Volume 1 • Issue 2 • January 2026

ISSN (Print): 3106-3780

BGS

Bangladesh Journal of Gastrointestinal and Liver Diseases

BANGLADESH GASTROENTEROLOGY SOCIETY

Contents

Bangladesh Journal of Gastrointestinal and Liver Diseases

Volume 1 • Issue 2 • January 2026

An Official Publication of the Bangladesh Gastroenterology Society

Bangladesh Journal of Gastrointestinal and Liver Diseases

Volume 1 • Issue 2 • January 2026

An Official Publication of the Bangladesh Gastroenterology Society

Board of Reviewers (Bangladesh)

Board of Reviewers (International)

Bangladesh Journal of Gastrointestinal and Liver Diseases

Volume 1 • Issue 2 • January 2026

An Official Publication of the Bangladesh Gastroenterology Society

Bangladesh Journal of Gastrointestinal and Liver Diseases

The Bangladesh Journal of Gastrointestinal and Liver diseases (BJGL) is a biannually published journal dedicated to advancing knowledge and promoting research in the field of gastroenterology and liver diseases. BJGL welcomes submissions of high-quality original articles, review articles, case reports, special articles, case reports, different gastrointestinal images (radiological and endoscopic), letter to the editor and short communications related to various topics of gastrointestinal and liver diseases. Please read these instructions carefully to ensure your submission adheres to our guidelines. Manuscripts that do not comply with these instructions may be returned to the authors without review.

Scope of the Journal

BJGL publishes articles on all aspects of gastroenterology, hepatology, gastrointestinal surgery,
gastrointestinal oncology, endoscopy, and related fields. All submissions should be relevant to the study, diagnosis, treatment and prevention of gastrointestinal and liver diseases.

Submission Process

• Manuscripts should be submitted electronically via the journal's email: journalbgs@gmail.com or bgsdigestivediseasechronicle@gmail.com

• A cover letter must accompany the submission, confirming that the manuscript is original, has not been previously published, and is not under consideration for publication elsewhere. The letter should clearly state the type of article (e.g., original research, review, case report). The letter should provide the full contact details (including email, phone number, and affiliation) of the corresponding author.

Manuscript Preparation

General Format

• Manuscripts should be submitted in English.

• Use double spacing throughout, including Referencess, tables, and figure legends.

• Use 12-point Times New Roman font.

• Submit the manuscript in Microsoft Word (.doc or .docx) format.

Images/Figures:

Submit high-resolution images in JPEG, or PNG format. Ensure that figures are numbered consecutively and cited in the text. Should also be in the result section and approximately follow the text References. They should have brief title, cited consecutively in the text and numbered using Arabic numerals (e.g Figure 1, 2) at the bottom of the figure or images.

• Number all pages consecutively, starting with the title page.

• Use one-inch (2.5 cm) margins on all sides.

• Tables:

Create tables using the table function in Microsoft Word. Do not submit tables as images. The total number of tables and figures combined should not exceed five. Should be in the result section and should approximately follow the text References. All tables should be numbered consecutively using Arabic numerals (e.g Table 1, 2) on the top of the table and must be cited in the text in numerical order. Each table must carry a brief title and avoid internal horizontal and vertical lines. Include explanatory footnotes for tables, if necessary

Manuscript Structure:

Title Page:

The title should be concise and informative. Title should contain of the names of all authors, highest academic degrees, institutional affiliations, corresponding author's name, address, email and telephone number.

Abstract:

Structured (Background, Methods, Results, Conclusion) within 250 words Three to six key words should be included on the abstract page under the heading'Keywords'.

Main Text:

IMRAD format: Introduction, Materials and Methods, Results, Discussion. In result section, duplication of information that is presented in tables or figures should be minimal in the text. The maximum length of the main text (excluding the abstract, tables, figures and Referencess) should be 5000 words.

Conclusion:

This section should clearly state the main conclusions of the study and may include relevant recommendations based on the findings.

Acknowledgments:

Acknowledge any individuals or organizations that provided support for the study (e.g., funding sources, technical assistance).

Conflict of Interest Disclosure:

Declare any potential conflicts of interest of any author (e.g., financial interests, personal relationships). If there are no conflicts of interest, state "The authors declare that they have no conflicts of interest."

• Sections for different manuscripts

***Original Article*:**

Abstract (Structured) with Keywords, Introduction, Materials and Methods, Results, Discussion (including limitations), Conclusion (including recommendations), Acknowledgement (including contribution of authors), Disclosure of funding and conflict of interest, Referencess.

***Review Article*:**

Abstract (Structured) with Keywords, Introduction, Search Strategy, Discussion, Conclusion

Bangladesh Journal of Gastrointestinal and Liver Diseases
Volume 1 • Issue 2 • January 2026
An Official Publication of the Bangladesh Gastroenterology Society

(including recommendations), Disclosure of funding and conflict of interest, Referencess.

***Case Report*:**
Abstract with Keywords, Introduction, Case Report (may include a maximum of 3 tables, figures, or pictures), Discussion, Conclusion (including recommendations), Acknowledgement (including contribution of authors),Disclosure of funding and conflict of interest, Referencess.

***Special Article*:**
The format may follow that of an Original Article or a Review Article, depending on the nature of the submission. Please consult the editors if you have questions regarding the structure of a special article.

***Letter to the Editor*:**
Communications pertaining to articles recently published in the journal or addressing current health problems of importance in the field will be considered as Letters to the Editor. Letters should be concise, with a maximum length of 750 words.

References:
• Referencess should be numbered consecutively in the order they appear in the text.
• Use Vancouver style.
• Examples:
• Journal Article:
Rahman MM, Ahmed S. Liver abscess in a tertiary hospital in Bangladesh. *Bangladesh J Gastroenterol Liver.* 2020;35(2):123–128.
• Book:
Sherlock S, Dooley J. Diseases of the Liver and Biliary System. 11th ed. Blackwell Science; 2002.
• Cite Referencess within the text using superscript numbers after the punctuation mark.
• In the References list, list all authors when there are six or fewer; when there are seven or more authors, list the first six and add "et al."
• No of Referencess for review and original articles should be at least 15, but not more than 30. For case reports it should be at least 10, but not more than 20.

Ethical Considerations

Ethical Approval:
Research involving human subjects or animals must have been approved by an appropriate institutional review board (IRB) or ethics committee. The name of the approving body and the approval number should be stated in the Methods section of the manuscript.

Informed Consent:
Studies involving human subjects must have obtained informed consent from the participants. A statement confirming that informed consent was obtained should be included in the methods section.

Conflicts of Interest: Must be disclosed by all authors.

Plagiarism:
The journal has a strict policy against plagiarism. All submitted manuscripts may be screened for plagiarism using appropriate software. Manuscripts found to contain plagiarized material will be rejected.

Authorship:
• All authors must have made substantial contributions to the work and must agree to be accountable for the content of the manuscript. Authorship credit should be based on: (a) substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work; AND (b) drafting the work or revising it critically for important intellectual content; AND (c) final approval of the version to be published; AND (d) agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.
• Participation solely in the acquisition of funding, the collection of data, or general supervision of the research group does not qualify for authorship. Any changes to the author list after the initial submission (including additions, deletions, or changes in the order of authors) must be approved in writing by all authors. The corresponding author must provide a clear explanation for any such alterations to the Editor. All individuals who contributed to the work but do not meet the criteria for authorship should be listed in the Acknowledgements section.

Peer Review
All submitted manuscripts are subject to peer review by experts in the field. The journal uses a double-blind peer review process, meaning that the identities of the authors and reviewers are concealed from each other. The editorial board reserves the right to make editorial changes as necessary.

Manuscript Acceptance and Publication
If a manuscript is accepted for publication, the corresponding author will be notified. The accepted manuscript may be subject to further editing for clarity, grammar, and style.

Contact Information
For any queries or submission, please contact:

Editor-in-Chief
Prof. Dr. Dewan Saifuddin Ahmed, FCPS (Medicine)
MD, Gastroenterology, Professor and Chairman,
Dept. of Gastroenterology, Bangladesh Medical University.

Bangladesh Journal of Gastrointestinal and Liver Diseases
Email: bgsdigestivediseasechronicle@gmail.com
Website: https://bgs-bd.com

Editorial

The Transformative Role of Artificial Intelligence in Gastroenterology

Mushtaque Ahmad Rana

Artificial Intelligence (AI) is emerging as a transformative force in healthcare, and the field of gastroenterology is no exception. It is worthwhile for every gastroenterologist to understand the basics of what it does, how to use it, and what to watch for. Gastroenterology is a complex and demanding field that requires a high level of skills and expertise from physicians. Gastroenterologists also encounter many challenges in their daily work, such as increasing workload, administrative burden, regulatory compliance, and rising costs. One way to address these challenges and improve gastroenterologists' operational efficiency and well-being is to use the power of artificial intelligence.

The development of AI, which began in the 1950s with programs simulating human cognition, has accelerated dramatically. The 1980s introduced machine learning (ML), followed by deep learning in the 2010s, which uses complex artificial neural networks to imitate the human brain. A landmark moment occurred in 2022 with the launch of ChatGPT, bringing generative AI and large language models (LLMs) to the forefront.

In medicine, AI's impact is most profound in two areas: image recognition and big data analysis. AI can reach the desired output within seconds and with more consistent performance. Doctors may have inconsistent performance due to insufficient training or exhaustion from busy clinical demands. A visual assessment by a physician is qualitative, subjective, and prone to error, and subject to intra-observer and inter-observer variability. AI may have better performance than physicians in some cases and it has great promise to reduce clinician workload and the cost of medical care.

