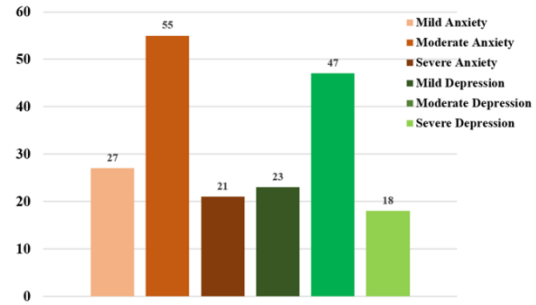
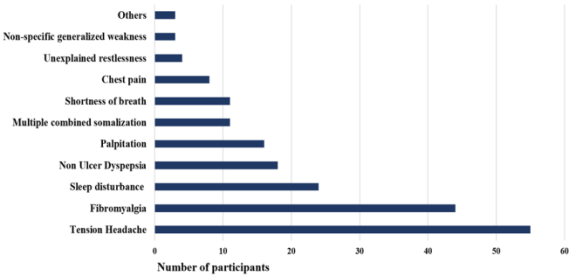


Figure 5: Prevalence of different somatization symptoms among participants



Discussion

The characteristic features of anxiety include excessive apprehension and worry, often accompanied by physiological hyper arousal and behavioral avoidance.[11]Depression, on the other hand, is defined by pervasive low mood, anhedonia, and cognitive impairments such as feeling of worthlessness and suicidal ideation.[12] Sometimes both the mental conditions can co-exist in the same individual[13] as was found in 3% of participants in this study. Somatic symptom disorders include a wide spectrum of conditions including somatization disorders, conversion disorders and, pain disorders secondary to psychological issues.[13,14] Somatization involves the manifestation of physical symptoms of any system that lack a clear medical explanation or identifiable organic cause, often associated with underlying psychological distress. Anxiety, depression, and somatization represent critical domains of psychopathology that significantly influence an individual's psychological, physical, and social functioning[3,15]. The interplay between these disorders has profound implications for clinical diagnosis, treatment, and the broader understanding of mental health.

This study critically analyzed the patterns and prevalence of various somatic symptoms among a specific group of women whose husbands lived or worked abroad and who exhibited suggestive Hamilton scores for anxiety and/or depression. Generally, 20-30% of individuals with anxiety display somatization[15,16], whereas Chinese studies reported such features in only 7.4% of anxious patients[17,18]. In the present study, all participants were admitted to the Medicine inpatient department rather than the Psychiatry department, indicating that somatic symptoms were the primary reason for their hospitalization.

Headaches, body aches, chest pain, palpitations, breathlessness, abdominal pain, diarrhea, constipation, sleep disturbances, and sweating are common somatic symptoms often classified as 'functional' in patients with underlying anxiety and depression. These symptoms can overlap with genuine systemic organic conditions, intensifying patient distress and

perpetuating a cycle of emotional and physical discomfort.[14,19]In this study, tension-type headaches were the most frequently reported somatic symptom among the participants, a finding consistent with their common occurrence in individuals with depression and anxiety.[20] Factors such as sleep deprivation, stress, and caffeine consumption may contribute to these headaches.[21]However, presence of these factors were not explored in the present study. Notably, none of the participants exhibited functional neurological disorder or conversion disorder, which are reportedly common among individuals under stress.[22] This may be because such patients are more likely to seek care from neuropsychiatric specialists rather than general medicine departments.

Fibromyalgia, which is twice as common in women as in men, is highly prevalent among individuals with anxiety and depression, with the risk being notably higher in those suffering from depression. The likelihood of fibromyalgia increases by up to 90% in cases of depression.[22,23,24]In the present study, 22.3% of participants with anxiety, depression, or both met the criteria for fibromyalgia, making it the second most common somatic symptom among the group. This study, conducted exclusively on women, demonstrated a stronger association of fibromyalgia with depression (92%) compared to anxiety.

Although all necessary investigations were carried out in this study to rule out organic differential diagnoses, extensive testing is not recommended when there is sufficient evidence to support a diagnosis of anxiety, depression, and somatization.[25,26] Studies have shown that 33–44% of patients undergoing coronary angiograms for chest pain have normal results and are subsequently diagnosed with panic disorders or related conditions.[27] A thorough understanding of somatic symptoms in patients with anxiety and depression is crucial, as it helps avoid unnecessary treatments and reduces the risk of polypharmacy.[28]

Theoretically, irritable bowel syndrome (IBS) is strongly associated with anxiety and depressive disorders.[29] However, in this study, the majority of participants with gastrointestinal somatization presented with functional dyspepsia. Six participants reported occasional diarrhea, and two experienced constipation, but none met the Rome IV criteria for an IBS diagnosis.[10]

The factors contributing to anxiety and depression in the studied group of women were complex, and the reasons for their presentation with somatic symptoms remain only partially understood. A recent study from the same region of Bangladesh reported a higher prevalence of anxiety and depression among women whose husbands work abroad.[2] Consistent with these findings, this study revealed that more than half of the participants experienced anxiety. Furthermore, emerging concepts such as cyberchondrias is, pathological health anxiety, and illness anxiety disorder are being explored in this field, providing valuable insights into potential underlying mechanisms.[30]

To gain a deeper understanding of the complexities of anxiety, depression, and somatization among women facing family-related stress, future research should prioritize longitudinal studies to explore social and physical causal pathways, as well as shared vulnerabilities. Advances in neuroimaging and biomarker research could further enhance the identification of trans diagnostic mechanisms underlying these conditions, paving the way for the development of personalized therapeutic interventions.[6,30,31]

Conclusion

Anxiety, depression, and somatization form a triad of closely interrelated conditions with profound implications for individuals and healthcare systems alike. Their shared pathophysiological mechanisms and high rates of co morbidity highlight the critical need for integrated approaches to diagnosis and treatment. Women whose husbands live or work abroad are particularly vulnerable to various mental health challenges. In this group, somatization often presents as a misleading factor, creating significant diagnostic and therapeutic challenges for healthcare professionals.

Limitation: Findings in this study were not compared with a control group.

Conflict of interest: It was a self-funded research and authors declare no conflict of interest.

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Original Article

Guillain-Barre Syndrome in North-Eastern population of Bangladesh: Electrophysiologic Pattern from the largest local cohort

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Abstract:

Objective: To determine the pattern of electrophysiologic findings in Guillain-Barre Syndrome (GBS) patients in north-eastern Bangladeshi population.

Methods: This observational study was carried out at neurophysiology laboratory in a specialized center of Sylhet with the diagnostic facility for nerve conduction study, from July 22- July 2024, Total patient was 248. Data were collected from the patients who underwent NCS with a provisional diagnosis of GBS.

Result: A total of 248 patients, mean age of 43 ± 13.24 years and a slightly male dominance (57.4%) were evaluated. Alost half of the patients (43%) reported a preceding diarrheal illness. Ascending motor paralysis was observed in majority of the patients (86%), and 2% were identified as Miller Fischer variant. In the nerve conduction study, majority (36.7%) were found to have motor Axonal polyneuropathy involving both Peroneal nerves, followed by Motor Sensory Axonal Polyneuropathy among 34.3%. Acute motor axonal polyneuropathy (AMAN) was the most frequent subtype (60.9%), followed by Acute motor sensory axonal polyneuropathy (AMSAN) in 34.3% and Acute inflammatory demyelinating polyneuropathy (AIDP) in only 4.8%.

Conclusion: GBS might have some regional variations despite having the demographic as well as clinical similarity. With AMAN being the predominant subtype, AIDP is much less common in north eastern Bangladeshi population.

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Key words: GBS, peroneal neuropathy.

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Introduction:

Guillain-Barré syndrome (GBS) is an acute, mostly fulminant polyradiculoneuropathy that manifests with symmetrical muscle weakness with or without sensory symptoms and signs, resulting from immune-mediated nerve damage[1,2]. Electrodiagnostic studies helps in classifying the disease which also have prognostic implications[2]. The electrophysiologic evidence of loss of axon or myelin not only have immunologic implications but also have subsequent therapeutic decision making and prognosis of the patient.

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An insight into electrophysiologic pattern of GBS for Bangladeshi population has already been provided by Badrul Islam et al[1]. But the cohort in that study was taken only from in tertiary hospital in Dhaka city which might not reflect the regional variation in disease pattern. The International GBS Outcome Study (IGOS) has provided enough evidence that factors related to geography have a major influence on clinical phenotype, disease severity, electrophysiological subtype, and outcome of Guillain-Barre ´ syndrome[3]. This IGOS group had classified three regional patterns; namely European/ American, Asian and Bangladeshi. It is well recognized that acute inflammatory demyelinating polyneuropathy (AIDP) variant is frequent in Europe and the USA, whereas, acute motor axonal polyneuropathy (AMAN) in Asia[3].

But they did not sub classify the pattern in different regions of Bangladesh[3].

Mathew et Al have shown the influence of climate on occurrence of GBS[4]. There has been variation in climate in north eastern part of Bangladesh from the rest[5]. So, in this study we tried to explore the regional pattern of GBS in north eastern part of the country.

Methods:

Patient

This study included a cohort of 248 GBS patients presenting between July 2022 to June 2024 in a tertiary care hospital in Sylhet, Bangladesh (Mount Adora Hospital). All patients who matched the inclusion criteria were enrolled. Informed written consent was taken and ethical approval were obtained from institutional review board. Each patient was reviewed by the first author. Relevant history and proper physical examination was done. Later the patients were finally evaluated and classified by consultant neurologist. A diagnosis of GBS was made according to the National Institute of Neurological Disorders and Stroke criteria for GBS[6,7]. Clinical and electrophysiologic data were recorded according to standardized protocol.

Nerve conduction Studies (NCS)

NCS was done using a standardized protocol on one side, using a Cadwel EMG system (USA). Motor nerve study and F waves analysis with 10 distal stimuli were recorded for the median nerve (wrist and elbow elbow-abductor pollicis brevis muscle), ulnar nerve (wrist, below elbow, above elbow-abductor digiti minimi), Deep branch of fibular nerve (below head of Fibula, popliteal fossa- extensor digitorum brevis muscle), and tibial (ankle, popliteal fossa-abductor hallucis muscle). antidromic Sensory nerve study was evaluated in the median nerve (wrist-second digit), ulnar nerve (wrist-fifth digit), and sural nerve (at lower lateral calf-behind lateral malleoli). Amplitude, area, duration of negative portion of compound muscle action potential (CMAP), distal motor latency (DML), motor conduction velocity (MCV) per segment, F-latency, amplitude of the negative portion of the sensory nerve action potential (SNAP). Cut-off values of different parameters were defined according to the requirements for each criteria. Criteria of Hadden and Uncini, requirements evaluated as ''at least one of the following in at least two nerves" was interpreted where relevant feature was to be present in at least two nerves.

Data interpretation and Analysis

Demographic information and disease profile were assessed properly. Data analysis was performed with the help of Statistical Package for the Social Sciences (SPSS) version 19. Appropriate statistical methods were applied for data analysis. We compared different variables with 95% confidence interval (CI) where p value $\leq 0.05$was considered as significant.

Results:

Table-1: Demographic and clinical features of the GBS patients (N= 248)

Demographic/Clinical Feature	Value/ Percentage
Age	
Mean	43 ± 13.24 years
Gender	
Male	52.4
Female	47.6
Preceding Illness	
Diarrheas	43%
Respiratory Infection	14%
Presentation at onset	
Ascending paralysis	86%
Descending paralysis	12%
Miller Fischer	2%
Autonomic dysfunction	13%
Cranial Nerve deficit	56%
Sensory deficit	7%

The study included a cohort of 248 GBS patients, with mean age of 43 ± 13.24 years and a slightly male dominance (57.4%) (Table-1). All most half of the patients (43%) reported a preceding diarrheal illness while only 14% had respiratory infection. Ascending type of motor paralysis was observed in majority patients

(86%), whereas only 12% had a descending pattern and 2% were identified as Miller Fischer variant (Table-1). More than half of the patients had associated cranial nerve palsy, while only 13% had autonomic dysfunction and 7% had objective sensory deficit (Table-1).

Table-2: Findings in nerve conduction studies (N= 248)

Findings	Percentage
Motor Sensory Axonal Polyneuropathy	34.3%
Left Peroneal Motor Neuropathy	1.2%
Motor Neuropathy involving both Peroneal nerves	2.4%
Motor Axonal polyneuropathy involving both Peroneal nerves	36.7%
Bilateral Axonal Peroneal Neuropathy	12.9%
Demyelinating Motor Neuropathy	4.8%
Motor Axonal Polyneuropathy	8.5%

In the nerve conduction studies, majority (36.7%) were found to have motor Axonal polyneuropathy involving both Peroneal nerves, followed by Motor Sensory Axonal Polyneuropathy among 34.3% (Table-2). Bilateral Axonal Peroneal Neuropathy (12.9%), Motor Axonal Polyneuropathy (8.5%), Demyelinating Motor Neuropathy (4.8%), Motor Neuropathy involving both Peroneal nerves (2.4%), Left Peroneal Motor Neuropathy (1.2%) were also reported (Table-2).

Table-3: Electrophysiologic types of GBS (N= 248)

Findings	Percentage
Acute inflammatory demyelinating polyneuropathy (AIDP)	4.8%
Acute motor axonal polyneuropathy (AMAN)	60.9%
Acute motor sensory axonal polyneuropathy (AMSAN)	34.3%

Acute motor axonal polyneuropathy (AMAN) was the most frequent subtype (60.9%), followed by Acute motor sensory axonal polyneuropathy (AMSAN) in 34.3% and Acute inflammatory demyelinating polyneuropathy (AIDP) in only 4.8% (Table-3).