Image recognition AI softwares have the capacity to compensate for human error and improve the efficiency and quality of upper endoscopies and colonoscopies. Image recognition can fall into a category of computer aided detection (CADe) or computer aided diagnosis (CADx). CADe can help with identification and localization of an abnormality and CADx can help distinguish between diagnoses.

In the esophagus and stomach, identifying premalignant conditions and subtle changes of malignancy can be assisted by CADe and CADx. Identifying possible areas of Barrett's esophagus is of utmost importance for endoscopists as early detection is associated with decreased mortality from esophageal adenocarcinoma. A meta-analysis from 2021 showed that AI systems have a high accuracy, of up to 90%, in detection of all upper GI neoplasias including gastric cancers.[1]

One of the most well studied areas in AI endoscopy is the use of CADe in adenoma detection during colonoscopies. Adenoma detection rate (ADR) is one of the most validated indicators of colonoscopy quality, and increased ADR is associated with decreased interval colorectal cancers. In addition to CADe and CADx, colonoscopy may incorporate AI for monitoring withdrawal time, another important indicator of colonoscopy quality.

Another area of potential benefit in endoscopic management of GI pathologies is in assessment of GI bleeds (GIB). ML models may be used to predict rebleeding risk, success of potential interventions, and mortality in GIB with a greater accuracy than existing clinical risk stratification tools.[2]

Wireless video capsule endoscopy (VCE) allows for less invasive intraluminal image capture than traditional endoscopy. However, image analysis in VCE is time consuming and operator dependent, which can result in missed lesions or pathologies. Use of AI is being studied to improve the detection of lesions, bleeding, and other pathologies.

The role of AI in advanced endoscopy, specifically endoscopic ultrasound (EUS) and endoscopic retrograde cholangiopancreatography (ERCP) is linked to a need to diagnose malignant and premalignant pancreaticobiliary lesions. Pancreatic cancer has been difficult to diagnose at an early stage, and for this reason, mortality has increased by 53% over the past 25 years. Sometimes pancreatic cancer arises from a pancreatic cyst. Pancreatic cysts are often initially identified by CT or MRI. Once a high-risk pancreatic cyst is identified, usually defined by above 2 cm, growth of 5 mm in 1 year, dilated pancreatic duct, mural nodules, etc., the patient is usually referred to gastroenterology for an endoscopic ultrasound for further characterization and potential sampling through fine needle aspiration or biopsy. Detection of high-risk pancreatic cysts is critical in preventing the progression to pancreatic cancer.AI assisted EUS in premalignant pancreatic cysts has been evaluated as a potential answer to the limitations of CT, MRI, and traditional EUS in differentiating between benign lesions, high risk intraductal papillary mucinous neoplasms (IPMNs) and malignancy.3

AI is increasingly used to predict disease development, outcomes, and treatment responses. For gastric cancer, deep learning models have demonstrated higher accuracy than the traditional TNM staging system in predicting the likelihood of metastasis. In inflammatory bowel disease (IBD), AI analyzes large datasets to identify genetic risk factors. ML models can predict IBD flares by analyzing data on hospitalizations, steroid use, and biologic initiations, sometimes outperforming traditional biomarkers like fecal calprotectin. This highlights the potential cost advantage of using existing EMR data. In hepatology, ML models like random forest and artificial neural networks are being applied to predict outcomes for liver transplantation, showing performance comparable to or better than traditional statistical models like MELD in predicting post-transplant survival.[3]

AI can also augment gastroenterologists' roles in education and communication. Virtual assistants can provide patients with pre- and post-procedure information and guidance. Furthermore, AI can help create interactive educational content—such as case studies, simulations, and quizzes—for trainees and practicing gastroenterologists, fostering lifelong learning and skill enhancement.[4]

AI has the potential to transform the practice of gastroenterology, but it also faces some challenges and limitations. The first concern is Data quality and availability. AI relies on large and diverse datasets to learn and perform its tasks, but the data in gastroenterology may be incomplete, inconsistent or inaccurate, which can affect the reliability and validity of the AI outputs.The second is Ethical and legal issues.AI raises some ethical and legal issues in gastroenterology, such as privacy, consent, accountability and liability. For example, how to protect the privacy and security of patients' data and ensure their informed consent for the use of AI in their care?[4]

AI is not meant to replace human gastroenterologists but to augment and assist them in their work. However, there may be some challenges and barriers to effective and efficient human-AI collaboration in gastroenterology, such as trust, acceptance and adoption. For example, how to build and maintain the trust and confidence of gastroenterologists and patients in the AI systems and their outputs? How to ensure the acceptance and adoption of AI solutions by gastroenterologists and patients and overcome resistance and skepticism? How to optimize the workflow and interaction between humans and AI and ensure their complementarity and synergy? These are some of the issues that need to be addressed and resolved by the human-AI collaboration models and strategies in gastroenterology.[4]

Finally, as AI becomes integrated into care, the value of human interaction—empathy, critical thinking, and the physician-patient relationship—remains irreplaceable. AI should be a tool that enables physicians to provide better care, not a substitute for their judgment.

Mushtaque Ahmad Rana
Professor and Head, Department of Gastroenterology and Hepatology, Bangladesh Medical College.

Corresponding Author:
Mushtaque Ahmad Rana
Room No: 408, Bangladesh Medical College,
House 34, Road 14, Dhanmondi R/A,
Dhaka-1209, Bangladesh
E-mail: ranamushtaque2@yahoo.com,
Cell: 01552352039

Referencess:

1. Arribas J et al. Standalone performance of artificial intelligence for upper GI neoplasia: A meta-analysis. *Gut.* 2021;70(8):1458-1468

2. Shung D et al. Machine learning to predict outcomes in patients with acute gastrointestinal bleeding: A systematic review. *Digestive Diseases and Sciences.* 2019;64(8):2078-2087

3. Sood N, Chirayath S, Bahirwani J, Patel H, Kim E, Reddy-Patel N, et al. Applications of Artificial Intelligence in Gastroenterology and Hepatology [Internet]. Artificial Intelligence. *IntechOpen;* 2024. Available from: http://dx.doi.org/10.5772/intechopen.115047

4. Kaushal N. How Artificial Intelligence Can Enhance Operational Efficiency and Prevent Physician Burnout in Gastroenterology. [Internet].2025. [cited 2025 Nov 7].
Available from: https://www.asge.org/home/resources/key-resources/-blog/view/practical-solutions/2024/07/25/how-artificial-intelligence-can-enhance-operational-efficiency-and-prevent-physician-burnout-in-gastroenterology

Original Article

Efficacy of Rifaximin in Ulcerative Colitis Patient with Mild to Moderate Flare Up: A Randomized Controlled Trial

N Tabassum,[1] S Islam,[2] SA Mohiuddin,[3] CK Ghosh,[4] R Alam,[5] MA Rahman[6]

Abstract

Background: Gut microbiota is now being considered an important factor in promoting and maintaining inflammation in inflammatory bowel disease as well as in ulcerative colitis. Enteric infection is a common cause of microbial dysbiosis and is frequently found in ulcerative colitis patients. Traditional antibiotics use may induce untoward effect during long term use. Rifaximin, a rifampicin derivative is virtually unabsorbed after oral administration and does not cause serious systemic side effects. The potential therapeutic activity of Rifaximin in ulcerative colitis patients during mild to moderate flare up is not determined clearly still now.

Objectives: To assess efficacy of Rifaximin in ulcerative colitis patients with mild to moderate flare up.

Materials & Methods: This open label randomized controlled trial was conducted among 100 ulcerative colitis patients with mild to moderate disease. Intervention group received Rifaximin 550 mg twice daily along with 2.4 gm/d Mesalamine for 28 days. Control group received maximum dose of mesalamine 4.8 gm/ day for 28 days. All the patients were assessed by Partial Mayo Score along with CBC with ESR, CRP, Stool R/E, C/S, S. Albumin, Fecal Calprotectin at baseline and after 28 days.

Results: In patients with mild disease activity 56.9% of patients were in Rifaximin group and 43.1% were in Mesalamine group. In patients with moderate disease activity 53.1% were in Rifaximin with Mesalamine group and 46.1% were in Masalamine group. After 28 days of intervention in Rifaximin with Mesalamine group 54.5% patients achieve remission with a statistically significant difference over Mesalamine group (33.1%), 43.6% patients had mild disease activity, 1.8% patients had moderate disease activity, none of the patients had severe disease whether 20% patient from Mesalamine group developed severe disease after intervention. Side effects including hair fall and constipation were more in control group.

Conclusion: Patient response to Rifaximin in ulcerative colitis with mild to moderate flare up appears to be favorable in this randomized controlled trial. To establish its efficacy longer follow up is warranted.

Keywords: Gut microbiota, Ulcerative colitis, Partial Mayo Score, Rifaximin.

Authors:
Nowrin Tabassum, Registrar, Department of Gastroenterology, SSMC Mitford Hospital, Email : niturmc49@gmail.com

Sumona Islam, Resident, SUNY Upstate Medical University. Email: sumona.islam.borsha@gmail.com

Syed Arafat Mohiuddin, Assistant Professor, Chattagram International Medical College, Email: arafat116@gmail.com

Chanchal Kumar Ghosh, Professor, Department of Gastroenterology, BMU. Email: dr.chanchalghosh@gmail.com

Md. Razibul Alam, Professor, Department of Gastroenterology, BMU. Email: razibulalam73@gmail.com

Md. Ashiqur Rahman, Assistant Professor, National Gastroliver Institute & Hospital, Email: a.r.papel.cmc@gmail.com

Corresponding Author:
Nowrin Tabassum, Registrar, Department of Gastroenterology, SSMC Mitford Hospital, Email : niturmc49@gmail.com
Mobile: 01722706217.

Introduction:

Inflammatory bowel disease (IBD) is characterized by repetitive episodes of inflammation of GIT caused by an abnormal immune response to gut microflora in a susceptible individual. Ulcerative colitis (UC) affects the rectum mostly, but it may involve whole colon up to caecum usually in a continuous fashion. Crohn's disease (CD) result in trans mural ulceration of any portion of GIT most often affecting the ileo-colonic region.[1]

Although most IBD occurs in people aged 15 to 30 years, up to 25% of patients will develop IBD by adolescence. There may be a bimodal distribution with a 2nd peak of 10% to 15% developing IBD after age 60 years.[2]

The etiology of IBD still remains obscure. Genetic, immunological, environmental and psychological factors all play a role in the pathophysiology of IBD. This immunological activity causes release of inflammatory mediators which not only serve to amplify the immune and inflammatory response, but they also have direct effects on epithelial function and on repair mechanisms, thus increasing collagen synthesis.[3] A breakdown in the qualitative balance between protective and harmful bacteria proposed as potential mechanism.[4] Microbial dysbiosis in ulcerative colitis patients may result in increase in inflammatory cytokine levels and mucosal permeability may contribute to more intestinal wall damage.[5] In IBD patient luminal bacteria shows decrease in beneficial bacteria and increase in pathogenic bacteria.[6]

Various meta-analysis has demonstrated that antibiotics such as metronidazole, ciprofloxacin, clofazimine and antibiotic combination can be successfully employed in IBD including Ulcerative Colitis.[7]

Rifaximin, α-polymer, a rifampicin derivative, is a locally acting antibacterial agent that is unabsorbed after oral administration, is mostly exerted as unchanged drug in the stools in the course of intestinal disorders, and thus devoid of systemic side effect.

5-Aminosalicylic acid derivatives have a variety of anti-inflammatory effects, considered as mainstay of treatment during mild to moderate flare up and during remission .5 - ASA derivatives reduce fecal concentration of sulfide. Therefore, some bacteria harbor in intestinal mucosa and damage the protective structure. Antibiotics may result in positive outcomes by destroying the pathogenic bacteria.

Materials & Methods

This randomized controlled clinical trial was conducted among patients of both sexes aged more than 18 years attending IBD clinic, inpatient and outpatient of Gastroenterology department, BSMMU in between January 2022 to June 2023, who (n=100) met the selection criteria including

mild to moderate flare up of ulcerative colitis using Truelove and Witts criteria initially enrolled for the study.

All of them were previously diagnosed as Ulcerative Colitis by compatible history, examination, biochemical, endoscopic and histological findings. Sampling was done by convenient and judgmental sampling. Study population was allocated into two groups: Mesalamine (n=45) and Rifaximin group with Mesalamine group (n=55) by randomization. Randomization was done by lottery. All the patients of both groups were assessed by their clinical history, examination and some biochemical parameters eg: CBC with ESR, CRP, Stool R/E, C/S, S. albumin, Fecal calprotectin. They were assessed by Partial Mayo Scoring Index of ulcerative colitis at baseline. All of them were maintained with Mesalamine 2.4gm/day. There was no blinding in drug distribution. After giving consent Mesalamine group received maximum dose of Mesalamine up to 4.8 g/day for four weeks for induction of remission. Rifaximin with Mesalamine group received Rifaximin 550mg twice daily for 4 weeks in addition to maintenance dose of Mesalamine (2.4 gm/day) for induction. Throughout the study they were advised to continue their usual dietary practice. Within these four weeks they did not receive any other medication. Adverse drug reactions were documented. No serious adverse event was not noticed during study period. After four weeks they were assessed by Partial Mayo Scoring Index of ulcerative colitis. Some biochemical tests were done at the end of the therapy eg: CBC with ESR, CRP, S. Albumin and data were recorded in data collection sheet. If the condition deteriorated then he was excluded from the study.

Result

Among study population mean age of Rifaximin with Mesalamine group was 32.78 years and mean age of Mesalamine group was 35.96 years. Left sided colitis was more prevalent both in Rifaximin with Mesalamine group (49.1%) & Mesalamine group (50.9%). In Rifaximin with Mesalamine group pouchitis was present in four patients but no one in Mesalamine group. At the time of enrollment, disease severity was assessed by Truelove and Witts criteria of ulcerative colitis. In intervention group 56.9% had mild disease and 53.1% had moderate disease activity. Disease severity assessed by Partial Mayo Scoring index of ulcerative colitis (mild and moderate) at baseline showed no statistically significant difference between two groups. Hb%, CRP, S. albumin & Fecal calprotectin at baseline between two groups showed no statistical difference (Table I). But after 28 days of treatment significant improvement was noticed in intervention group.

Table I: Disease severity of UC patients at baseline

Variables	Intervention group Rifaximin with Mesalamine group (55)	Control group Mesalamine group (45)	P value
Partial Mayo score (after 28 days)			
Remission	30 (54.5%)	15 (33.3%)	**0.001^{S}**
Mild	24 (43.6%)	12 (26.7%)	
Moderate	1 (1.8%)	9 (20%)	
Severe	0 (0%)	9 (20%)	

When subgroups of Partial Mayo Scoring index of UC between two groups were observed it showed that patient's improvement was more marked in Rifaximin group in terms of stool frequency. In Rifaximin with Mesalamine group no patient developed stool frequency more than five times than normal whereas 13.3% patients developed stool frequency more than 5 times than normal in Mesalamine group. After 28 days of intervention only serum albumin exhibits statistically significant P value between two groups (Table II).

Table II: Distribution of the participants according to components of Partial Mayo scoring index of UC after intervention.

Variables	Intervention group Rifaximin with Mesalamine group (55)	Control group Mesalamine group (45)
1-2 stools more than normal	37 (67.3%)	3 (6.7%)
3-4 stools more than normal	18 (32.7%)	36 (80%)
5 or more stools more than normal	0	6 (13.3%)
Rectal Bleeding		
No blood seen	11 (20%)	2 (4.4%)
Streaks of blood	23 (41.8%)	9 (20%)
Obvious blood with stool	20 (36.4%)	30 (66.7%)
Blood alone	1 (1.8%)	
Physicians global assessment		
Normal	11 (20%)	1 (2.2%)
Mild disease	25 (45.5%)	21 (46.7%)
Moderate disease	19 (34.5%)	18 (40%)
Severe disease	0	5 (11.1%)

Among adverse drug reaction, constipation and hair fall were reported in both groups. Patient who developed hair fall during invervention, among them 75% belong to Mesalamine group whereas 25% were in Rifaximin with Mesalamine group. Constipation was also more prevalent in Mesalamine group.

Discussion

Given the chronicity of the disease, it is always important to explore effective therapeutic options with less side effects. Recent guidelines recommend using maximum dose of Mesalamine in mild to moderate ulcerative colitis. Though safety profile of Mesalamine is high, sometimes it carries serious dose dependent side effects eg: pancreatitis, bone marrow suppression, impaired liver function, interstitial nephritis.[8] So if we can use gut friendly antibiotic targeting intestinal dysbiosis in mild to moderate ulcerative colitis, it may create a new treatment strategy for ulcerative colitis patients.

In this trial, it was found that major portion of participants (40%) fell in the 26-35 age group, with an average age of 34.21 years. Regarding gender distribution, most of the participants were male (65%). Chowdhury et al. (2013) conducted a study in BSMMU, Dhaka showed similar demographic profile with mean age 34.14 years.[9] It was also a male predominant study. A study conducted by Lamet in 2011 had similar demographic characteristics as this study; with exception that it was a female predominant study.[10]

Regarding involvement of ulcerative colitis, in the Rifaximin with Mesalamine group, more patients were diagnosed as left sided colitis previously. Then pancolitis followed by proctitis and pouchitis were more prevalent. In Mesalamine group left sided colitis was more observed than proctitis and pancolitis. Previous study showed that proctitis and proctosigmoiditis were more prevalent and pancolitis was least prevalent in mesalamine group.[11] Variation may be related to geographical and environmental differences of study place. Further larger study is needed to explore the extent of involvement in ulcerative colitis in our country.

At baseline all the patients were assessed by Partial Mayo Scoring Index of Ulcerative Colitis. After 28 days of intervention two groups were assessed again with Partial Mayo Scoring index of ulcerative colitis. A significant (P<0.001) association was found in the Partial Mayo Score after 28 days of intervention, where Rifaximin with Mesalamine had more remission (54.5%) than Mesalamine (33.3%) group.