Discussion:

This is the only study reporting a GBS cohort from North Eastern part of Bangladesh. We have found a considerable difference in several characteristics of GBS patients in North Eastern region than the data reported from the center. Although the demographic characteristics are similar to the reports of Islam B et al[1], Hossain et al[2], Islam MR et al[8]. But there was considerable difference in history and clinical presentation across different studies conducted in Bangladesh.

Our reports coincided more with the findings of Islam B et al[1]. Out cohort had a H/O of preceding diarrheal illness among almost half of the patients whereas Islam MR et al[8] reported only among one third of the patients. Similarly, the percentage of patients presenting with ascending paralysis were two times higher than those of the above mention study. This might be attributable to the lower number of study population and selection bias from a single center. The pattern of cranial nerve involvement, autonomic dysfunction and sensory involvement were more or less similar across different studies.

In line with the reports of largest cohort of GBS patients of Bangladesh, AMAN was the commonest subtype of GBS in North Eastern population[1]. But surprisingly enough, Hossain et al[2] and Islam MR et al[8] reported a higher burden of AIDP (51% AND 54%) in their cohort. The probable explanation would again be center specific selection bias and much smaller number of the study population.

In this study, electrophysiologic pattern of nerve involvement were also investigated and reported. Motor Axonal polyneuropathy involving both Peroneal nerves were the commonest electrophysiologic sign of involvement. But other studies have not reported this specific signs of involvement.

This study has some limitations. The major limitation would be lack of involvement of all

the regional clinics. Secondly, there was no long term follow up data. But this is the only study that focused on specific regional population which the actual strength of the study along with the considerable number of GBS patients in the cohort.

Conclusion:
Although the demographic and clinical presentations are similar, GBS might have some regional variations. There are some variations in preceding history and mode of clinical presentations in different reported studies. Similar to the largest reported cohort AMAN was the predominant subtype, while AIDP is much less common in north eastern Bangladeshi population.

Conflict of interest: This research project was not funded by any group or any institution and there was no conflict of interest.

Authors Contribution:
Rahat Amin Chowdhury was involved in planning the study, setting the methodology, performing NCS and data collection for this study. ATM Hasibul Hasan was involved in data analysis, interpretation and manuscript writing. Others involved in consultation and data collection. All the authors have read and approved the manuscript.

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Original Article

Intravenous Fluid Therapy in neonates receiving phototherapy for Hyperbilirubinemia - A randomized control trial

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Abstract:

Background: Most newborn babies experience jaundice after birth. In most cases, jaundice is benign; however, infants must be supervised since bilirubin may be dangerous to their health. Acute bilirubin encephalopathy or kernicterus is the potentially fatal side effect of extreme hyperbilirubinemia. Therefore, the treatment of neonatal jaundice or hyperbilirubinemia is crucial.

Method: This randomized controlled study was conducted in the department of Pediatrics, Sylhet Women's Medical College, Hospital in order to determine the requirement of intravenous fluid therapy in neonatal jaundice. By simple random sampling, 300 neonates with non-hemolytic jaundice were enrolled. 134 neonates were allocated to Group A, and they received intravenous (IV) fluid along with phototherapy. Another 166 neonates were included in Group B who received phototherapy only. Total serum bilirubin (TSB) (Both direct and indirect) was measured initially and every 24 hours during treatment. All the neonates were allowed to breastfeed during the treatment. The hydration status of neonates was checked at the beginning of the study, then every 8 hours for the next 24-72 hours. All the data were processed and analyzed in SPSS version 26. Chi-square test and independent sample 't' test were used. p value < 0.05 was considered as significant.

Result: The serum total, direct and indirect bilirubin levels of the neonates were similar (p>0.05) in both groups. Upon discharge, total serum bilirubin levels were reduced earlier in infants who received intravenous supplementation in conjunction with phototherapy, but the result was not significant (p>0.05). The clinical outcome of the neonates was insignificant. The discharge period for neonates in both groups was comparable (p>0.05).

Conclusion: In healthy term neonates with hyperbilirubinemia, intravenous fluid therapy does not provide significant benefits during phototherapy.

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Introduction:

A newborn's skin and sclera turn yellowish in neonatal jaundice, which is caused by bilirubin.[1] Hyperbilirubinemia refers to an increase in the serum bilirubin level in a newborn greater than 5 mg/dl. Jaundice develops in about 60% of term babies and 80% of preterm infants during their first week of life.[2,3,4]

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A few of them develop serious health conditions. Jaundice occurs when unconjugated bilirubin accumulates in the skin and mucous membranes.[3] If the bilirubin level rises to dangerous levels, treatments and close observation are required to prevent the complications and fatal consequences.[5] Treatment of neonates with jaundice has been investigated using a number of different approaches. Phototherapy is the commonly used technique.[6] Itis the most frequently utilized treatment because it is safe, inexpensive, non-invasive, and widely accessible.[7] Phototherapy has various adverse effects. Dehydration is the most frequent of them. When phototherapy fails

to reduce total serum bilirubin (TSB) levels gradually, exchange transfusion is necessary. But, this may raise the risk of consequences such as sepsis, emboli, anemia, apnea, hypocalcemia, and transfusion reactions.[8] Therefore, the use of intravenous fluid (IV fluid) along with phototherapy to accelerate the treatment is recommended by some studies.[8-10] The researchers suggested that, IV fluids support the maintenance of ideal hydration levels. It increases blood flow and the liver's capacity to metabolize and eliminate bilirubin, therefore aiding in a shorter hospital stay.[8-10]

Lumirubin is an isomer of bilirubin, a water-soluble product that is produced during phototherapy. Lumirubin is excreted through bile and urine. Adding intravenous fluid corrects dehydration and also improves urine output and enhances the body's ability to eliminate lumirubin.[7] But there is rising concern regarding the use of IV fluid along with phototherapy. A study by Goyal et al., 2018 found the mean rate of decrease in serum total bilirubin (STB) levels did not decline significantly when IV fluid was given to the neonates. They stated that, IV fluid supplementation is an invasive procedure and causes several other complications. Dehydration of the neonates can be corrected by frequent breast feeding.[11]

Regarding the use of IV fluids, there is some disagreement among doctors. Therefore, the goal of the current study was to determine the necessity of intravenous fluid therapy in infants receiving phototherapy for non-hemolytic hyperbilirubinemia.

Methods:

This randomized controlled trial was conducted at the Department of Pediatrics, Sylhet Women's Medical College Hospital, Sylhet. This study was commenced after approval by Ethical Review Committee of Sylhet Women's Medical College and Hospital, and the duration of the study was 1 year after approval of the protocol. The targeted population of the study were neonates with hyperbilirubinemia who had bilirubin level at the phototherapy threshold according to the bilirubin chart. The investigation encompassed a total of 300 neonates.

Simple random sampling method was adopted for the selection of the subjects. All the patients were enrolled from department of Pediatrics, Sylhet Women's Medical College Hospital, Sylhet. Premature infants with gestational age of less than 37 weeks, presence of any congenital malformations, presence of any systemic disease, sepsis, evidence of hemolysis, patients who required blood transfusion at the beginning of hospitalization, infants received intravenous (IV) fluids previously for any reason and neonates with signs of bilirubin encephalopathy (kernicterus) were excluded.

After selection, the objectives, nature, purpose, benefits of the study and likely risks of all the procedures was explained to each guardian of neonates. After enrollment, informed written consent was obtained from the guardians. The patient's level of dehydration was evaluated by a pediatrician (researcher) at the bedside. Total, direct and indirect serum bilirubin level were measured initially. Two envelopes, one labeled with phototherapy and another labeled with phototherapy with intravenous fluid was given to each guardian to pick up. Based on the envelope they selected, the neonates were allocated to either Group A or Group B. 134 neonates were enrolled in group A, and 166 neonates were enrolled in group B. Group A neonates received intravenous fluid supplementation along with phototherapy and group B received only phototherapy during the study. The extra fluids group (Group A) were given IV fluid supplementation with Quarter strength normal saline (0.225%) in 10% dextrose for a period of 24 hours via a peripheral vein during phototherapy. The volume of the fluid supplement was 50 mL/kg presumed deficit (corresponding to mild dehydration), half of the daily maintenance requirements for a period of 24 hours. Group B patients received only phototherapy. Both group of neonates were also permitted to breastfeed. The hydration status was evaluated and I/V fluid was stopped at the end of 24-hours period in neonates of Group-A.

Each phototherapy device were included four unique blue lamps that were positioned 20 cm above the babies' cribs. Serum bilirubin levels, both total and direct, were assessed initially and

then every 24 hours. The total serum bilirubin (TSB) were monitored and phototherapy was continued until it dropped to less than 14 mg/dl. Kramer scale was be used to determine the clinical outcome of the jaundice. Heart rate, respiratory rate, capillary filling time, anterior fontanelle, skin turgor, oral mucosa, weight, and urine output all were monitored to look for clinical indicators of dehydration. These were noted at the beginning of the study, then every 8 hours for the next 24-72 hours. Data were recorded in a predesignated checklist and analyzed by SPSS version 26.

Results:

The mean age of group A was 7.03±2.177 days and group B was 7.07±035 days. The mean age difference of supplement group and phototherapy group showed no significant differences (p=0.721) (Table 1). Majority of both groups belonged to rural area (p>0.05) (Table 2). There were no significant difference observed in terms of gestational age, prenatal checkups, place of delivery and birth attendants (p>0.05) (Table 3).

The mean weight and length of both groups of neonates were similar (p>0.05) (Table 4).

The onset of jaundice among neonates also showed no significant difference (p>0.05) (Table 5).

Results showed no significant difference (p>0.05) in the serum total, direct and indirect bilirubin levels of neonates of both groups (Table 6). Total serum bilirubin levels were similar in both the study groups when they were discharged (p>0.05) (Figure 1). The clinical outcome of the patients were similar in both groups (p>0.05) (Table 7). The time of discharge of the neonates were also similar in both groups (p>0.05) (Figure 2).

Table 1: Mean age of the study subjects (N=300).

Variable	Group A	Group B	p value
Age (Days)	7.03±2.177	7.07±035	0.721
Range	3-13	4-13	

Statistical analysis was done by unpaired 't' test; values in the parenthesis indicate range; N= total number of the subjects.

Table 2: Demographic variables of the patients (N=300)

Variables		Group A (n=134) Frequency (%)	Group B (n=166) (Frequency %)	*p* value
Residence	Rural	76 (56.72)	106 (63.85)	0.417
	Urban	58 (43.23)	60 (36.14)	
Socioeconomic status	Lower	40 (29.85)	46 (27.71)	0.108
	Middle	94 (70.15)	120 (72.29)	

Statistical analysis was done by chi-square (χ^2) test.; values in the parenthesis indicate percentages; n=number of the subject in each group; N= total number of the subjects.

Table 3: Obstetrical history of the patients (N=300)

Variables		Group A(n=134) (Frequency %)	Group B(n=166) (Frequency %)	*p* value
Gestational age	38	56 (41.79)	68 (40.96)	
	39	70 (52.23)	90 (54.22)	0.889
	40	08 (5.97)	08 (4.82)	
Antenatal checkup	Yes	34 (25.37)	56 (33.73)	0.129
	No	100 (7.46)	110 (66.27)	
Mode of delivery	VD	74 (55.22)	86 (51.80)	0.568
	LUCS	60 (44.78)	80 (84.19)	
Place of delivery	Home	30 (22.39)	30 (18.07)	
	SWMC	50 (37.31)	82 (49.39)	0.111
	Other hospital	54 (40.29)	54 (32.53)	
Delivered by	Doctors	104 (77.61)	134 (80.72)	
	Nurse	08 (5.97)	02 (1.02)	0.150
	Dai	14 (10.45)	20 (12.04)	
	Relatives	08 (5.94)	10 (6.02)	

Statistical analysis was done by chi-square (χ^2) test.; values in the parenthesis indicate percentages; n=number of the subject in each group; N= total number of the subjects.

Table 4: Mean weight and length of the patients (N=300)

	Group A (n=134) (Mean ± SD)	Group B (n=166) (Mean ± SD)	*p* value
Weight (Kg)	2.99±0.28 (2.50-3.70)	2.93±0.27 (2.50-3.70)	0.911
Length (cm)	49.89±0.95 (47-52)	49.84±2.57 (30-60)	0.824

Statistical analysis was done by unpaired 't' test; values in the parenthesis indicate range; n=number of the subject in each group; N= total number of the subjects.

Table 5: Day of onset of jaundice among study patients (N=300)

	Group A (n=134) Frequency (%)	Group B (n=166) (Mean ± SD)	*p* value
Day 2	41(30.59)	61 (27.75)	0.509
Day 3	85(63.43)	97(58.43)	
Day 4	06(4.48)	10 (6.02)	

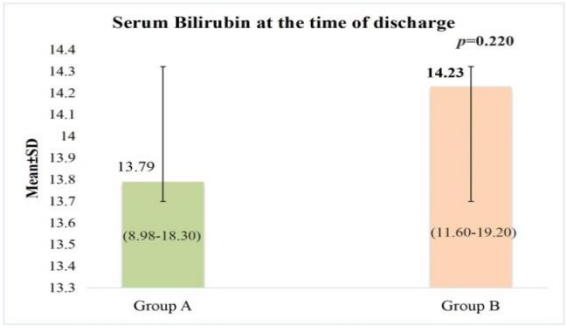
Statistical analysis was done by chi-square (χ^2) test.; values in the parenthesis indicate percentages; n=number of the subject in each group; N= total number of the subjects.