Antibiotics are not standard therapy, and their effects are still under investigation and a matter of debate. Previous studies showed a correlation between changes in the composition of the intestinal microbiota and IBD. At the moment current guidelines do not recommend use of antibiotics in IBD, except for the treatment of septic complication of crohns disease and pouchitis. A meta-analysis from 2012 included 11 randomized controlled trials, involving 832 crohns disease patient, treated with broad spectrum antibiotics, including Ciprofloxacin, Metronidazole, Combination, Rifaximin and others.[12] Treatment duration was variable between 2-16 weeks. Clinical improvement occurred in antibiotic group (56.1%) compared to placebo group (37.9%). Yuriko Nishikawa (2021) reported that combination of Amoxicillin, Fosfomycin and Metronidazole compared with Amoxicillin, Tetracycline and Metronidazole was more effective and safer in active ulcerative colitis .[13] Long term use of systemic antibiotics such as Metronidazole and Ciprofloxacin is associated with high number of adverse effects. Rifaximin is a rifamycin derived antibiotic that has a large antimicrobial coverage against Gram positive and Gram-negative bacteria including aerobes and anaerobes and poor absorption after oral administration and complete fecal excretion as unchanged drug. An open label pilot study conducted by Guslandi in 2004 showed statistically significant response with Rifaximin in ulcerative colitis flare patients.[14] But the study conducted by Goinchetti in 1999 showed no statistically significant difference between Rifaximin and placebo group in steroid refractory severe ulcerative colitis patients in their clinical outcome.[15] But there was more reduction of stool frequency in Rifaximin group over the placebo group. Variation of result may be due to disease severity as they conducted their study upon severe ulcerative colitis patients.

Rifaximin is a gut specific human pregnane X receptor (PXR) agonist.[16] PXR is a ligand activated transcription factor important drug transcription and metabolism. Recent data suggests PXR may play role in pathogenesis of IBD. Langmann et al. (2004) showed that gene expression analysis of tissue obtained from ulcerative colitis patients a significant reduction of PXR activity compared to normal intestinal tissue.[17]

Cheng et al. (2010) showed that preventive and therapeutic role of Rifaximin on IBD through human PXR mediated inhibition of NF-KB signaling cascade, suggesting that human PXR may be an effective target in treatment of IBD.[18]

In mild relapse of ulcerative colitis, aggressive treatment with immunosuppressive (Corticosteroid, azathioprine, biologic therapy) may be associated with higher rate of possible side effect.[19] In this study mean of Partial Mayo Score between two groups after intervention showed statistically significant difference; more reduction of score was observed in Rifaximin group. So, if Rifaximin is added with maintenance dose of Mesalamine may be a new approach in mild to moderate ulcerative colitis patients who have history of intolerance to high dose Mesalamine and may open a corticosteroid sparing regimen as patient intolerant to high dose Mesalamine are usually treated with corticosteroid. No laboratory parameters were found to be statistically significant; at baseline but after 28 days of treatment S. albumin showed significant P value between two groups (39.13±2.04 vs 37.24±5.94).

In this study, it was found that Rifaximin was able to decrease stool frequency to 1-2 times (67.3%); whereas Mesalamine was able to limit stool frequency to 3-4 times (80%) and >5 stools (13.3%). Tursi et al. (2010)found that Mesalamine also was unable to reduce bowel frequency significantly.[20] ASCEND II trial (2005) demonstrated that high dose Mesalamine (4.8 gm/d) is more effective (72%) than low dose (2.4 gm/d) Mesalamine (59%) during flare.[21]

By the physician's global assessment, Rifaximin was able to return the major portion of the participants to normal (20%) or mild disease status (45.5%). Moderately severe disease (40%) and severe disease (11.1%) were predominantly in Mesalamine group. Lorenzetti and Prantera (2013) reported several open label multicenter studies on Chrons disease and Ulcerative colitis where remission rate was 59-67% after 16-12 weeks of Rifaximin treatment.[22] Lichtenstein (2007) showed that treatment with Rifaximin 1600mg/dl had the highest clinical remission, clinical response and lowest rate of treatment failure than the placebo group.[23] Gawronska et al. (2017) found that Rifaximin group had the highest cure rate (78.6%).[24]

In this study hair fall and constipation were reported in both groups. But frequency of hair fall (75%) and constipation (42.60%) were prevalent in Mesalamine group. Sninsky et al. (1991) found that headache was the prevalent adverse reaction among the treatment group with Rifaximin.[25] But Muniyappa et al. (2009) found headache to be more frequent.[26]

Conclusion

Targeting microbial dysbiosis in ulcerative colitis patient Rifaximin has promising role in comparison to Mesalamine in ulcerative colitis patient with mild to moderate flare up. Moreover, high dose of Mesalamine sometimes can cause adverse events compared to Rifaximin. High dose and long term Rifaximin use has no negative impact on disease behavior, rather associated with significant improvement.

Conflict of Interest:

There is no conflict of interest of any authors in this study.

Acknowledgement:

We are grateful to the hospital authority and staffs of the department of Gastroenterology, Bangladesh Medical University for their kind co-operation.

References:

1. Maaser C, Sturm A, Vavricka SR, Kucharzik T, Fiorino G, Annese V, Calabrese E, Baumgart DC, Bettenworth D, Borralho Nunes P, Burisch J. *ECCO-ESGAR Guideline for Diagnostic Assessment in IBD Part 1:* Initial diagnosis, monitoring of known IBD, detection of complications. *Journal of Crohn's and Colitis.* 2019 Feb 1;13(2):144-164k.

2. Su HJ, Chiu YT, Chiu CT, Lin YC, Wang CY, Hsieh JY, Wei SC. Inflammatory bowel disease and its treatment in 2018: Global and Taiwanese status updates. *Journal of the Formosan Medical Association.* 2019 Jul 1;118(7):1083-1092.

3. Ardizzone S. Ulcerative colitis. Orphanet encyclopedia [Internet]. 2003

4. Tamboli CP, Neut C, Desreumaux P, Colombel JF. Dysbiosis in inflammatory bowel disease. Gut. 2004 Jan 1;53(1):1-4.

5. Shen ZH, Zhu CX, Quan YS, Yang ZY, Wu S, Luo WW, Tan B, Wang XY. Relationship between intestinal microbiota and ulcerative colitis: Mechanisms and clinical application of probiotics and fecal microbiota transplantation. *World journal of gastroenterology.* 2018 Jan 7;24(1)

6. Neut C, Bulois P, Desreumaux P, Membreé JM, Lederman E, Gambiez L, Cortot A, Quandalle P, Van Kruiningen H, Colombel JF. Changes in the bacterial flora of the neoterminal ileum after ileocolonic resection for Crohn's disease. *Official journal of the American College of Gastroenterology* ACG. 2002 Apr 1;97(4):939-946.

7. Rahimi R, Nikfar S, Rezaie A, Abdollahi M. A meta-analysis of antibiotic therapy for active ulcerative colitis. *Digestive diseases and sciences.* 2007 Nov;52(11):2920-2925.

8. Ford AC, Bernstein CN, Khan KJ, Abreu MT, Marshall JK, Talley NJ, Moayyedi P. Glucocorticosteroid therapy in inflammatory bowel disease: systematic review and meta-analysis. *Official journal of the American College of Gastroenterology* ACG. 2011 Apr 1;106(4):590-599.

9. Chowdhury MS, Khan MM, Rahman MZ, Saha M, Dhakal GP, Roy PK, Raihan AS. Clinical presentation of ulcerative colitis among Bangladeshi population twenty years experience from a tertiary care hospital in Bangladesh. *Bangabandhu Sheikh Mujib Medical University Journal.* 2013;6(1):16-20.

10. Lamet M. A multicenter, randomized study to evaluate the efficacy and safety of mesalamine suppositories 1 g at bedtime and 500 mg twice daily in patients with active mild-to-moderate ulcerative proctitis. *Digestive diseases and sciences.* 2011 Feb;56(2):513-522.

11. Rahimi R, Nikfar S, Rezaie A, Abdollahi M. Comparison of mesalazine and balsalazide in induction and maintenance of remission in patients with ulcerative colitis: a meta-analysis. *Digestive diseases and sciences.* 2009 Apr;54(4):712-721.

12. Vuyyuru SK, Solitano V, Narula N, Lee MJ, MacDonald JK, McCurdy JD, Singh S, Ma C, Jairath V. Pharmacological therapies for the management of fistulizing Crohn's disease: a systematic review and meta-analysis. *Journal of Crohn's and Colitis.* 2024 Apr 1;18(4):589-603.

13. Nishikawa Y, Sato N, Tsukinaga S, Uchiyama K, Koido S, Ishikawa D, Ohkusa T. Long-term outcomes of antibiotic combination therapy for ulcerative colitis. *Therapeutic Advances in Chronic Disease.* 2021 Jul;12:20406223211028790.

14. Guslandi, M., Giollo, P. and Testoni, P.A., 2004. Corticosteroid-sparing effect of rifaximin, a nonabsorbable oral antibiotic, in active ulcerative colitis: preliminary clinical experience. Current therapeutic research, 65(3), 292-296.

15. Gionchetti P, Rizzello F, Ferrieri A, Venturi A, Brignola C, Ferretti M, Peruzzo S, Miglioli M, Campieri M. Rifaximin in patients with moderate or severe ulcerative colitis refractory to steroid-treatment: a double-blind, placebo-controlled trial. *Digestive diseases and sciences.* 1999 Jun;44(6):1220-1221.

16. Ma X, Shah YM, Guo GL, Wang T, Krausz KW, Idle JR, Gonzalez FJ. Rifaximin is a gut-specific human pregnane X receptor activator. *The Journal of pharmacology and experimental therapeutics.* 2007 Jul 1;322(1):391-398.