Table 6: Bilirubin levels of the patients at the time of admission (N=300)

Variables	Group A (n=134) (Mean ± SD)	Group B (n=166) (Mean ± SD)	*p* value
S. total bilirubin	18.79±2.06 (15-25.40)	18.32±2.15 (15-27.50)	0.51
S. direct bilirubin	0.43±0.14 (0.20-0.80)	0.68±1.88 (0.20-0.82)	0.074
S. indirect bilirubin	18.43±1.95 (15.00-25.00)	17.63±2.81 (12.52-27.20)	0.566

Statistical analysis was done by unpaired 't' test; values in the parenthesis indicate range; n=number of the subject in each group; N= total number of the subjects.

Figure 1: Serum bilirubin at the time of discharge of the patients (N=300)



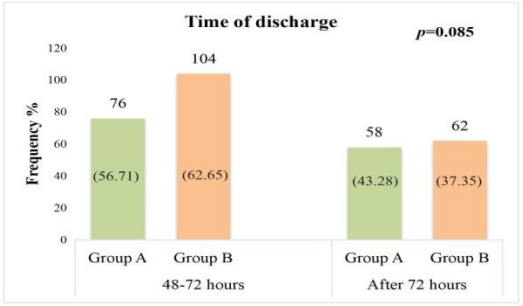
Statistical analysis was done by unpaired 't' test; values in the parenthesis indicate range;
N= total number of the subjects.

Table 7: Clinical outcome of the patients (N=300)

Clinical outcome	Group A (n=134) (Frequency %)	Group B (n=166) (Frequency %)	*p* value improvement
Within 24-48 hours	20(14.92)	29 (17.47)	
Within 48-72 hours	50(37.31)	70 (42.17)	0.417
After 72 hours	64(47.67)	67 (40.36)	

Statistical analysis was done by chi-square (χ^2) test.; values in the parenthesis indicate percentages; n=number of the subject in each group; N= total number of the subjects.

Figure 2: Time of discharge after treatment (N=300



Statistical analysis was done by chi-square (χ^2) test.; values in the parenthesis indicate percentages; N= total number of the subjects.

Discussion:

Jaundice is the most common clinical problem among neonates.[9] Phototherapy is the most common approach for the treatment of neonatal hyperbilirubinemia.[12] Fluid treatment is regarded as an effective technique to reduce bilirubin level.[12] The aim of this study was to ascertain the need of intravenous fluid therapy among infants undergoing phototherapy.

The mean age, accommodation types, socioeconomic status, gestational age, prenatal checkups, birth methods, place of delivery, and attendants were comparable in both groups ($p>0.05$) (Table: 1,2,3). Similar results were observed in a study conducted by Eghbalian et al. in 2022.[9] The two groups in their study were comparable regarding number, sex, age, gestational age, birth weight, weight at admission, and mode of delivery. Similar findings were also observed by Enad, Imran and Hussein, 2018.[13] The mean weight and length showed no difference between the groups. The weight of newborns upon admission was not significantly different between the intravenous supplement group and the phototherapy group, as noted by Morshed et al., 2017.[14] Most of the neonates developed jaundice at the third day of their life (Table: 5). Although the result was not significant. This finding was well supported by Hansen, 2021.[15] He stated that TSB eventually manifests with detectable bilirubin, typically on the second–third day of life.

The serum total, direct and indirect bilirubin levels of the newborns showed similar variation ($p>0.05$) (Table: 6). At the time of discharge, total serum bilirubin level were lower in IV supplement group neonates (Figure: 1). Although, the result was nonsignificant in both groups ($p>0.05$). This study was consistent with other study.[5,10] Eghbalian et al., 2022 observed that, the mean levels of serum bilirubin on admission, 6, 12, 24, and 48 hours after treatment decreased progressively in both groups; however, the difference was not statistically significant between the two groups.[5] In a study in Iran, the mean rate of decrease in TSB levels during the first 12 hours of phototherapy was not statistically significant in either the supplemented or non-supplemented groups ($p=0.13$).The duration of phototherapy required in the supplemented and non supplemented groups was also comparable ($p=0.13$).[10] Different outcomes were observed in certain investigations.[13,14] Total blood bilirubin (TSB) levels before treatment were found to be comparable in both groups in a study conducted by Enad, Imran, and Hussein, 2018.[13] But, after 24 hours of IV fluid supplementation, TSB level was significantly reduced in neonates who received IV fluid along with phototherapy in their study. The rate of bilirubin reduction was also statistically significant in the study group. Morshed et al., 2017 observed that, at 48 hours, the serum total bilirubin levels were significantly lowered in the IV supplement group ($p<0.001$).[14]

The clinical outcomes of the majority of newborns improved after 72 hours. Even yet, the findings were insignificant ($p>0.05$). Kaur et al., 2018 observed that the administration of additional intravenous fluids during the initial eight hours can substantially reduce TSB levels in severe non-hemolytic neonatal hyperbilirubinemia.[16] A study conducted by Eghbalian et al. in 2022 revealed similar findings with our study. The mean serum bilirubin level in their study groups showed no significant difference after 24 hours of phototherapy and phototherapy with IV supplementation in neonates.[9]

Although, the discharge time for neonates in the IV supplement group earlier, but the result was non-significant. In the intravenous supplement group, 56.71% were discharged between 48 to 72 hours, and 43.28% were discharged after 72 hours. Conversely, neonates who underwent only phototherapy had 62.657% discharged within 48 to 72 hours, while 37.35% were discharged beyond 72 hours. Comparable results were noted by Eghbalian et al., 2022. They found that, the duration of hospitalization was reduced in the fluid supplementation group; however, the results were also not significant in their study.[9]

Maintaining hydration with IV fluids is important for treating newborn jaundice because dehydration can make it challenging for the

body to get rid of bilirubin. Study observed that, IV supplementation keeps the patient hydrated and may speed up the drop in bilirubin levels, which makes neonates easier to eliminate bilirubin and reduce hospital duration. When neonates with mild to severe jaundice get phototherapy with IV fluid supplementation and drink breast milk, these may reduce the duration of hospital stays.[17,18]Butintra-venous fluid supplementation during phototherapy in newborns with hyper bilirubinemia did not have a meaningful effect in the present study.

Conclusion:

The current study suggests that the serum bilirubin level reduction, hospital duration, and clinical outcome during conventional phototherapy are not affected by the administration of additional intravenous fluid to healthy, term neonates with jaundice. So whenever phototherapy is sufficient to lower the serum bilirubin, we should avoid intravenous fluid therapy and promote frequent breast feeding to maintain hydration. Further study is suggested to assess the impact of intravenous supplementation in neonatal Hyperbilirubinemia with a larger sample size.

Ethical clearance: Taken from institutional ethics committee.

Conflict of interest: Nil.

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Review Article

Mpox Outbreak 2024: Epidemiological Shifts, Challenges, and Future Directions

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Abstract:

Mpox was once a zoonotic viral disease confined to a few countries in Central and West Africa. Its primary reservoir animals included rodents and nonhuman primates inhabiting the tropical rainforests of the region. The disease began to flourish in humans primarily after the 1980s, when smallpox vaccination was discontinued. This vaccine provided 85% cross-protection against mpox, limiting its spread in earlier decades. Over time, the mpox virus has undergone mutational evolution, enhancing its adaptability to human hosts and establishing itself as a globally infectious human virus. Following the challenges posed by the COVID-19 pandemic, mpox now looms as a potential new global health threat. It is crucial to raise awareness and implement rigorous precautionary measures to prevent its further spread. This requires a thorough understanding of the virology, evolution, and current disease patterns of mpox.

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Introduction:

Mpox has been classified as a neglected tropical disease since its first human case was detected in the Democratic Republic of Congo (DRC) in 1970.[1,2] Over time, this once-local zoonotic endemic disease has evolved into a global infectious threat due to a lack of adequate attention and prevention strategies. Notable outbreaks include the large 2017 outbreak in Nigeria, marking the virus's re-emergence in the country after 39 years; the 2022 international outbreak; and the 2023 major outbreak in the DRC, which exhibited atypical disease patterns in several aspects.[3,4,5] According to World Health Organization (WHO) data, from January 2022 to June 2024, a total of 99,176 laboratory-confirmed cases and 208 deaths were reported across 116 countries spanning all six WHO regions.

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According to World Health Organization (WHO) data, from January 2022 to June 2024, a total of 99,176 laboratory-confirmed cases and 208 deaths were reported across 116 countries spanning all six WHO regions. The highest number of cases were recorded in the African region, followed by the Americas and Europe.[6]

To date, Bangladesh has not reported any confirmed mpox cases. However, its neighboring country, India, reported 23 confirmed cases, including one death, during the 2022 outbreak, and one confirmed case in the 2023 outbreak. These developments signal a potential risk for Bangladesh.[7,8]

It is imperative to act now, fully acknowledge the emerging challenges, and implement appropriate preventive measures to safeguard the future. Without proactive efforts, we risk facing another catastrophic pandemic while still recovering from the traumatic experiences of COVID-19. A comprehensive understanding of

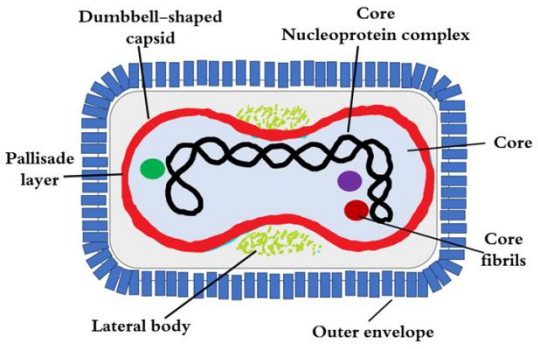
the virology, evolution, and emerging strains of the mpox virus, as well as its changing epidemiological patterns, is essential to formulate effective strategies. This review article will provide an overview of these critical points.

The Mpox virus:

Mpox is caused by the mpox virus, first isolated in Denmark in 1958 from a captive monkey colony infected with a pox-like disease. As a result, it was initially named 'monkeypox'.[2] However, subsequent research has suggested that rodents are likely the virus's primary reservoir. To combat the racist and stigmatizing attitudes that arose during the 2022–2023 outbreak, the World Health Organization (WHO) officially renamed the disease 'mpox' in November 2022.[9]

The mpox virus belongs to the *Orthopoxvirus* genus, part of the *Poxviridae* family and *Chordopoxvirinae* subfamily. Other members of this genus include the variola virus, vaccinia virus, camelpox virus, and cowpox virus. All are human pathogens, but mpox and variola viruses are the most significant due to their high virulence. These two viruses share 96.3% genomic similarity,[4] which is why the vaccinia virus vaccine, originally developed for smallpox (variola virus), provides 85% cross-protection against mpox.[10]

Figure 1: Different parts of the mpox virus *(courtesy: Dr. Tamanna Afroze)*



Among human pathogenic viruses, poxviruses are the largest, enabling them to be observed with a light microscope. Structurally, they are also the most complex. Under an electron microscope, the mpox virus (Figure 1) appears as an enveloped, double-stranded DNA (dsDNA) particle measuring 200–250 nm in diameter. The virus envelope consists of two layers, the outer and the inner. Numerous tubular-shaped proteins are attached to the outer layer, interacting with glycosaminoglycans (GAGs) and other host cell proteins. The nucleocapsid is dumbbell-shaped or resembles a box with concavities on the upper and lower surfaces. Each concavity contains an amorphous protein complex known as the lateral body. While their exact function remains unknown, lateral bodies are believed to play a role in immune modulation. The genome of the mpox virus is a large ds DNA molecule approximately 70 kbp in length, with terminally attached strands. Unlike other DNA viruses, mpox replicates in the cytoplasm of the host cell, showcasing a unique feature of poxviruses.[8,11]

Clades of the virus:

Before 2022, mpox viruses were classified into two distinct clades: the Central African clade, or clade I, typically found in the Congo Basin of Central Africa, and the West African clade, or clade II, primarily found in West Africa. Clade I is more virulent, causing severe clinical symptoms and a mortality rate of up to 10%. In contrast, clade II is less virulent, associated with milder symptoms and a mortality rate of less than 1%.[12,13]

Historically, both clades were primarily transmitted through zoonotic pathways, with limited human-to-human transmission. The maximum sequential human transmission chains were limited to six, with household transmission rates of approximately 10%. Consequently, previous outbreaks were confined to the rainforests of Central and West Africa, where reservoir hosts such as rodents and non-human primates had contact with impoverished local populations. Outbreaks outside Africa were rare, small, and localized, typically linked to travel to endemic areas or the importation of infected animals.[14]

The 2017 outbreak in Nigeria marked a significant shift, leading to the emergence of a mutated subclade of clade II capable of sustained human-to-human transmission. The older strain was then designated as clade IIa and the new one was named clade IIb. The B.1 lineage of the clade IIb was responsible for the

2022 international outbreak. Similarly, during the September 2023 outbreak in the Democratic Republic of Congo (DRC), genomic analysis identified a new subclade of clade I, named clade Ib, which demonstrated higher transmissibility among humans compared to the older mother clade Ia. Despite the enhanced transmissibility of these mutated strains, their virulence was significantly lower than that of their parent clades. As a result, while both the 2022 and 2023 outbreaks affected a large number of people, hospitalization and death rates remained low (Table 1).[15,16]

Table 1: Global outbreaks due to different clades of mpox

	Clade Ia	Clade Ib	Clade IIa	Clade IIb
Origin	Central Africa	2023 DRC outbreak	West Africa	2017 Nigeria outbreak
Distribution	Congo basin of central Africa	Africa specially DRC	West Africa	Global
Mode of transmission	Mainly zoonotic	human to human mainly through sexual contact	Mainly zoonotic	Human to human mainly through sexual contact
Affected population	Children	Adult (middle aged men)	Children	Adult (middle aged men.
Symptoms	Severe than IIa	More severe than Ia	Less severe than Ia	Less severe than IIa
Mortality rate	10%	5%	1%	0.2%

Epidemiological shift:

After the first isolation of virus, from 1958 to 1968, nine outbreaks occurred among non-human primates kept in laboratories and zoos, but no research person or zoo keeper was infected during handling the sick animals. This indicates, at that time the virus was incapable of infecting humans. After the 1st human case detection in the DRC in 1970, mpox has been confined for many years to the tropical rainforest regions of Central and West Africa with endemic and sporadic outbreaks.[14,2] As of 2003, cases have been reported from 11 African countries namely Benin, Cameroon, Central African Republic, Congo, Côte d'Ivoire, DRC, Gabon, Liberia, Nigeria, Sierra Leone and South Sudan.[13] The most affected country is the DRC with less than 500 cases in 2001 but more than 2500 cases in 2018.[17,18] Until 2021, all the outbreaks outside Africa have been linked to contact with infected animals or travel to endemic countries in Africa (Table 2).[19,20,21]

Table 2: Global epidemiological shift through the century

Year	Affected country	Number of cases	Source
2003	US	47	Imported infected rodents from Ghana.
2018	UK	3	travel to Nigeria
2018	Israel	1	travel to Nigeria
2019	Singapore	1	travel to Nigeria
2021	US	2	travel to Nigeria
2021	UK	3	travel to Nigeria

Here should be noted that Nigeria was facing the biggest mpox outbreak in its history since 2017. Till 2021, 226 laboratory-confirmed cases and eight deaths were reported from here.[22]

In 2022, the first large international outbreak of mpox occurred, marking a significant departure from all previous non-endemic outbreaks. Most cases were reported from non-endemic countries with no direct links to endemic regions or signature cases. The initial case in the UK, reported on May 6, 2022, involved an individual with recent travel history to Nigeria.[23] However, shortly thereafter, thousands of cases were identified across a wide range of non-endemic regions, particularly in Europe and the Americas.[22,24] Less than 1% of cases originated from endemic zones.[15,25]

Genomic analysis later confirmed that the mpox virus had undergone multiple genetic mutations, enabling better adaptation and transmission between human hosts. This led to the emergence of a new genetic variant of the West African clade (clade IIb). The outbreak also exhibited several unprecedented characteristics, including the demographics of affected populations,

modes of transmission, and clinical presentations.[23,26] A study by Ilic et al, analyzing data from 43 locations across 16 countries, revealed that 98% of the affected individuals were male, with a median age of 38 years. Among these, 99% identified as gay or bisexual men.[27] Although mpox virus DNA was detected in semen in some cases, the evidence is insufficient to classify it as a sexually transmitted virus. It is believed that the virus entered the MSM (men who have sex with men) group coincidentally through close physical contact.[24] The clinical features of this outbreak included atypical presentations, such as the absence or mild nature of prodromal symptoms, rashes preceding prodromes, or rashes manifesting as isolated ulcers or lesions, often confined to the anogenital and/or perineal areas. Predominantly, inguinal lymphadenopathy was observed. The severity of the disease was generally mild to moderate, with hospitalization rates ranging between 4–10%.[28]

Due to the rapid rise in cases, unusual transmission patterns, and atypical symptoms in non-endemic regions, the World Health Organization (WHO) declared the outbreak a Public Health Emergency of International Concern (PHEIC) on July 23, 2022.[29] Thanks to efficient surveillance and control measures by local and international health authorities, the number of cases began to decline in January 2023. In May 2023, WHO announced the end of the emergency declaration. As of October 2024, sporadic low-level infections with the new strain continue in various regions. A total of 110,570 confirmed cases and 237 deaths have been reported across 123 countries.[8]

2024 outbreak in DRC:

In September 2023, mpox cases began to rise again in the Democratic Republic of Congo (DRC). Genomic analysis of samples revealed that this outbreak originated from the Central African clade. While clade Ia was already endemic in the DRC, the human-to-human transmissibility of clade Ib in this outbreak was significantly higher than in previous instances.[12] The initial cases were reported from the Kamituga Health Zone in South Kivu Province, located in eastern DRC. This is a densely populated mining region with numerous bars forming part of the local sex industry. Demographic analysis of PCR-confirmed cases from September 2023 to May 2024 showed that over 50% of cases were female, with a median age of 22 years. Approximately 30% of these cases involved individuals engaged in sex work, suggesting transmission primarily through sexual contact or close physical interaction.[30]

Subsequently, adult-to-child and child-to-child transmission became apparent, leading to an increase in cases among children. Pregnant women were also affected, with reports of miscarriages linked to the outbreak. Cases began to emerge from various provinces across the DRC, including regions previously unaffected, as the outbreak expanded.[8,16,17]

According to the African CDC, the number of mpox cases in the DRC increased by 160% in 2024 compared to 2023. Children under the age of 15 accounted for 70% of reported cases and 85% of deaths. The outbreak also spread to neighboring countries, including Burundi, Rwanda, Uganda, and Kenya, marking them as newly affected African nations. Additionally, cases were reported in two new countries outside Africa, Sweden and Thailand.[24] As of August 24 over 21000 cases have been reported from 15 countries and over 96% reported from the DRC. On 14 August 2024, WHO again declared the epidemic as a PHEIC.[13,31]

Challenges with mpox outbreak trends

The mpox virus is undergoing a continuous evolutionary process, resulting in significant changes from its emergence to the present day. This evolution has been explored in various research studies. Deforestation and urbanization have contributed to the decline in reservoir animal populations, disrupting the micro-environments suitable for the virus's development. Consequently, mutations have facilitated increased animal-to-human spillover, adaptation to human hosts, and human-to-human transmission, including through novel routes such as sexual contact.[2,32]

Theoretically, as a DNA virus with the largest and most complex DNA genome among viruses, mpox mutations should occur at a slow rate. However, the 2022 mpox strain (Clade IIb) exhibited an unexpected 6-12-fold higher mutation rate than anticipated, with 46 single

nucleotide polymorphisms (SNPs) identified compared to the NCBI reference sequence NC_063383. This international outbreak was exclusively driven by human-to-human transmission. Similarly, the 2023 Clade Ib strain emerged from mutational variations and is still under genomic analysis. Clade Ib demonstrates higher transmissibility in humans compared to Clade Ia and has expanded into previously unaffected neighboring countries of the DRC, showcasing its pandemic potential.[32,33]

Research has identified the host cell enzyme APOBEC3 as a key factor behind the high mutation rate in both clades, with approximately 20 APOBEC3-driven mutations occurring annually due to human-to-human transmission. Notably, 40% of individuals infected during the 2022 outbreak were HIV-positive, a condition that significantly increases APOBEC3 expression.[16,18,24] This suggests that HIV-positive individuals may play a direct role in mpox mutations. Continued mutation of the virus raises the possibility of the emergence of a novel mpox strain that existing cross-protective vaccines may not counter, necessitating the development of a specific mpox vaccine. Currently, no mpox-specific vaccines or antiviral drugs are available.[4,7,14] Furthermore, if the virus establishes new animal reservoirs in non-endemic areas, it could create new endemic zones, escalating the risk of a future pandemic.[15]

Future directions:

Preventing human-to-human transmission is critical to averting a serious pandemic. Each country must develop a robust strategy through early planning, effective public relations, and multinational cooperation.

Key recommendations include:

1. *Surveillance and Monitoring:* Governments and healthcare authorities should establish strong surveillance systems to prevent mpox from entering through international routes and ensure immediate detection of new cases. This includes training personnel to recognize the early symptoms of mpox.[5,6]
2. *Training on Diagnostics:* Health technicians and relevant personnel should be trained in CDC-recommended guidelines for specimen collection and biosafety for mpox diagnosis. Accurate RT-PCR results, the latest diagnostic method, depend on proper specimen collection, storage, transportation, and testing. Adequate stocks of PCR kits should be maintained for prompt diagnosis.[7,9,26]
3. *Treatment and Management:* Updated treatment and management guidelines should be developed. Sufficient vaccine and antiviral drug supplies should be ensured, prioritizing non-endemic but high-risk countries like Bangladesh, India, Pakistan, and Afghanistan, in addition to endemic regions. Densely populated countries in these regions would face significant challenges in containing outbreaks.[10,24,34]
4. *Vaccine Prioritization and Production:* Countries should create vaccine priority plans to rapidly cover vulnerable populations. Potential countries should consider producing vaccines and drugs domestically. International organizations must accelerate efforts to develop specific mpox vaccines and antiviral drugs.

Collaborative global efforts and proactive measures are essential to mitigate the threat posed by the evolving mpox virus.

Conclusion:

Epidemiological analysis shows a gradual decline in mpox cases. However, in some areas or communities, viral transmission and mutations persist at low, often undetectable levels, increasing the risk of a new strain emerging and potentially triggering another outbreak. While the World Health Organization has already issued public health alerts, heightened vigilance remains essential. Bangladesh must implement meaningful and proactive measures to protect its population and contribute to global health security. Through widespread public awareness, prompt action, and strong national and international collaboration, this emerging global health threat can be effectively managed.

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Case Report

Autoimmune Hepatitis in Children: A Case Report and Literature Review

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Abstract:

Autoimmune hepatitis (AIH), is an relatively uncommon cause of liver disease, especially in children. It has wide range of presentation like asymptomatic rise of liver enzyme to advance stages of liver disease. When other causes of liver disease ruled out, AIH should always to taken into account. We presenting the case of 9-year-old girl, who had manifestations of liver disease and we diagnosed her as a case of AIH.

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JSWMC 2025 [15(01)] P: 51-54

Introduction:

Autoimmune hepatitis (AIH), a chronic inflammatory condition with unknown etiology, The hallmarks of the disease include hypergammaglobulinemia and/or circulating auto-antibodies, profound changes in hepatic histology, and a marked response to immunosuppressive treatment[1]. Women are affected more frequently than men, 75% of patients of AIH are female, and the disease is seen in all ethnic groups and at all ages.[2] In Bangladesh, Benzamin et al. found that, about 8% of children with chronic liver disease with hepatomegaly and or splenomegaly are due to AIH.[3]

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In accordance with serological profile, AIH are two types; AIH-1 is defined as positive for antinuclear antibody (ANA) or anti-smooth muscle antibody (SMA) or both; AIH-2 is defined as positivite for anti-liver kidney microsomal type 1 antibody (anti-LKM-1) or for anti-liver cytosol type 1 antibody (anti-LC-1) or both.[4]

Case Report:

A 9-year old immunized girl, 2nd issue of non-consanguineous parents, got admitted at Pediatrics Department, Sylhet M AG Osmani Medical College Hospital, with complaints of jaundice for 3 months, abdominal distension for same duration and nausea with anorexia for 6 months. She had no history of gastrointestinal bleeding, abdominal pain, altered sleep pattern, rash, arthralgia, blood transfusion, any surgical procedure, family history of such type of illness, and any offending drug exposure. On examination she had jaundice and splenomegaly. Her vital signs were within normal limit and anthropometrically she was well thriving. Investigations showed raised serum bilirubin, serum ALT and thrombocytopenia. Ultrasonography showed coarse hepatic parenchyma with splenomegaly. Investigations for hepatitis B virus, hepatitis C virus and

Wilson's disease were negative. ANA was positive, anti LKM1 was negative. Serum TSH was elevated but serum T3 and T4 were normal. Endoscopy of upper gastrointestinal tract showed Grade II esophageal varices (fig1) and histopathology of liver tissue compatible with autoimmune hepatitis with cirrhosis. So finally we diagnosed her as a case of chronic liver disease (CLD) with autoimmune hepatitis with portal hypertension with subclinical hypothyroidism. We treated her with Tab. Prednisolone 2mg/kg/day and Tab. Azathioprine 1-2mg/kg/day were added 2 weeks later of prednisolone started. Tab. Propranolol 1mg/kg/day was given as primary prophylaxis of portal hypertension and Tab. Levothyroxine 1.5 mcg/kg/day was started for subclinical hypothyroidism.