17. Langmann T, Moehle C, Mauerer R, Scharl M, Liebisch G, Zahn A, Stremmel W, Schmitz G. Loss of detoxification in inflammatory bowel disease: dysregulation of pregnane X receptor target genes. *Gastroenterology.* 2004 Jul 1;127(1):26-40.

18. Cheng J, Shah YM, Ma X, Pang X, Tanaka T, Kodama T, Krausz KW, Gonzalez FJ. Therapeutic role of rifaximin in inflammatory bowel disease: clinical implication of human pregnane X receptor activation. *The Journal of pharmacology and experimental therapeutics.* 2010 Oct 1;335(1):32-41.

19. Ghosh CK, Islam S, Tabassum N, Mohiuddin SA, Hossain MM, Sarkar A, Bari A, Tabassum Jr N, Mohiuddin SA, Hossain MM, Sarker A. Quality of life of patients with inflammatory bowel disease in Bangladesh. *Cureus.* 2023 Jun 4;15(6).

20. Tursi A, Brandimarte G, Papa A, Giglio A, Elisei W, Giorgetti GM, Forti G, Morini S, Hassan C, Pistoia MA, Modeo ME. Treatment of relapsing mild-to-moderate ulcerative colitis with the probiotic VSL# 3 as adjunctive to a standard pharmaceutical treatment: a double-blind, randomized, placebo-controlled study. *Official journal of the American College of Gastroenterology* ACG. 2010 Oct 1;105(10):2218-2227.

21. Hanauer SB, Sandborn WJ, Kornbluth A, Katz S, Safdi M, Woogen S, Regalli G, Yeh C, Smith-Hall N, Ajayi F. Delayed-release oral mesalamine at 4.8 g/day (800 mg tablet) for the treatment of moderately active ulcerative colitis: the ASCEND II trial. *Official journal of the American College of Gastroenterology* ACG. 2005 Nov 1;100(11):2478-2485.

22. Lorenzetti R, Prantera C. Rationale for the use of rifaximin in inflammatory bowel diseases based on clinical trial results. *Clin Invest.* 2013;3(12):1187–93.

23. Lichtenstein GR. Rifaximin: recent advances in gastroenterology and hepatology. *Gastroenterology & Hepatology.* 2007 Jun;3(6):474.

24. Gawronska A, Banasiuk M, Lachowicz D, Pituch H, Albrecht P, Banaszkiewicz A. Metronidazole or rifaximin for treatment of Clostridium difficile in pediatric patients with inflammatory bowel disease: a randomized clinical trial. Inflammatory bowel diseases. 2017 Dec 1;23(12):2209-2214.

25. Sninsky CA, Cort DH, Shanahan F, Powers BJ, Sessions JT, Pruitt RE, Jacobs WH, Lo SK, Targan SR, Cerda JJ, Gremillion DE. Oral mesalamine (Asacol) for mildly to moderately active ulcerative colitis: a multicenter study. *Annals of internal medicine.* 1991 Sep 1;115(5):350-355.

26. Muniyappa P, Gulati R, Mohr F, Hupertz V. Use and safety of rifaximin in children with inflammatory bowel disease. *Journal of pediatric gastroenterology and nutrition.* 2009 Oct;49(4):400-404.

Original Article

Clinical, biochemical and utrasonographic presentation of Cirrhosis of liver in a tertiary care hospital

Saha M,[1] Chowdhury DP,[2] Aurko MS,[3] Islam ATMR,[4] Chowdhury TF,[5] Sumaiya FI,[6] Keya SD,[7] Sutradhar A[8]

Abstract

Objective: This study was designed to see the presentation and frequency of viral aetiology in chronic liver disease (CLD) in hospitalised patients.

Materials & Methods: Retrospective study included all hospitalised patients with CLD and decompensated cirrhosis. Data were retrieved from registrar and analysed.

Results: Total 185 cases of Chronic liver disease with or without decompensation were included. Age of varied from 15 years to 87 years with mean 55.43 and SD 13.13. Among them 107 (57.8%) and 78 (42.2%) were male and female respectively. About three-fourth of patients were above 45 years age group. Common presentations were ascites (118, 65%), abdominal pain (87, 47.0%), oedema (39, 21.1%) and encephalopathy (23, 12.4%). In this series 86 (46.5%), 08 (4.3%) and one (0.5%) had hepatitis B, hepatitis C infection and Wilson's disease respectively. In this series 45 (24.3%) patients had gall stone disease.

Conclusion: Common reasons for hospitalisation were ascites, abdominal pain, oedema and encephalopathy. Hepatitis B Virus infection was the common aetiology of CLD and cirrhosis.

Keywords: Chronic liver disease, Viral aetiology of cirrhosis, Gallstone disease.

Authors:
Dr. Madhusudan Saha, MBBS MD (Gastroenterology)
Professor and Head, Department of Gastroenterology
Sylhet Women's Medical College, Sylhet
Email: madhunibedita@gmail.com

Dr. Debangshu Paul Chowdhury, MBBS, Assistant Registrar
Gastroenterology, SWMC
Email: debangshupaul28@gmail.com

Aurko MS, 5th year Medical Student, Dhaka Medical College, Dhaka, aurko2024@gmail.com

Dr. A T M Redowanul Islam, MBBS, Medical Officer
Gastroenterology, Sylhet Women's Medical College, Sylhet
Email: redowanulislam96@gmail.com.

Dr. Tamanna Ferdous Chowdhury, MBBS, Medical Officer
Sylhet Women's Medical College, Sylhet
Email: tamanna20@gmail.com

Dr. Farzana Islam Sumaiya, MBBS, Medical Officer,
Gastroenterology, SWMC Sylhet Women's Medical College, Sylhet
email: sumaiyaislam8@gmail.com

Dr. Suprova Das Keya, MBBS, Medical Officer,
Gastroenterology, Sylhet Women's Medical College, Sylhet
Email: suprovadas94@gmail.com

Dr. Aporajita Sutradhar, MBBS, Medical Officer,
Gastroenterology, Sylhet Women's Medical College, Sylhet
Email: aporajitasutradhar7@gmail.com

Corresponding Author:
Dr. Madhusudan Saha, MBBS MD (Gastroenterology)
Professor and Head, Department of Gastroenterology
Sylhet Women's Medical College, Sylhet
Email: madhunibedita@gmail.com
Phone: +8801711367847

Introduction:

Chronic liver disease (CLD) is a disease causing high morbidity and mortality and leading to more than one million deaths per year.[1] Cirrhosis, the final common end stage condition of CLD irrespective of aetiology, is defined as loss of normal architecture, diffuse fibrosis and regenerative nodule formation.[2,3]

Prevalence of cirrhosis in the USA is 0.15% to 0.27%.[4] But worldwide prevalence is unknown. Cirrhosis of liver is the 11th leading cause of death in the world.[5] CLD related mortality in the world is increasing specially in low and low-middle income countries of Asia and Africa.[6] Alcohol and viral hepatitis are commonest causes worldwide.[7,8]

Aetiology of CLD and cirrhosis of liver in developed countries are alcohol abuse, HCV infection and non-alcoholic hepatitis while in developing countries hepatitis B (HBV) and hepatitis C (HCV) virus infection dominate the cause 4. Other causes of cirrhosis of liver are autoimmune hepatitis,[9] non-alcoholic liver disease and inherent metabolic disease.[10] Transition from hepatitis to cirrhosis occur in about 10-20% patients in 5-30 years[11]. In Bangladesh common causes of CLD are HBV (40.22%) followed by NASH (21.04%) and HCV (14.68%).[11]

Diagnosis of CLD and cirrhosis may be incidental in imaging. But it may be present with non-specific symptoms or with features of complications like ascites, spontaneous bacterial peritonitis, encephalopathy, hepatorenal syndrome, portopulmonary syndrome, variceal bleeding, jaundice and hepatocellular carcinoma.[12-14] Commonest presentations are ascites, encephalopathy, variceal bleeding and HCC.[13-16] Prevalence of gall stone disease in CLD is interestingly higher than general people and recent reports showed the global prevalence is about 29.4%.[17]

With this background this retrospective study was designed to see the presentation and aetiological contribution of viral infection of CLD and cirrhosis in a tertiary care hospital in Sylhet.

Materials & methods:

All patients admitted under department of Gastroenterology, Sylhet Women's Medical College Hospital from May 2022 to March 2025 with diagnosis of CLD were included in this study. Patients' epidemiological data, clinical and laboratory data were retrieved from the records. Approval from institutional review committee was taken.

Cirrhosis of liver was diagnosed from history, clinical signs like jaundice, ascites, oedema, splenomegaly, laboratory finding i.e. abnormality in ALT, AST, bilirubin, albumin, prothrombin time, viral markers and imaging features i.e., coarse, bright liver, shrunken liver, irregularity of margin, space occupying lesion, presence of ascites, and splenomegaly and oesophageal varices at endoscopic examination. Presence of cholelithiasis, evidence of biliary obstruction due to presence of gall stone, microlithiasis, sludge or history of cholecystectomy due to cholelithiasis were taken as evidence of gall stone disease. For aetiology Hepatitis B surface Antigen (HBSAg), Anti-HBc (total) in HBSAg negative patients and antigen against Hepatitis C virus (Anti-HCV) were done. Investigations for Autoimmune hepatitis, metabolic disease were performed in very limited cases.

Statistical analysis was done using SPSS 20 version. Mean, Range, mean and standard deviation were calculated for continuous data and percentage was calculated for categorical data. Chi-square test was done to see relations between variables.