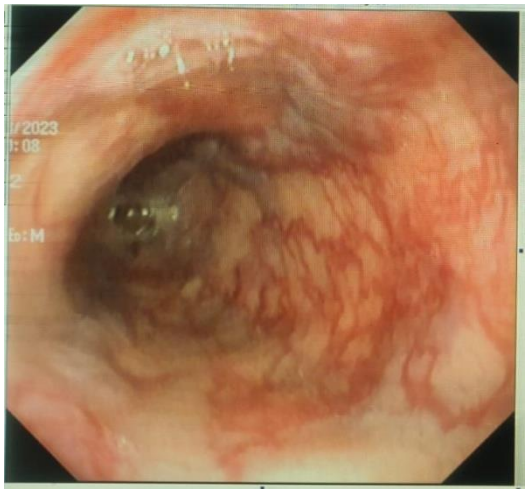

Fig 1: Oesophageal varices

Discussion:

AIH has a variety of clinical presentations. Range of presentations includes non-specific symptoms like progressive fatigue, malaise, nausea, anorexia, vomiting, joint pain, amenorrhea, headache, abdominal pain to acute hepatitis, relapsing jaundice or fulminant hepatic failure or chronic liver disease, complication of cirrhosis and portal hypertension without history of previous liver disease and jaundice. Non specific symptoms may last for 6 months to few years before diagnosis. Incidental finding of raised ALT without sign symptoms is another presentation of AIH.[4]Our case presented with non specific symptoms such as anorexia, nausea for 6 months followed by jaundice for 3 months. With these symptoms she visited to several physicians and evaluated for common liver diseases, but remained undiagnosed. Suspicion of autoimmune hepatitis always to be considered after excluding all causes of acute and chronic hepatitis. The diagnosis of AIH requires compatible histological findings and is further supported by the following features: (1) elevated serum aminotransaminase levels (ALT/AST); (2) elevated serum IgG level and/or positive serological marker(s); (3) exclusion of viral, hereditary, metabolic, cholestatic, and drug-induced diseases that may resemble AIH. Initial serological testing should include ANA, SMA, anti-LC-1 and anti-LKM1 in children; consider additional autoantibody tests if warranted to secure the diagnosis.[5]In our case, we found elevated serum ALT, ANA positive, histology suggestive of AIH and we excluded viral causes and Wilson disease. She also had portal hypertension, as evidenced by oesophageal varices. Histopathology of liver tissue is one of the key diagnostic tools for diagnosis of AIH. Regardless of serologic type, the classic histological findings on liver biopsy are similar. Biopsy shows portal and lobular inflammation, composed of lymphocytes and, most commonly, conspicuous plasma cells. The portal inflammation typically creates "interface hepatitis" where the limiting plate is disrupted and the inflammation involves the adjacent liver parenchyma; concomitant hepatocyte necrosis may or may not be present. Lobular inflammation can range from mild to severe, with either scattered necrotic hepatocytes, or in more severe cases centrilobular and/or bridging necrosis. Rosette formation and emperipolesis are two uncommon characteristics.[6]In our case, histopathology of liver tissue was consistent with autoimmune hepatitis. In both types, a family history of autoimmune disease is frequent (40%) and approximately 20% of patients have associated auto immune disorders either present at diagnosis or developing during follow-up, including thyroiditis, inflammatory bowel disease (IBD), hemolytic anemia, vitiligo, celiac disease, insulin-dependent diabetes, Behchet disease, Sjo¨gren syndrome, glomerulonephritis, idiopathic thrombocytopenia, urticaria

pigmentosa, hypoparathyroidism, and addison disease (mainly in AIH-2).[4]Our case have no family history of autoimmune disease but she had hypothyroidism. But autoimmune thyroiditis was not rule out. After diagnosis, prompt treatment should be started. AIH is responsive to immunosuppressive drug. Treatment consists of two parts- induction of remission and maintenance phase. With the exception of a fulminant presentation with encephalopathy, AIH responds satisfactorily (remission rate of up to 90%) to immunosuppressive treatment whatever the degree of liver impairment. Conventional treatment of AIH consists of prednisolone 2mg/ kg/day (maximum 60mg/day), which is gradually decreased during a period of 4 to 8 weeks, in parallel to the decline of transaminase levels, to a maintenance dose of 2.5 to 5mg/day. In most patients, an 80% decrease of the aminotransferase levels is achieved in the first 2 months, but their complete normalization may take several months.[4]In our case, Tab. Prednisolone 2mg/kg/day and Tab. Azathioprine 1-2mg/kg/day was added 2 weeks after starting prednisolone. On follow up after 2 month, her bilirubin was 3.6 mg/dl and ALT was 103 U/L. That's indicating partial improvement.

Investigations	Findings	Normal
CBC (02/08/2023)	Hb 13.1 gm/dl MCV 82.9 fL MCH 27.7 pg WBC 4800 per cumm. Platelet 100000 per cumm.	Male- 13-17 gm/dl , Female 12-15 gm/dl 83-101 fL 27-32 pg 4000-11000/cumm. 150000-400000/cumm
PBF	No evidence of haemolysis, Thrombocytopenia	
SGPT	154U/L	Female <35U/L
S.Bilirubin	4 mg/dl	Female: mg/dl
Prothrombin time with INR	Patient 14 Sec. Control 12 Sec. Index 85.7% Ratio 1.16:1 INR 1.20	
S.Albumin	1.9 mg/dl	3.5-5 mg/dl
USG of HBS	Coarse hepatic parenchyma with splenomegaly	
HbsAg	Negative	
Anti HCV	Negative	
S.Ceruloplasmin	319 mg/L	200-600 mg/L
Eye evaluation for KF ring	No K-F ring or Sun-flower cataract	
S. IgG	Not done	
ANA	96.1 AU/ml	>40 AU/ml Positive
Anti LKM1 antibody	Negetive	
Anti Ds DNA	15.20 IU/ml	> 25 IU/ml Positive
S.TSH	10.4 mIU/L	Children (2-12 yrs):0.72-4.4 mIU/L
FT4	1.82 ng/dl	0.78-2.19 ng/dl
Endoscopy of UGIT	Oesophageal varices	

Histopathology of Liver Tissue	Hepatocytes had mild feathery degeneration, the portal tract shows evidence of bile duct injury, infiltration of lymphocytes, plasma cells, some polymorphs and marked fibrosis including bridging fibrosis, the fibrous bands have encircled the hepatic plates forming cirrhotic nodules, mild piecemeal necrosis, interface hepatitis and emperipoiesis of hepatocytes by lymphocytes are seen, no dysplasia or malignancy are seen.	

Conclusion:

AIH is relatively a rare disease; it remains a diagnostic& therapeutic challenge, especially among the children and adolescents. All unexplained liver disease should screen for AIH, which will make early diagnosis and will ensure good outcome.

Ethical issue: Inform written consent was taken from parents.

Acknowledgement: All members of Pediatric Research Group, Sylhet, Bangladesh.

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Case Report

Unraveling Paget's Disease of the Skull and Spine and Therapeutic Response with Bisphosphonate: A Case Report

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Abstract:

Background: Paget's disease of bone is a chronic disorder characterized by abnormal bone remodeling, often leading to structurally compromised bones. Although it can be asymptomatic, involvement of the skull and other bones can cause significant complications. This case report presents a postmenopausal woman with Paget's disease, complicated by iron deficiency anemia and secondary hyperparathyroidism due to vitamin D deficiency.

Case Presentation: A 56-year-old woman with a 12-year history of asthma and hypertension presented with vertigo, a recent fall, and mild headaches. Examination revealed notable cranial deformities and scalp vein dilation. Investigations showed severe microcytic hypochromic anemia, elevated parathyroid hormone, low vitamin D, and high alkaline phosphatase levels. Imaging confirmed extensive Paget's disease affecting the skull and other bones. She was treated for iron deficiency anemia and vitamin D deficiency and received bisphosphonate therapy with intravenous zoledronic acid. Over five years, her biochemical markers normalized, though physical and radiological improvements were minimal.

Conclusion: This case highlights the importance of early diagnosis and long-term management of Paget's disease, particularly when complicated by metabolic disturbances. Comprehensive treatment, including vitamin D supplementation and bisphosphonates, can lead to significant biochemical improvement, although physical changes may persist. Compliance with treatment and regular follow-ups are crucial for successful disease management.

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Key words: Paget's disease of bone, vitamin D-deficiency, raised alkaline phosphatase, secondary hyperparathyroidism.

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Introduction:

Paget disease of bone (PDB) is a chronic progressive disease of the bone of uncertain etiology, characterized initially by an increase in bone resorption, followed by a disorganized and excessive formation of bone, leading to pain, fractures, and deformities.[1,2]

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This condition can manifest as a monostotic or polyostotic disease. Involvement of the skull can result in neurological symptoms such as vertigo, headaches, and cranial nerve compression, which significantly impacta patient's quality of life.[3]Though the exact etiology remains unclear, genetic, viral, immunogenic and neoplastic origins have been postulated. PDB often runs in families, and several genetic mutations, especially in the SQSTM1 gene have been linked to increased susceptibility. There is some evidence suggesting that viral infections (e.g., paramyxovirus) may trigger the abnormal bone remodeling process, though this theory remains controversial.[4,5]It is common in the Anglo-Saxon population, but is relatively rare in India. PDB is

often asymptomatic and is commoner among the aging population.[2,6]Its diagnosis is often incidental, made through imaging studies that reveal characteristic bone changes.[7]Markedly elevated serum alkaline phosphatase (SAP) is a constant feature while calcium and phosphate levels are usually within normal limits.[8] Management typically involves the use of bisphosphonates, a group of anti-resorptive drugs that can decrease morbidity and mortality associated with the disease.[8,9]

This article presents a complex case of Paget's disease in a postmenopausal woman, who had been hypertensive and asthmatic for many years, and then came to special attention due to a fall and subsequent revealing of characteristic radiological features. The diagnosis was then quite straight forward due the presence of the classic features of PDB with craniofacial involvement resulting in Leontiasis Ossea (lion like face), cotton wool appearance of the skull on CT scan of the head and an elevated SAP. However, her condition was complicated by the presence of severe iron deficiency anemia and secondary hyperparathyroidism due to vitamin D deficiency. This case highlights the incidental nature of diagnosis and the slow response to therapy due to interrupted intervention owing to the COVID-19 pandemic.

Case summary:

A 56-year-old housewife and mother of three, with a 12-year history of asthma and hypertension, presented to the outpatient medicine clinic with complaints of vertigo lasting several months and a recent fall. She also reported occasional mild headaches and back pain but denied experiencing loss of consciousness, bone pain, hearing loss, visual disturbances, or tingling and numbness in her limbs. She mentioned occasional chest pain and palpitations but had not experienced ankle swelling, frothy sputum, or orthopnea. She had a history of pallor following excessive menstrual bleeding before menopause, which she entered six years ago. Her current medications included Losartan potassium 50 mg daily for hypertension and a Salmeterol (25 µg) - Fluticasone (250 µg) inhaler, two puffs twice daily for asthma.

On examination, she appeared to have an average build, weighing 50 kg, and was fully conscious. Her pulse was regular, with a large volume, and recorded as 90 beats per minute. Her blood pressure was 160/80 mmHg, with no postural drop. She had notable physical features, including a large head with prominent frontal bossing, prominent cheeks, and a flat nose **(Figure 1-a, b)**. Dilated scalp veins, known as the 'scalp vein sign' **(Figure 1-c)**, were visible on her forehead. There was no localized tenderness over the skull bones, and no warm areas on the scalp were noted. Her breath sounds were vesicular with prolonged expiration, accompanied by a few rhonchi throughout the chest. A precordial flow murmur was detected. Neurological examination revealed no focal deficits. She appeared moderately anemic, though there were no signs of jaundice, purpura, bony tenderness, hepato-splenomegaly, or lymphadenopathy.

Routine blood tests **(Table 1)** revealed a hemoglobin level of 6.5 g/dL and an ESR of 53 mm in the first hour. Although her white blood cell and platelet counts were normal, the MCV (Mean Corpuscular Volume) was 67 fL, the MCH (Mean Corpuscular Hemoglobin) was 18 pg, MCHC (Mean Corpuscular Hemoglobin Concentration) was 26.87 gm/dL and the RDW (Red Cell Distribution Width) was 19.4%. Urinalysis and microscopic examination were normal. Other tests showed a normal serum TSH level of 2.93 µmol/L, creatinine at 0.5 mg/dL, albumin at 4.2 g/dL, and phosphate at 0.99 mmol/L. A peripheral blood smear confirmed microcytic hypochromic anemia with some poikilocytes. The iron profile was consistent with iron deficiency, showing serum iron at 12.4 µmol/L, ferritin at 5.1 ng/mL, total iron-binding capacity at 490 µmol/dL, and transferrin saturation at 2.53%. Lipid levels indicated mild dyslipidemia, with total cholesterol at 220 mmol/L, triglycerides at 201 mmol/L, HDL at 57 mmol/L, and LDL at 123 mmol/L. Serum calcium was slightly low at 8.8 mg/dL, vitamin D3 was deficient at 15 ng/mL, parathyroid hormone was elevated at 269.9 pg/mL, and alkaline phosphatase was high at 960 U/L. A chest X-ray was normal, and a 2-D and M-mode echocardiogram showed a 60% ejection fraction,

with normal chamber and valve dimensions **(Figure 2)**. Abdominal ultrasound results were normal, and a stool occult blood test was negative.

An X-ray skull lateral view showed widened deploic space of the frontal, parietal and, occipital bones with irregular densities **(Figure 3).** An axial CT scan of the head, performed to rule out head injury, revealed normal brain structures but incidentally identified mixed lucencies with osteolytic and osteoblastic areas of bone enlargement and coarsened trabeculae, giving a 'cotton wool' appearance **(Figure 4)** suggestive of Paget's disease of the skull. Fibrous dysplasia was also considered a strong radiological differential. A skull X-ray showed thickened, sclerosed, and irregular skull bones with scattered lucent areas. A Tc-99m HDP bone scan showed increased radiotracer uptake in the skull, several thoracic (specially 3rd and 4th) and lumbar vertebrae, sacral vertebrae, pelvic bones, and both sacroiliac joints **(Figure 5)**.

The patient was transfused with three units of packed red blood cells and started on daily oral iron polymaltose (47 mg) combined with folic acid (0.5 mg) and zinc (22.5 mg) to correct her anemia. Her hemoglobin increased to 10.3 g/dL after the transfusion. Along with treating her anemia, she was diagnosed with secondary hyperparathyroidism due to vitamin D deficiency, and suspected Paget's disease. She was prescribed 1000 mg of oral calcium and weekly 40,000 IU vitamin D supplements for eight weeks, after which she received an intravenous infusion of 5 mg zoledronic acid. She continued daily oral calcium (500 mg) and vitamin D (800 IU) supplements after the infusion.

Annual tests for serum alkaline phosphatase, calcium, phosphate, and parathyroid hormone were conducted. Unfortunately, she was lost to follow-up during the second year due to the COVID-19 pandemic but resumed treatment in the third year with repeated zoledronic acid injections at the end of the third and fourth years. By the fifth year, her serum alkaline phosphatase normalized to 81 U/L, and her levels of serum vitamin D3 (49.9 ng/mL), calcium (9.1 mg/dL), and parathyroid hormone (56 pg/mL) were also within normal limits. Bone densitometry showed improvement **(Figure 6)**, although there was little change in her physical appearance or bone scan findings. Throughout her treatment, adjustments were made to her antihypertensive, lipid-lowering, and asthma medications as needed.

Figure 1: a) large head with frontal bossing; b) flat nose, broad forehead; c) scalp vein sign

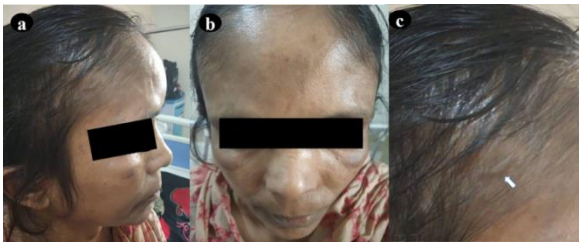

Figure 2: Normal echocardiographic features

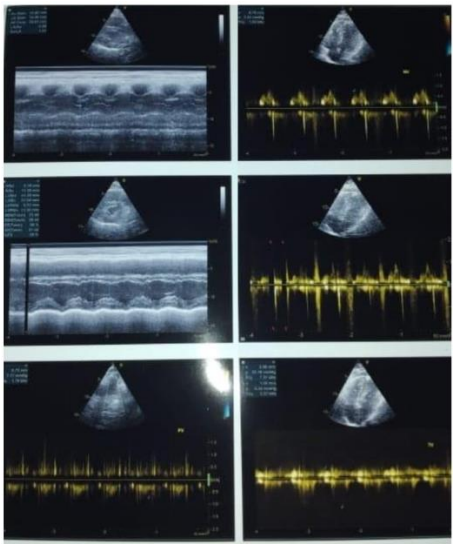

Figure 3: X-ray skull lateral view showing wide deploic space with irregular densities

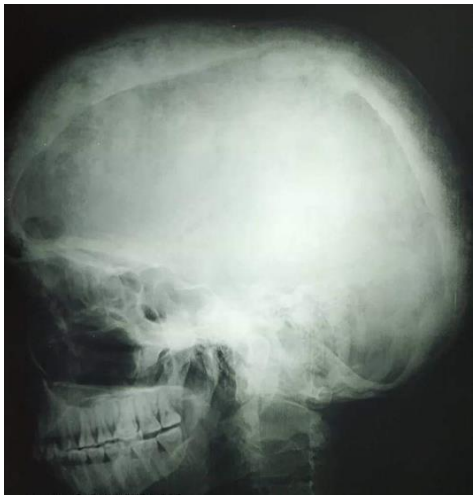

Figure 4: Mixed lucencies of osteolytic and osteoblastic zones (arrow) of bone enlargement and coarsened trabeculae

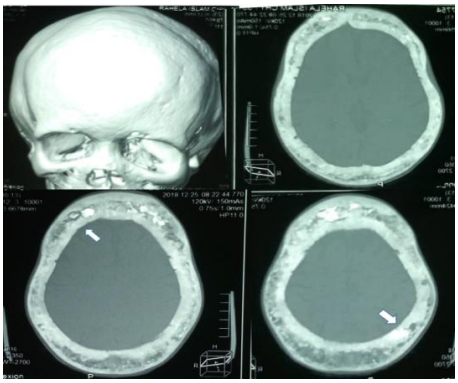

Figure 5: Isotope Tc-99m HDP whole body bone scan showing
increased radiotracer concentration in- a) skull, b) thoracic vertebrae,
c) lumbar vertebrae, d) all sacral vertebrae, e) pelvic bones

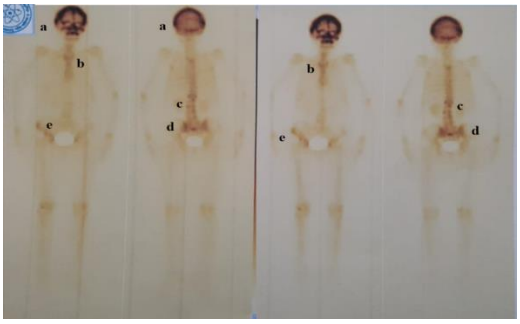

Figure 6: Normal bone densitometry at 5th year

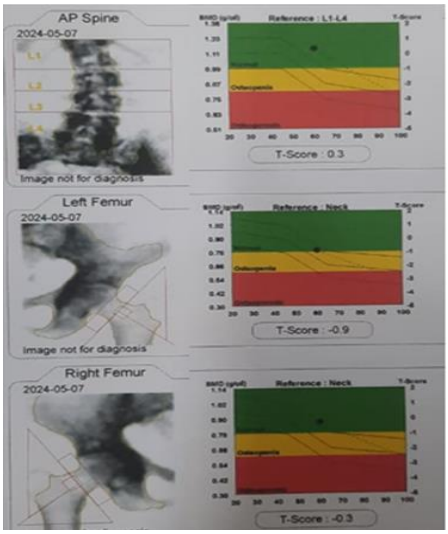

Table 1: Summary of blood test values

	Test parameter	At presentation	At follow up at 5th year
1	**Hemoglobin**	6.5 gm/dl	10.3 gm/dl
2	**ESR**	53mm	40mm
3	**Total count of WBC**	9,000/cu-mm	10,000/cu-mm
4	**Differential count of WBC**	N-60%, L- 35%, M-4%, E-1%, B- 0%	N-64%, L-32%, M-4%, E-0%, B-0%
5	**Platelet count**	1,60,000/cu-mm	2,10,400/cu-mm
6	**Red cell indices**	MCV- 67fL MCH- 18pg MCHC- 26.87 gm/dL RDW-19.4%	MCV- 87fL MCH- 30pg MCHC- 34.48 gm/dL RDW-12.3%
7	**Iron profile**	Serum Iron- 12.4 µmol/L Serum Ferritin- 5.1 ng/mL TIBC- 490 µmol/dL Percent saturation- 2.53%	-
8	**Creatinine**	0.5mg/dl	0.91mg/dl
9	**Electrolytes**	Na- 137 mmol/L K- 4.0momol/L Cl- 102 mmol/L HCO3-22 mmol/L	Na- 135 mmol/L K- 3.6 momol/L Cl- 110 mmol/L HCO3- 23mmol/L
10	**Fasting lipid profile**	TC- 220 mmol/L TG- 201mmol/L HDL- 57mmol/L LDL- 123mmmol/L	-
11	**Serum TSH**	2.93 µmol/L	1.06 µmol/L
12	**Serum parathyroid hormone**	269.9 pg/mL	56 pg/mL

13	**Serum alkaline phosphatase**	960 U/L	81 U/L
14	**ABG**	PH- 7.43 PaO2- 65mmHg CO2-30mmHg HCO3- 22mmol/L	-
15	**Serum albumin**	4.2 g/dL	-
16	**Serum Calcium**	8.8 mg/dL	9.1 mg/dL
17	**Serum phosphate**	0.99 mmol/L	0.82mmol/L
18	**Serum vitamin D_3**	15 ng/mL	49.9 ng/mL

ESR-Erythrocyte Sedimentation Rate, WBC-White Blood Corpuscle, TSH-Thyroid Stimulating Hormone, ABG-Arterial Blood Gas

Discussion:

Paget's disease of bone (PDB), also known as osteitis deformans, is rare in Asia, Africa, the Middle East, and Scandinavia. Epidemiological trends have demonstrated an increasing prevalence in the United States and, an overall decrease in prevalence in the United Kingdom and other European countries over the past two decades. The disease prevalence increases after 55 years of age in all communities. However, the exact reasons behind such geographical and age distribution are unclear.[6]

Diagnosis is often incidental to radiology or raised alkaline phosphatase levels.[7,11] The reported case, who was in her late fifties, had been seeking medical help for hypertension and asthma for many years until she underwent a CT scan of her head following a fall and it was then only, that the diagnosis of PDB incidentally unraveled. Such incidental diagnosis in asymptomatic patients is quite common in this condition.[12]

Though bone pain, bone deformities (e.g., bowing of long bones, skull enlargement), fractures, and hearing loss are the common skeletal symptoms, the index patient had minimal symptoms of PDB. Her vertigo might be due to her severe anemia as she had no vestibulo-cochlear problem. However, it could also be due to the involvement of the skull due to PDB. Other than this she had no other symptoms attributable to PDB. Other skeletal sites of involvement she had were the pelvis and, the vertebral bodies without any symptom of involvement. Long bones of the lower limbs are the affected sites, as reported in other cases.[6,13] The severe microcytic, hypochromic anemia that she had, as reflected by her low hemoglobin, reduced MCV, and MCH was most likely due to iron deficiency due to menorrhagia. The patient's persistent symptoms of fatigue and dizziness with a history of recent falls required correction through iron supplementation and packed cell transfusion. Her positive response to treatment was reflected by the rise in hemoglobin to 10.3 g/dL following the packed cell transfusion and the iron supplementation.

The pathogenesis of PDB involves abnormal bone destruction by increased osteoclastic activity followed by disorganized bone formation by osteoblastic activity, resulting in structurally weak and deformed bones. Some have described three stages of pathogenesis, i.e. the lytic stage (due to osteoclastic activity), the mixed stage (both osteoclastic and osteoblastic activity), and the inactive stage (due to osteoblastic activity). The pathological fractures of the affected bones take place during the lytic stage and also in the mixed stage due to the formation of large, deformed, and disorganized bones. The disease manifestations gradually decline in the inactive stage.[14]This forms the basis of therapy with bisphosphonates that induce remission through enhanced osteoblastic activity.[15]

The diagnosis of Paget's disease of bone (PDB) primarily relies on the radiological and biochemical findings. In this patient, an early CT scan of the head, performed for an unrelated reason, incidentally revealed characteristic features of PDB.[6] Typically, CT or MRI scans of the skeleton are not necessary after plain X-rays have been used for diagnosis. However, CT scans can be useful for identifying fractures, and MRI scans can detect missed malignant lesions such as sarcomas, giant cell tumors, or metastases within the pagetic bone.[8,16] X-rays are important primary investigations as they can reveal PDB by a number of radiological features like osteolytic/osteosclerotic areas, thickened cortex/ trabeculae and bone expansion/deformity. In the index case, plain X-ray skull was doneon purpose after the CT scan,

and the both of themrevealed features of PDB. As a result, the classical radiographic signs of PDB, such as the 'picture-frame vertebra' or the 'ivory vertebra' in the spine, or the 'brim sign' in the pelvis, were not explored. [8,16,17]

While the radionuclide bone scans or scintigraph offer greater sensitivity as compared to plain radiographs, they have lower specificity in diagnosis. The low specificity of this test is because of its failure to differentiate between bone metastasis, metabolic bone disease and, fibrous dysplasia.[12,15] The index case was diagnosed with the help of combined typical features in CT scan and bone scan. Fibrous dysplasia was a strong differential according to the CT scan features. However, scintigraphy revealing a well preserved bony outline favored the diagnosis of PDB. Smith *et al* found that more than 70% of the symptomatic patients with positive scintigraph have normal bone radiograph.[18,19]

Among biochemical markers, serum alkaline phosphatase (ALP) is the marker of choice, as its elevation correlates with disease severity and activity. This test is also very cheap and widely available.[14,20]Serum osteocalcin, a marker of bone formation, is not recommended for diagnosis or prognosis in clinical practice. More recently, the bone formation marker P1NP and bone resorption markers such as N-telopeptide or C-telopeptide have been considered better alternatives to total ALP. Serum calcium and phosphate levels are usually within normal ranges in the PDB. Sometimes there are minimally decreased calcium and elevated parathyroid hormone levels.[8,21] The index case had a complicated scenario of mild hypocalcemia, hypovitaminosis D and hyperparathyroidism.