Results:

Total 185 patients were included. Age of them varied from 15 years to 87 years (mean 55.43 and SD ± 13.134). Among them 107 (57.8%) and 78 (42.2%) were male and female respectively. About 75% of patients were above 45 years of age (Table-I). About three fourth of them were from rural area. Of them 75 (40.5%) were house wife and 48 (25.9%) were farmers. In this series 75 (40.5%) and 71(38.4%) were diabetic and hypertensive respectively. Of them 51 (27.6%), 20(2.3%) were smoker and betel leaves chewer respectively. While only one (0.5%) was alcoholic. In the study group 68(36.2%), 58(31.4%) and 35(18.9%) had history of jaundice blood transfusion and surgery respectively.

Table I. Distribution of patients according to demographic features (N=185)

Variables		Number (%)
Sex	Male	107 (57.8)
	female	78 (42.2)
Residence	Rural	135(73.0)
	Urban	50 (27.0)
Age	Up to 25 years	3 (1.6)
	26 to 45 years	44 (23.8)
	46 to 60 years	77 (41.6)
	Above	61 (33.0)
Education	Noinstitutional education	58 (31.4)
	Up to class five	47 (25.4)
	Class six to SSC	45 (24.3)
	Above	35 (18.9)
Occupation	Housewife	75 (40.5)
	Farmer	48 (25.9)
	Abroad	12 (6.5)
	Business	14 (7.6)
	Unemployed	8 (4.3)
	Day labourer	6 (3.2)
	Service	4 (2.2)
	others	18 (9.73)
Diabetes	Yes	75 (40.5)
Hypertension	Yes	71 (38.4)
Smoker	Yes	51 (27.6)
Tobacco chewer	Yes	55 (29.7)
Betel leaves and nut	Yes	130(70.3)
Alcohol	Yes	1 (0.5)
History of jaundice	Yes	68 (36.8)
History of blood transfusion	Yes	58 (31.4)
History of surgery	Yes	35 (18.9)

Most common presenting symptoms were abdominal distension (118;63.8%), abdominal pain (87;47%) and swelling of legs (39; 21.1%) (Table II).

Table II. Distribution of patients according to presenting symptom

Variables	Number (%)
Haematemesis	7 (3.8)
Melaena	9 4.9
Encephalopathy	23 (12.4)
Oedema	39 (21.1)
Fever	20 (10.8)
Vomiting	18 (9.7)
Abdominal distension	118 (63.8)
Weakness	17 (9.2)
Respiratory distress	13 (7.0)
Cons tipation	18 (9.7)
Pain abdomen	87 (47.0)
Low urine output	18 (9.7)
Jaundice	21 (11.4)

Of them 86(46.5%) had HBV surface antigen positive and 8 eight (4.3%) had antigen to Hepatitis C virus positive (Table 3). One patient aged 15 years was diagnosed as Wilson's disease. Biochemical examination revealed hypoalbuminaemia in 163 (88.1%). Mean corpuscular volume (MCV) was above 96 fl/lit was in 18 (9.7%) patients. Prothrombin time was up to 15 seconds ((control 12 seconds) were in 42 (22.7%). And ALT levels were within normal limit in 94 (50.8%) patients (Table III).

Table III. Biochemical, haematological and virological features.

Variables		Number (%)
S. albumin	Up to 2 gm/dl	20 (10.8)
	2.1 to 3.5 gm/dl	143 (77.3)
	3.51 and above	22 (11.9)
Prothrombin time	Up to 15.00 sec	42 (22.7)
	15.01 to 18.00 sec	48 (25.9
	Above 18.0 sec	95 (51.4)
Mean corpuscular volume	Up to 75 fl/l	23 (12.4)
	75.1 to 96 fl/l	144 (77.8)
	Above 96 fl/l	18 (9.7)
ALT level	Up to 40 iu/ dl	84 (50.8)
	41 to 80 iu / dl	52 (28.1)
	Above 80 iu/ dl	39 (21.1)
Viral marker	HBsAg positive	86 (46.5)
	HCV positive	8 (4.3)

Sonography revealed typical cirrhotic change in liver in 151(81.6%), heterogeneous echotexture in 13(7.0%) and fatty liver disease in 10(5.4%). Remaining patient with sonologically normal liver had positive viral markers and altered biochemical tests. In this series 20(10.8%) patients had space occupying lesion in liver, 150(81.1%) had ascites and 45(24.3%) had gall stone disease (11 patients underwent laparoscopic cholecystectomy). Three (1.6%) patients of this series had portal vein thrombus (Table IV).

Table IV. Findings at ultrasonographic imaging

Variables	Number (%)
Cirrhotic liver	151 (81.6)
Fatty liver disease	10 (5.4)
Normal	7 (3.8)
Hepatomegaly	4 (2.2)
Irregular heterogenous liver	13 (7.0)
Space occupying lesion	20 (10.8)
Ascites	150 (81.08)
Portal vein thrombus	3 (1.6)
Gall stone disease	45 (24.3)

Table V. Relation of HBV infection with age groups and sex.

Variables		HBV		P value
		Negative N(%)	Positive N(%)	
Age group	Up to 25 y (3)	1(33.3)	2(66.66)	0.005
	26 – 45 y (44)	16(36.36)	28(63.63)	
	46 - 60 y (77)	39.(50.65)	38(49.35)	
	Above 60 (61)	43(70.49)	18(29.51)	
Sex	Female(78)	53(67.95)	25(32.05)	0.001
	Male (107)	46(42.99)	61(5701)	

(Chi-square test was done).

Discussion

The age of patients of CLD & cirrhosis in our series was 15 to 87 years (mean 55.43) with higher incidence above 45 years (74%) which is higher than one report from our country and reports from central India and Eastern coastal India.[18-20] But it is similar to report from Northern India and Eastern India report from Pakistan.[21-23] Another report from central India found patients of CLD and cirrhosis were mostly above 40 years age group with median age 58.5.[24] This difference may be due to difference in population, study design and sample size. In our series males are predominantly affected which is consistent with previous reports from our country, India and Pakistan.[18-24]

In our series most of cases were from rural community which is consistent with report from Ethiopia and contradict with report from Pakistan.[23,25] Incidence of CLD and cirrhosis of liver varies from region to region. Cirrhosis was more common among patients of lower education level and poor economic status in our country from Nepal, India, Pakistan, Ethiopia and Europe.[19-23, 25, 26] Most common presentation of patients in our series was ascites with or without oedema followed by oedema and encephalopathy which is consistent with one report from our country from Nepal.[27-29]

Predominant etiology of CLD and cirrhosis in our series was HBV infection in nearly 46% patients which is consistent with report from our country[27, 28, 30] HCV infection was about 4% only. But report from Pakistan showed higher incidence of HCV infection.[23] But alcoholism is the most common cause in India and Nepal and Srilanka.[29,32,33] In our series further investigations for detection of other causes in majority of cases were not done.

Conclusion:

CLD is a common disease with morbidity and mortality leading to hospitalization. Most common cause of hospitalisaton are ascites, oedema, abdominal pain and encephalopathy. most of patients presented at age 45 years and above. Majority of patients were from rural community with lower economic background. Most common aetiology was HBV infection.

Conflict of Interest:

There is no conflict of interest of any authors in this study.

Acknowledgement:

We are grateful to the hospital authority and staffs of the department of Gastroenterology, Sylhet Women's Medical College Hospital for their kind co-operation.

References:

1. Nilsson E, Anderson H, Sargenti K, Lindgren S, Prytz H. Incidence, clinical presentation and mortality of liver cirrhosis in Southern Sweden: a 10-year population-based study. *Aliment Pharmacol Ther.* 2016 Jun;43(12):1330-1339. doi: 10.1111/apt.13635. Epub 2016 Apr 19. PMID: 27091240.

2. Anthony PP, Ishak KG, Nayak NC, Poulsen HE, Scheuer PJ, Sobin LH. The morphology of cirrhosis: definition, nomenclature, and classification. *Bull World Health Organ.* 1977;55(4):521-540. PMID: 304393; PMCID: PMC2366674.

3. Hytiroglou P, Snover DC, Alves V, Balabaud C, Bhathal PS, Bioulac-Sage P, et al. Beyond "cirrhosis": a proposal from the International Liver Pathology Study Group. *Am J Clin Pathol.* 2012 Jan;137(1):5-9. doi: 10.1309/AJCP2T2OHTAPBTMP. PMID: 22180471.

4. Sharma B, John S. Hepatic Cirrhosis. [Updated 2022 Oct 31]. In: StatPearls [Internet]. Treasure Island (FL): *StatPearls Publishing;* 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/-books/NBK482419/

5. WHO Global Health Estimates 2015: Death bycause, age, sex, by country *World Health Organisation 2016*

6. Mokdad, A.A., Lopez, A.D., Shahraz, S. et al. Liver cirrhosis mortality in 187 countries between 1980 and 2010: a systematic analysis. *BMC Med* 12, 145 (2014). https://-doi.org/10.1186/s12916-014-0145-y

7. Grant BF, Dufour MC, Harford TC. Epidemiology of alcoholic liver disease. *Semin Liver Dis.* 1988 Feb;8(1):12-25. doi: 10.1055/s-2008-1040525. PMID: 3283941.

8. Perz JF, Armstrong GL, Farrington LA, Hutin YJ, Bell BP. The contributions of hepatitis B virus and hepatitis C virus infections to cirrhosis and primary liver cancer worldwide. *J Hepatol*. 2006 Oct;45(4):529-538. doi: 10.1016/j.jhep.2006.05.013. Epub 2006 Jun 23. PMID: 16879891.

9. Selmi C, Bowlus CL, Gershwin ME, Coppel RL. Primary Biliary Cirrhosis, *Lancet* 2011; 377(9777): 1600-1609

10. Brunt EM, Wong VW, Nobili V, Day CP, Sookoian S, Maher JJ, et al. Nonalcoholic fatty liver disease. *Nat Rev Dis Primers*. 2015 Dec 17;1:15080. doi: 10.1038/nrdp.2015.80. PMID: 27188459.

11. Das DC, Mahtab MA , Rahim MA, Malakar D, Kabir A, Rahman S, Hepatitis B virus remains the leading cause of cirrhosis of liver in Bangladesh. *Bangladesh Med J.* 2016 Sept; 45(3): 164-166

12. Poordad FF. Presentation and complications associated with cirrhosis of the liver. *Curr Med Res Opin.* 2015 May;31(5):925-937. doi: 10.1185/03007995.2015.1021905. PMID: 25697811.

13. Hakan Yıldız, Meral Akdoğan, Nuretdin Suna, Erkin Öztaş, Ufuk B. Kuzu, Zülfükar Bilge, et al. Cirrhosis with ascites: Is the presence of hemorrhagic ascites an indicator of poor prognosis? *Turk J Gastroenterol* 2016; 27: 349-353, • DOI: 10.5152/tjg.2016.160042

14. Ellul MA, Gholkar SA, Cross TJ. Hepatic encephalopathy due to liver cirrhosis. *BMJ.* 2015 Aug 11;351:h4187. doi: 10.1136/bmj.h4187. PMID: 26265724.

15. Dy SM, Cromwell DM, Thuluvath PJ, Bass EB. Hospital experience and outcomes for esophageal variceal bleeding. *Int J Qual Health Care.* 2003 Apr;15(2):139-146. doi: 10.1093/intqhc/mzg016. PMID: 12705707.

16. Pinter M, Trauner M, Peck-Radosavljevic M, Sieghart W. Cancer and liver cirrhosis: implications on prognosis and management. *ESMO Open.* 2016 Mar 17;1(2):e000042. doi: 10.1136/esmoopen-2016-000042. PMID: 27843598; PMCID: PMC5070280.

17. Conte D, Barisani D, Mandelli C, Bodini P, Borzio M, Pistoso S, et al. Cholelithiasis in cirrhosis: analysis of 500 cases. *Am J Gastroenterol.* 1991 Nov;86(11):1629-1632. PMID: 1951241.

18. Afroz R, Deb SR, Kabir A, Red cell indices in chronic liver disease. *J MEDICINE* 2024; 25: 41-45

19. Mukherjee PS, Vishnubhatla S, Amarapurkar DN, Das K, Sood A, Chawla YK, et al.. Etiology and mode of presentation of chronic liver diseases in India: A multi centric study. *PLoS One.* 2017 Oct 26;12(10):e0187033. doi: 10.1371/journal.pone.0187033. PMID: 29073197; PMCID: PMC5658106.

20. Mishra D, Dash KR, Khatua C, Panigrahi S, Parida PK, Behera SK, et al. A Study on the Temporal Trends in the Etiology of Cirrhosis of Liver in Coastal Eastern Odisha. *Euroasian J Hepatogastroenterol.* 2020 Jan-Jun;10(1):1-6. doi: 10.5005/jp-journals-10018-1312. PMID: 32742964; PMCID: PMC7376596..

21. Goyal P, Goyal O, Kaur D, Chhina RS. Etiological Profile of Cirrhosis in a Tertiary Care Institute in Northern India. *J Gastrointest Infect* 2018;8(1):28-31.

22. Mehta KS, Sunny S, Naje Z, Priyadarshini K, Singh P. Trends of chronic liver disease in a Tertiary care centre in Eastern part of India: A retrospective study. *Cureus* 2023; 15(3): e 36662. PMCID: PMC5736952.

23. Salim A, Farooq MO, Saleem S, Malik K. Financial burden and social implications of chronic liver disease in a patient population group in Pakistan. *Pak J Med Sci.* 2024 Aug;40(7):1503-1508. doi: 10.12669/pjms.40.7.7976. PMID: 39092038; PMCID: PMC11255803.

24. Banait, S., Badole, S.M., Jain, J. Thorat A. Risk factors for chronic liver disease in population of Central India: a case-control study from rural India. *Egypt Liver Journal* 11, 10 (2021). https://-doi.org/10.1186/s43066-021-00077-9

25. Alemnew A, M., Admasu D, Y., Eshetie N, M, Negash W T, Berihun SA . Treatment outcomes of chronic liver disease and associated factors among patients treated at hospitals in Bahir Dar city, north-west Ethiopia. *BMC Gastroenterol* 25, 141 (2025). https://doi.org/10.1186/s12876-025-03719-z

26. Stratfolini T, Sagnelli E, Sagnelli C, Morisco F, Babudieri S, Furlan C. The association of between education level and chronic liver disease of any etiology. *European J of Internal Med* 2020; 722 75: 55-59.

27. Rahman S, Faroque Ahmed M, Jamshed Alam M, Debnath CR, Hoque MI, Hussain MM. Distribution of Liver Disease in Bangladesh: A Cross-country Study. *Euroasian J Hepatogastroenterol.* 2014 Jan-Jun;4(1):25-30. doi: 10.5005/jp-journals-10018-10092. Epub 2014 Jan 22. PMID: 29264315; PMCID: PMC5736952.

28. Hossain M1, Islam MA, Bodiuzzaman MM, Talukder MSI, Islam MMS, Raha A. Mode of presentation of Liver cirrhosis.-Faridpur Med. Coll. J. 2021;16(1):39-41 *Faridpur Med. Coll. J.* 2021;16(1):39-41

29. Poudel SC, Acharya A, Maharjan S, Saroj GC, Shrestha R, Thapa S, et al. Chronic Liver Disease among Patients Admitted in the Department of Internal Medicine of a Tertiary Care Centre: A Descriptive Cross-sectional Study. *JNMA J Nepal Med Assoc.* 2023 Mar 1;61(259):212-215. doi: 10.31729/jnma.8092. PMID: 37203940; PMCID: PMC10231553.

30. Das DC, Mahtab MA. Hepatitis B virus remains the leading causes cirrhosis live in Bangladesh. Bangladesh Medical J 2016; 45(3): 164-166

31. Swaroop S, Vaishnav M, Arora U, Biswas S, Aggarwal A, Sarkar S, et al. Etiological Spectrum of Cirrhosis in India: A Systematic Review and Meta-analysis. *J Clin Exp Hepatol.* 2024 Mar-Apr;14(2):101291. doi: 10.1016/j.jceh.2023.10.002. Epub 2023 Oct 9. PMID: 38544766; PMCID: PMC10964076.

32. Shrestha, A K. Shrestha, A, Shah, S, Bhandari, A Clinicodemographic profile of chronic liver disease patients at a tertiary care hospital: a retrospective analysis. *Annals of Medicine & Surgery* 85(3):p 399-402, March 2023. | DOI: 10.1097/MS9.0000000000000248

33. Wijewantha HS. Liver Disease in Sri Lanka. *Euroasian J Hepatogastroenterol.* 2017 Jan-Jun;7(1):78-81. doi: 10.5005/-jp-journals-10018-10217. Epub 2017 May 5. PMID: 29201778; PMCID: PMC5663780.

Original Article

Palliative Esophageal Stenting in Advanced Esophageal Carcinoma: Experience from 80 Cases in Northern Bangladesh

Md. Shakhawat Hossain[1], Maknunnahar[2], Md. Naushad Ali[3], Khondoker Monirul Alam[4], Jahan Afroza Lucky[5], Md. Ershadul Haque[6], Md. Shafiqul Islam[7], Moni Rani[8], Khandokar Md. Arif Hasnat[9], Md. Makhsudul Alam[10]

Abstract

Background: Esophageal carcinoma is a major health burden in Bangladesh, frequently diagnosed at advanced, inoperable stages. In such patients, palliation of malignant dysphagia is a key therapeutic goal. Self-expanding metal stents (SEMS) offer rapid relief, yet data from larger Bangladeshi cohorts remain limited.

Objective: To evaluate the clinical outcomes, complication profile, and survival following SEMS placement for palliation of inoperable esophageal carcinoma in Northern Bangladesh.

Materials & Methods: A retrospective analysis was conducted on 80 consecutive patients undergoing SEMS placement over a period of 05 years at selected endoscopy centers of northern Bangladesh. Data on demographics, tumor characteristics, pre- and post-stent dysphagia scores, complications, and survival were analyzed. Dysphagia was scored from 0 (normal diet) to 4 (complete dysphagia) at baseline, 1 week, and 1 month.

Results: The cohort comprised 48 men (60%) and 32 women (40%), mean age 62.5 years. Squamous cell carcinoma predominated (65%), with the lower esophagus most frequently affected (55%). Baseline mean dysphagia score was 3.2 ± 0.5, improving to 1.1 ± 0.7 at 1 week and 1.0 ± 0.8 at 1 month post-stent ($p < 0.001$); 90% achieved a score ≤ 1 by 1 month. Complications occurred in 28 patients (35%), including chest pain >48 h (20%), stent migration (8.75%), blockage (7.5%), and bleeding requiring intervention (3.75%). No procedure-related mortality was observed. Median survival was 192 days (range: 28–480), with 56.25% surviving beyond 6 months.