The treatment strategy of PDB has changed with the development of potent and effective drugs. At present, treatment is indicated for both symptomatic and asymptomatic patients with active disease and with risk of complications. Considering the involvement of weight-bearing vertebrae, the index patient was subjected to bisphosphonate therapy. In 90% of cases ALP levels normalize within the first year after therapy.[21,22] But the reported case was slow to respond. This might be due to the interrupted follow-up due to the COVID-19 pandemic or may be due to other existing metabolic derangements in the patient. Though subcutaneous injection of salmon calcitonin has been approved as a treatment for PDB, it is rarely used now.[23]

Conclusion:

This case report demonstrates the complexity of managing multiple interrelated conditions in an elderly patient, particularly the interplay between Paget's disease, vitamin D deficiency, and iron deficiency anemia. Paget's disease, although often asymptomatic, can present with complications when the skull is involved, such as vertigo and headaches. Early diagnosis and appropriate treatment with bisphosphonates and vitamin D supplementation can lead to significant biochemical improvement, although physical and radiological changes may persist.

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Case Report

Hidden Intricacies of Chronic Liver Disease in a Young Adult: A Case Report on Wilson's Disease

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Abstract:

Wilson's disease, a rare autosomal recessive disorder of copper metabolism, often manifests with hepatic, neurological, or psychiatric symptoms due to toxic copper accumulation. This case report describes a 20-year-old male from a remote tea garden in Sylhet, Bangladesh, diagnosed with Wilson's disease during an evaluation for chronic liver disease. The patient presented with anemia, jaundice, ascites, and splenomegaly. Investigations revealed elevated liver enzymes, suggestive ultrasonographic findings, a raised hepatic Fibro-Scan score, and esophageal varices. Notably, the patient had a family history of Wilson's disease, with two older siblings previously diagnosed. Despite the absence of consanguinity between the parents, this prompted further biochemical workup. The patient's serum ceruloplasmin level was low, although 24-hour urinary copper excretion was within the normal range. Slit-lamp microscopy confirmed the presence of a Kayser-Fleischer ring in both the patient and the affected siblings. He was started on chelation therapy with penicillamine, alongside supportive management for chronic liver disease. This case highlights the extraordinary misfortune of three siblings from a non-consanguineous family being affected by a rare autosomal recessive disorder.

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Key words: Wilson's disease, Copper accumulation, hepatolenticular degeneration, Kayser Fleischer ring, hepatic elastogram, chronic liver disease

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Introduction:

Wilson's disease (WD) is a rare genetic disorder characterized by excessive copper accumulation in the body, leading to damage in the liver, brain, kidneys, and corneas.[1]

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It is caused by mutations in the ATP7B gene that impair copper transport, preventing copper from being excreted into bile or incorporated into ceruloplasmin, the copper-carrying protein in the blood.[2] Though consanguinity between parents is an expected condition in WD, its autosomal recessive inheritance pattern can affect individuals in any population, even from non-sanguineous parentage.[3]WD has a global prevalence of 1 in 30,000 to 1 in 40,000 live births.[4]The clinical presentation of WD pertaining to three main categories of symptoms i.e., hepatic, neurological, and neuropsychiatric manifestations, can be present independently or together, depending on factors like age, genetic variability, and the extent of copper accumulation.[5]Around half of WD patients present with liver-related symptoms, as is the reported case.[6]A high index of clinical suspicion is the key to prompt diagnosis of WD.[7] Slit lamp

ophthalmoscopic evaluation can be a pathognomonic, thus an important tool for diagnosis.[8] The non-invasive biomarkers, serum ceruloplasmin levels and urinary copper excretion, are crucial in monitoring disease progression.[5,6] Enhanced imaging techniques like quantitative MRI have also improved the accuracy of liver and brain assessments.[9] Genetic testing for diagnosis has evolved with the modern era but is seldom applied as a common practice.[10,11]WD is one of the treatable causes of chronic liver disease. So, early diagnosis and treatment have an enormous impact on outcome.[7,12] Moreover, its primary treatment is very simple with one of a few recommended oral medications. Zinc supplementation prevents copper absorption; penicillamine and trientine act by copper chelation in the treatment of WD.[5,6] This case report, a young man presenting with chronic liver disease with a background of positive family history, highlights the necessity of screening of siblings in relevant families so that the disease can be detected and treated at an early stage.

Case summary:

A 20-year-old male presented to the medicine inpatient department of Sylhet Women's Medical College with generalized body swelling for one week, which started in the abdomen. He mentioned generalized weakness and loss of appetite for the same period. Over the past nine months, he noticed yellowing of his skin and eyes. The abdominal swelling was not associated with pain, itching, dark urine, or pale stools. There was no history of hematemesis, melena, behavioral changes, or loss of consciousness. Additionally, there was no recent use of hepatotoxic drugs or blood transfusions. He experienced occasional difficulty in breathing onlying down, though this was not accompanied by fever, cough, palpitations, or chest pain. He was the youngest of four siblings, born to non-consanguineous parents, and two of his elder sisters had been diagnosed with Wilson's disease several years ago. On physical examination, he had anemia, jaundice, mild edema, huge ascites, and splenomegaly suggestive of chronic liver disease **(Figure 1)**. However, other stigmata of chronic liver disease (CLD) were absent. His jugular venous pressure was normal, and bedside urine examination did not show evidence of proteinuria or glycosuria. Examination of the respiratory system revealed a few coarse crepitations at the left lung base. However, the cardiovascular, locomotor, renal and neurological systems revealed unremarkable features.

His blood counts revealed hemoglobin 3.9 g/dl, ESR 02 mm, total count of WBC 1,720 /cu-mm with neutrophil 63% lymphocyte 27%, eosinophil 05%, monocyte 05%, platelet 50,000/cu-mm and, red cell 2,68,000 /cu-mm. Pancytopenia was the impression on peripheral blood film examination. His serum creatinine (0.76 mg/dl) and, electrolytes (sodium 135, potassium 3.6, chloride 110 and, bicarbonate 24 mmol/L) were normal. His iron profile revealed a low serum iron (12 mcg/dL) and, ferritin (8ng/mL). The liver function tests revealed some derangements, notably a raised serum bilirubin (3.8 mg/dl), reduced albumin (1.8 gm/dl), mildly raised alanine transaminase (59 U/L), alkaline phosphatase (223U/L) and, prolonged prothrombin time (25 sec) with INR 50. His viral markers, HBsAg and, anti HCV were negative. Urine for routine and microscopic examination revealed normal findings. USG of the whole abdomen showed coarse hepatic parenchyma, splenomegaly, huge ascites and, a thickened wall of gall bladder **(Figure 2a)**. Endoscopy of the upper gastrointestinal tract revealed medium to large varices at the lower end of the esophagus (Figure 2b). A hepatic elastogram revealed a hepatic stiffness index of 24.5 kPa indicating stage 4 (16-75 kPa) fibrosis **(Figure 3)**. His serum ceruloplasmin is low at 0.010 g/L (0.15-0.30), and 24-hour urinary copper excretion is 12.62 mcg/24 hours. On slit lamp examination of the eyes by an expert ophthalmologist showed bilateral Kayser Fleischerrings in the corneas **(Figure 4)**. His ECG was normal and chest X-ray showed a few opacities at the left lower zone **(Figure 5)**.

Figure 1: Ascites, pale complexion, jaundice

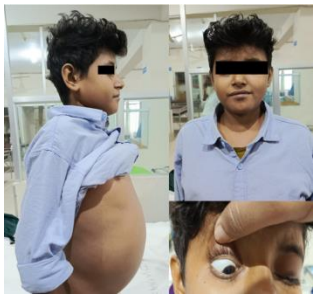


Figure 2: a. Ultrasonography of abdomen showing coarse liver, enlarged spleen and ascites b. Endoscopy showing esophageal varices. Please note arrows.

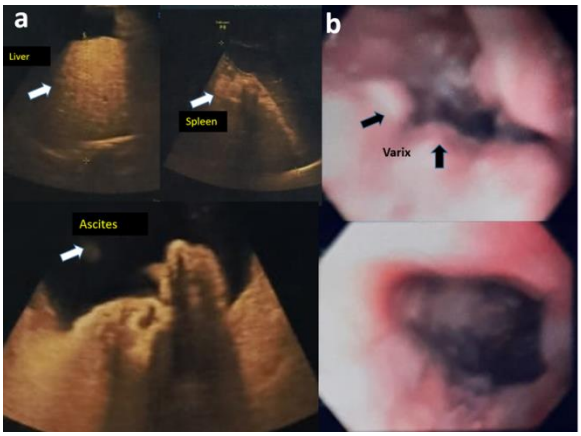


Figure 3: Hepatic elastogram revealing stiffness index of the liver

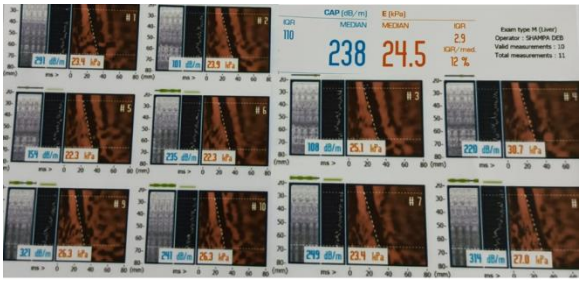


Figure 4: Slit-lamp microscopy showing brownish deposition of copper at the sclera-corneal junction- the KF ring (arrows)

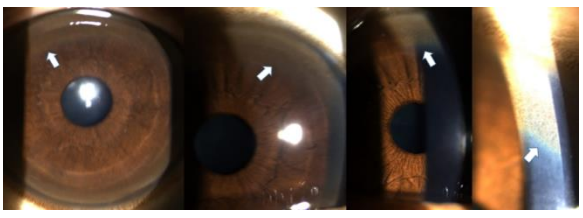
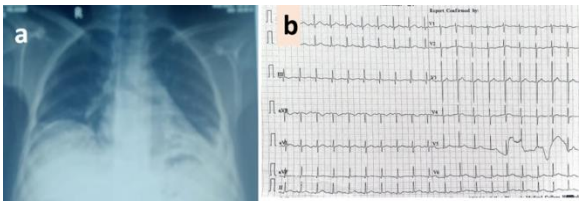


Figure 5: a. Chest X-ray showing a few opacities in left lower zone b. Normal ECG



Discussion:

Wilson's disease (WD) is an autosomal recessive disorder requiring the inheritance of two defective copies of the *ATP7B* gene, one from each parent, to manifest.[13]WD is more prevalent in populations with consanguineous marriages due to the increased likelihood of both parents carrying the same genetic variant. However, it can also occur in individuals born to non-consanguineous parents,[14] as was the case in this report. Unfortunately, this rare genetic transmission affected multiple siblings in this family. The index case has two elder sisters with WD who are living normal lives with medication. Both sisters were diagnosed at the same medical facility seven years ago, with the eldest sister's case reported at that time.[15]The second sister was screened and diagnosed concurrently, but the youngest brother missed screening due to the family's remote living conditions and failure to follow up.

Liver-related clinical presentation is more common than neurological or neuropsychiatric presentation, particularly in younger individuals with the genetic defect. These patients may present with hepatitis, fatty liver, cirrhosis, or acute liver failure.[1,2,16] Around 40% of cases, especially in older adolescents or young adults, exhibit neuropsychiatric features such as movement disorders, tremors, dystonia, and dysarthria, caused by copper accumulation in the brain. Parkinsonism-like symptoms may arise when deposition occurs in the basal ganglia. Neurological symptoms typically present later than hepatic symptoms and may dominate in those diagnosed later in life.[17]Additionally, 20–30% of patients present with mixed hepatic and neuropsychiatric symptoms, such as signs of chronic liver disease alongside cognitive decline, depression, mood swings, or personality

changes. Mixed presentations complicate management, as both hepatic and neurological symptoms require tailored treatment. Neuropsychiatric symptoms can be subtle and are often underreported, affecting an estimated 10-20% of patients, especially in the early stages. Symptoms include cognitive changes, depression, irritability, personality changes, and mood disorders, which can sometimes precede more overt neurological or hepatic symptoms.[9,10] In the index case, the patient and his siblings did not exhibit any neurological or psychiatric symptoms, although brain imaging was not conducted. Despite a low serum ceruloplasmin level, urinary copper excretion was within the normal range. It is worth noting that low ceruloplasmin levels can occur alongside hypoalbuminemia (1.8 g/dL), and urinary copper excretion levels, theoretically expected to exceed 100 mcg/dL, do not definitively rule out other chronic liver diseases.[6]However, urinary copper excretion can at times be false negative or false positive.[18]The diagnosis of WD in the reported case is primarily based on a strong family history and the presence of Kayser-Fleischer rings on slit-lamp examination, raising some concerns among investigators regarding the sufficiency of these criteria.[8,19] However, in the case of the patient's elder sister, the diagnosis was established through the fulfillment of the Leipzig scoring system having a score of 7.[15]

Markers for viral hepatitis, common causes of cirrhosis, were negative. Serum iron (12 mcg/dL) and ferritin (8 ng/mL) levels were low, but hypoalbuminemia likely influenced these values, complicating their interpretation.[20] However, iron deficiency anemia was considered a plausible cause of the patient's anemia, particularly given the presence of esophageal varices, which may have caused chronic blood loss. Treatment focused on copper chelation using oral penicillamine and, endoscopic band ligation was planned for the esophageal varices.

Advances in genetic screening enable earlier diagnosis, even in asymptomatic individuals, through family screening and newborn testing. Numerous mutations in the responsible gene have been identified that influence the severity of the condition.[3,7]However, genetic screening for WD is not routinely performed unless there is a family history or clinical suspicion. As a result, estimates of prevalence in non-consanguineous families rely on general carrier rates and probabilities associated with recessive inheritance.[6,9]Though genetic testing was beyond the means of the family of this case and the healthcare facility, counseling was provided regarding the hereditary nature of the disease, the importance of avoiding consanguineous marriages within the family, and the need for screening the next generation.

Zinc supplementation, which inhibits copper absorption, has gained popularity as an effective therapy, particularly in pre-symptomatic and mild cases. Chelating agents such as penicillamine and, trientine remain primary treatments, though newer formulations with fewer side effects are being explored. For instance, trientine has shown potential in reducing copper levels with lower toxicity.[7]A systematic review of 23 studies, involving over 2,000 patients, aimed to compare the therapeutic benefits and drawbacks of zinc, penicillamine, trientine, succimer, and no treatment. The findings indicated that zinc was safer than penicillamine while offering comparable therapeutic efficacy. However, the authors expressed concerns about the quality of the included trials.[21]The index case and his two siblings were treated with penicillamine due to its availability, and all three tolerated the therapy well.

Recent advancements in the management of Wilson's disease (WD) include experimental gene therapy targeting ATP7B gene mutations, which holds promise as a potential cure.[22,23] Progress has also been made in understanding the neurological effects of WD, including motor and cognitive symptoms. Innovative strategies, such as neuroprotective drugs and rehabilitative therapies, are being developed to address these issues and enhance patients' quality of life.[24] Liver transplantation remains a treatment option for patients who respond poorly to medical therapy or present with acute liver failure.[25]

Early diagnosis and targeted therapy are critical, as they can prevent severe organ damage and allow individuals with WD to ensure healthier lives of the patients. Ongoing research and clinical trials continue to improve understanding and management of this condition.

Conclusion:

In conclusion, WD exhibits a wide range of symptom patterns. Hepatic manifestations are more common in younger patients, while neurological symptoms are more prevalent in adults. Almost one third of the patients experience both hepatic and neurological or neuropsychiatric symptoms. These varied presentations highlight the critical need for early and accurate diagnosis, as timely treatment can profoundly influence the disease progression and thus improve quality of life. This case report emphasizes the importance of screening all family members, even in the absence of consanguinity.

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Case Report

An Unusual Case of Adult Intussusception - A Case Report

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Abstract:

Introduction: In Adults intussusception as a cause of small intestinal obstruction is a rare condition. Among the lead points for intussusception intestinal polyps are most common and also there are probabilities of malignant tumors which usually become greater from proximal to distal colon. Fibroid polyps are most uncommon benign pathology leading to intussusception of small bowel, presented in our case report.

Case presentation: A 45 years old lady admitted in surgery ward of Sylhet Women's Medical College Hospital with the features of small bowel obstruction for 5days. The abdominal ultrasonography report was suggestive of bowel mass resulting small bowel obstruction. Than multi axial contrast computed tomography of whole abdomen was done, which showed jejunoileal intussusception with intestinal obstruction. An intussusception with an intraluminal solid tumour at its lead point approximately 240cm distal to dudenojejunal flexure was found on exploratory laparotomy. Resection and anastomosis of affected bowel segment was done. Histopathological examination showed inflammatory fibroid polyp, ulcerated.

Conclusion: In case of small bowel obstruction due to intussusception with a lead point of inflammatory fibroid polyp surgical approach is the most preferable solution though it is a benign condition.

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Key words: Intussusception, bowel mass, fibroid polyp, intestinal obstruction.

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Introduction:

Intussusception is an unusual cause of small intestinal obstruction in adults.[1]Patients with intussusception usually present with either acute or chronic intermittent symptoms. In adults, cases are almost invariably associated with a lead point, which is usually a polyp (e.g. Peutz-Jeghers syndrome), a submucosal lipoma or other tumour.[2]We report a rare case of ileoileal intussusception in an adult patient caused by an inflammatory fibroid polyp, whose diagnosis was evidenced by histopathology.

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Case presentation:

A 45 years old lady admitted in surgery ward of SWMCH with the history of abdominal pain, nausea and absolute constipation for 5days. She had a complaint of intermittent constipation for last two years which was usually relieved by taking laxatives except the last episode. There was no history of hypertension, diabetes mellitus and any previous abdominal surgery.

On examination, she was anxious, dehydrated, had a heart rate of 110b/min, blood pressure 140/70mmHg and a temperature of 37.5ºC. Her abdomen was distended without any features of peritonitis, bowel sound was exaggerated. There was no abnormality found on Digital Rectal Examination (DRE).

Laboratory analysis demonstrated 6,700/cumm leukocytes with a predominance of neutrophils. Other laboratory findings were within the

normal limits. Abdominal radiology showed multiple air-fluid levels in central abdomen. The abdominal sonography report was suggestive of bowel mass resulting small bowel obstruction. Than multi axial contrast computed tomography was done, which showed jejunoileal intussusception with intestinal obstruction.

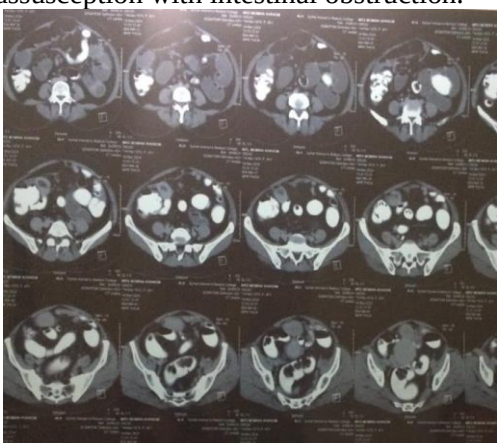

Figure 1: CT scan film of multiple axial section showing dilated small bowel loops with small bowel intussusception.

With the diagnosis of acute intestinal obstruction an exploratory laparotomy was performed. An intussusception with a mass lesion at its lead point approximately 240cm distal to dudenojejunal flexure was found. Intussusception was reduced during operation. Limited edema and erythema was present at the intussusceptum segment, which was the sign of the intussusception. Proximal jejunum was found distended. Affected segment was excised followed by end-to-end anastomosis. After opening of the lumen of resected specimen there was an intraluminal polypoid solid mass, approximately 4x3cmin size. Histopathological examination showed inflammatory fibroid polyp, ulcerated.

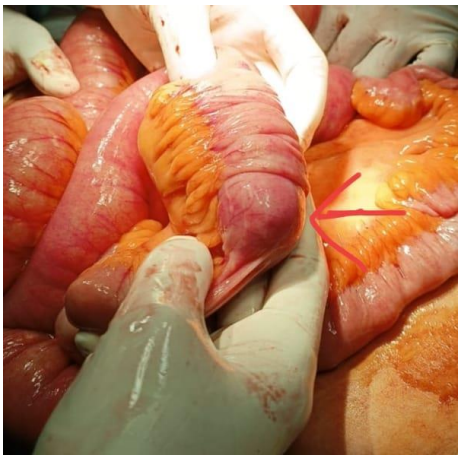

Figure 2: The lead point of intussusception

Figure 3: Resected specimen after cross-section

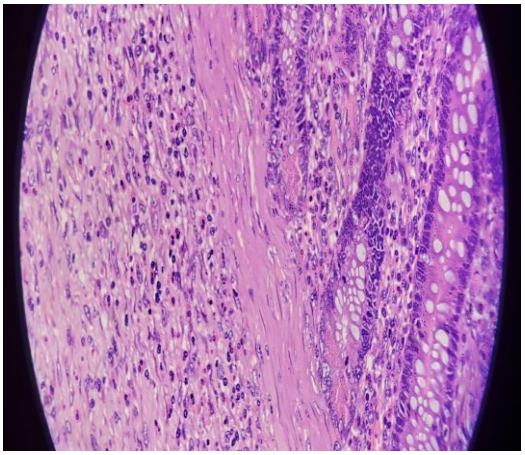

Figure 4: Microscopic view of cross section of the polypoid mass showing fibroblast, smooth muscle including chronic inflammatory cells.

Discussion:
Introversion of one portion of gut into an immediately adjoining segment is called intussusception. It is unusual in grown persons, considering for only 1-5% of all cases of bowel obstruction.[3] Adult intussusceptions are usually secondary to intestinal pathology, e.g. polyp, Meckel's diverticulum, benign or malignant tumors which act as a lead point.

Among these, lead points Inflammatory Fibroid Polyp (IFP) is a rare nonmalignant lesion of the intestinal tract. It commonly occurs among older adults in the fifth to sixth decade and has a slight predominance among females. This tumor usually affects the stomach followed by the small bowel and rarely the large bowel.[4] Patients usually present with gastrointestinal bleeding, abdominal pain and other symptoms of obstruction, and could also present as intussusception in an emergency situation like that of our case.

As there is possibility of malignancy or other pathological abnormalities, adult intussusceptions are managed by surgical approaches like laparotomy or laparoscopy.[5] Regarding the surgical options for intussusception, it still debatable among reduction of intussusception lesion or "en-bloc"resection without reduction or reduction followed by resection.[4]Each method has advantages and dis advantages; only reduction may cause recurrence, resection without reduction decreases the risk of tumor dissemination and extension, and reduction before resection lowers the risk of short-bowel syndrome. So, we have to decide the appropriate surgical option for every individual patient according to existence of malignancy or presence of ischemia of the affected bowel, site and extent of the lesion.

Conclusion:
Intussusception is an unusual cause for adult intestinal obstruction and inflammatory fibroid polyp is an infrequent cause of this unusual condition. Although IFPs are benign intestinal lesions, surgical treatment is the most preferred treatment option when the patient presents with small intestinal obstruction.

Patient perspective
Patient mentioned that she is now doing well and backed to her normal life, without any postoperative complications. An informed consent for this case report was taken from the patient.

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Conflict of interest: The authors declared no conflict of interest with this study.

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Short Communication

Dengue Outbreak in Sylhet, 2023: Experience at Pediatric Unit of a Tertiary Care Teaching Hospital

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Abstract:

Now a day's dengue is a global public health concerned, especially in tropical and subtropical countries. In 2023, dengue outbreak affects all 64 districts of Bangladesh. This prospective study was done at Department of Paediatrics, Sylhet M A G Osmani Medical College Hospital (SOMCH), Bangladesh during 2023 dengue pandemic. We found dengue test (NS1, dengue IgM) positive in only 38% children, others were diagnosed clinically. Warning signs were present in 25% cases (like diarrhea, vomiting, abdominal pain, hepatomegaly and restlessness), severe dengue were 25% cases (shock with AKI, dengue encephalitis, severe bleeding), isolated organopathy (dengue encephalitis) (16.7%) and co-infection was found in 17% cases.

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Introduction:

The World Health Organization reported the highest global burden of dengue fever cases in 2023, with the disease impacting over 80 countries.[1] Dengue is a mosquito-borne viral illness primarily transmitted by infected *Aedes* mosquitoes.[2]

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Dengue virus [DENV]- 1,2,3 and 4 are four distinct serotypes and a potential fifth serotype (DENV-5) recently identified in Malaysia.[2] In the capital city of Bangladesh, Dhaka the first recognized dengue outbreak was reported in 1964.[3]After then, sporadic dengue cases were reported until 2000,[4]when Bangladesh's major cities and towns saw its first widespread epidemic.[5]The dengue epidemic of 2023 hit all 64 districts, including Sylhet.[6]

Material and Methods:

This prospective observational study was done at Department of Paediatrics, Sylhet M A G Osmani Medical College Hospital (SOMCH), Bangladesh from July, 2023 to August, 2023. Children age less than 13 years admitted at SOMCH and diagnosed as case of dengue, were included in study.

Result and discussion:

Total 24 patients were admitted, among them, male 75% (mean age 70.83 ± 46.2 months), female 25% (mean age 75.33±42.98 months). Youngest patient was a 7 month old infant. Presenting clinical features were fever (100%),

body ache (8.3%), severe muscle pain (4.2%), rash (4.2%), headache (4.2%), diarrhea (12.5%), vomiting (8.3%), abdominal pain (4.3%), cough (16.8%), Convulsion (16.8%) and melena (4.2%). Most of the diagnosis was done on the basis of clinical features with leukopenia and thrombocytopenia (62.5%), dengue NS1 (25%), dengue IgM (8.3%) and dengue IgM with IgG (4.2%). In most of the cases NS1 was negative. This explain as DENV2 (51.5%) and DENV3 (43.9%) were the predominant serotype in 2023 outbreak.[7] Clinical diagnosis of admitted patients were dengue fever (62.5%), dengue hemorrhagic fever (16.7%), dengue expanded shock syndrome (shock with AKI) (4.2%) and isolated organopathy (dengue encephalitis) (16.7%). Warning signs were present in 25% cases (diarrhea, vomiting, abdominal pain, hepatomegaly and restlessness) and severe dengue was 25% cases (shock with AKI, dengue encephalitis, severe bleeding). Management was given according to national guideline for dengue management. Platelet transfusion was given to none and PRBC was given to a patient with melena. No death was encountered. These findings are little bit different from other study,[8]as most of the cases were primary dengue (only 4.2% Dengue IgG positive). Co-infection was found in 17% patient like rickettsial fever (8.3%), hepatitis A (4.2%) urinary tract infection (4.2%). Co-infection in dengue is not so uncommon and it has effect in dengue outcome.[9]

Conclusion:

Dengue cannot be excluded by negative dengue test (NS1, dengue IgM). Isolated organopathy and co-infection are not so uncommon.

Acknowledgement: All members of Pediatric Research Group, Sylhet, Bangladesh.

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