Conclusion: SEMS placement provides rapid, significant, and sustained relief of dysphagia in patients with inoperable esophageal carcinoma, with low immediate risk and manageable complications, offering tangible quality-of-life improvements within the limited survival period.

Keywords: Esophageal cancer, Self-expanding metal stent, Palliative care, Dysphagia, Bangladesh.

Authors:

Md. Shakhawat Hossain, Assistant Professor, Department of Gastroenterology, Rangpur Medical College, Rangpur
Email: shakhawat@rpmc.edu.bd;
ORCID ID: https://orcid.org/0000-0003-0624-4682

Maknunnahar, Ph.D Researcher Fellow, School of Science and Technology, Bangladesh Open University, Gazipur, Bangladesh.
Email: drauna89@gmail.com
ORCID ID: https://orcid.org/0000-0002-4999-4152

Md. Naushad Ali, Associate Professor, Department of Gastroenterology, Rangpur Medical College, Rangpur.
Email: dr.naushadali74@gmail.com

Khondoker Monirul Alam, Consultant (Medicine), Rangpur Medical College, Rangpur.
Email: russelrpmc35@gmail.com

Jahan Afroza Lucky, Assistant Professor, Department of Radiotherapy, Rangpur medical College, Rangpur.

Md. Ershadul Haque, Medical Officer, Department o Radiotherapy, Rangpur medical College, Rangpur.
Email: ershadulhaque757@gmail.com

Md. Shafiqul Islam, Radiotherapist, Department of Radiotherapy, Rangpur medical College, Rangpur.
Email: rshafiqul74@gmail.com

Moni Rani, Medical Officer, Department of Radiotherapy, Rangpur medical College, Rangpur.
Email: dr.monirani@gmail.com

Khandokar Md. Arif Hasnat, Medical Officer, Department of Radiotherapy, Rangpur medical College, Rangpur.
Email: inaba171@gmail.com

Md. Makhsudul Alam, Professor, Department of Gastroenterology, Rangpur Medical College, Rangpur.
Email: makhsudulalam@hotmail.com

Corresponding Author:
Md. Shakhawat Hossain, Assistant Professor, Department of Gastroenterology, Rangpur Medical College, Rangpur
Email: shakhawat@rpmc.edu.bd;
ORCID ID: https://orcid.org/0000-0003-0624-4682
Phone: +8801973223892

Introduction:

Esophageal carcinoma (EC) ranks as the 7th most common cancer globally (604,000 new cases/year) and the 6th leading cause of cancer mortality (544,000 deaths/year), with pronounced geographic disparities in incidence and histopathology.[1] In Bangladesh, EC represents a critical public health challenge, with age-standardized incidence rates of 8.4/100,000 in males and 5.1/100,000 in females which is significantly higher than global averages.[2] Northern Bangladesh reports particularly aggressive presentations, where >70% of patients are diagnosed at Stage III/IV due to limited screening infrastructure and delayed symptom recognition.[3] The histopathological dichotomy of EC is starkly region-dependent; a) Squamous cell carcinoma (SCC) dominates in Asia (80–95% of cases), linked to tobacco use, betel quid chewing, and nutritional deficiencies,[4] b) Adenocarcinoma prevails in Western populations (70–80%), associated with obesity and gastroesophageal reflux disease.[5]

In Bangladesh, SCC constitutes 65–80% of EC cases, with adenocarcinoma rising in urban cohorts.[6] For inoperable EC (60–70% of patients at diagnosis), malignant dysphagia causes devastating sequelae; i) 89% develop malnutrition (BMI <18.5 kg/m²),[7] ii) 45% suffer aspiration pneumonia,[8] iii) Quality-of-life scores plummet by 60–80% on EORTC QLQ-C30 scales.[9]

Self-expanding metal stents (SEMS) have emerged as a cornerstone for alleviating malignant dysphagia and improving quality of life. SEMS have established it's superiority after randomized trials established superiority over plastic stents and laser ablation. They have several benefits and privileges. Dysphagia relieves in 85–94% patients in contrast to radiotherapy where only 67-72% get it.[10] Another advantage is its rapid symptom relief within 24-48 hours after placement.[11] The most encouraging issue in resource limited countries is cost-effectiveness. SEMS demanding about $1,200-$1,800/stent in comparison to brachytherapy about $8,000-$12,000 per case.[12] In Bangladesh we have some specific challenges; like radiotherapy scarcity in cancer treatment. We have only 0.22 machines/million people vs. WHO-recommended 4/million.[13]

Others are low chemotherapy access i.e <20% receive systemic therapy due to cost/distance[14] and limitations of endoscopy machine and gastroenterologists (1.2/million in rural areas).[15] This retrospective study evaluates the clinical outcomes and complications of esophageal stenting in a larger cohort of patients with inoperable esophageal carcinoma in Northern Bangladesh.

Materials & Methods:

Retrospective data were collected from the endoscopy registries of Rangpur Medical College Hospital and three major private endoscopy centers in Northern Bangladesh. Records of 80 consecutive patients who underwent SEMS placement for palliation of inoperable esophageal carcinoma between January 2019 and December 2024 over 5 years were analyzed. Data extracted included demographic details, pre-procedural dysphagia score (scored 0-4: 0=normal diet, 1=some solids, 2=semi-solids, 3=liquids only, 4=complete dysphagia), tumor characteristics (location, histopathology), procedural details, post-stent complications, and survival duration. These data are usual follow up data after SEMS placement of those study centers. Dysphagia scores were assessed pre-stent and at 1-week and 1-month post-stent placement. Complications and survival data were also tracked until patient death or study conclusion as per intervention centers regulation. Data were analyzed by statistical software SPSS Version 15.0 (IBM Corp., Armonk, NY, USA). All continuous variables analyzed using Student's t-test, and categorical variables using Chi-square test. P-values of < 0.05 were considered as significant. Ethical permission was taken from the respective authority of Rangpur Medical college hospital.

Results:

In this study, out of 80 patients male were 48, (60%) and female were 32, (40%). Mean age were 62.5 years (Range: 38-88 years). Among presenting symptom dysphagia was universal (100%, mean pre-stent score: 3.2 ± 0.5). Significant weight loss (>10% body weight) was reported in 62 patients (77.5%). Other symptoms included regurgitation 35%, chest pain 28%. Location of tumor were distributed as lower esophagus 55%, mid 37.5%, upper 7.5%. Histopathology reveals Squamous cell carcinoma 65%, adenocarcinoma 35% (Table-I).

Table I: Distribution of patients according to baseline demographics and clinical characteristics (N=80)

Characteristic	Category	N=80(%)
Sex	Male	48 (60)
	Female	32 (40
Presenting symptoms	Dysphagia	80 (100)
	Weight loss >10% body weight	62 (77.5)
	Regurgitation	28(35)
	Chest pain	22(28)
Tumor location	Lower esophagus	44 (55)
	Mid esophagus	30 (37.5)
	Upper esophagus	6 (7.5)
Histopathology	Squamous cell carcinoma	52 (65)
	Adenocarcinoma	28 (35)

Note: Where only percentages were provided, counts are not shown.

Regarding dysphagia Relief, mean score improved to 1.1 at 1 week and 1.0 at 1 month ($p < 0.001$).By 1 month, 90% achieved score ≤1 (able to eat most solids/some solids). Median overall survival was 192 days (Range: 28-480 days). 45 patients (56.25%) survived for more than 6 months post-stent placement (Table II). Tumor stages at diagnosis and survival time were depicted at Figure 1 and 2.

Table II: Distribution of patients according to post stent outcomes and complications

Domain	Metric	Value	n (%)
Dysphagia relief	Mean score at 1 week	1.1 ± 0.7	—
	Mean score at 1 month	1.0 ± 0.8	—
Dysphagia relief	p-value (pre vs post)	<0.001	—
Dysphagia relief	Score ≤1 at 1 month	—	72(90)
Complications	Any complication	—	28(35)
	Chest pain (>48 h analgesics)	—	16(20)
	Stent migration	—	7(8.75)
	Stent blockage (food/ingrowth)	—	6(7.5)
	Persistent dysphagia (score ≥2 at 1 mo)	—	5(6.25)
	Bleeding (requiring intervention)	—	3(3.75)
	Stent misplacement	—	1(1.25)
	Procedure-related mortality	—	0
	Mean time to complication onset	54.3 ± 31.8 days	—
Survival	Median overall survival	192 days	—
	Survival range	28–480 days	—

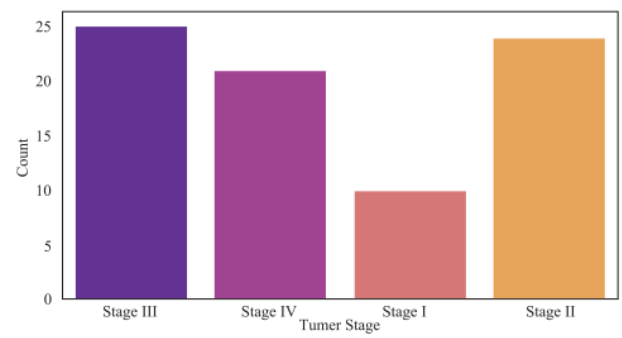

Figure-I: Distribution of Tumor Stages

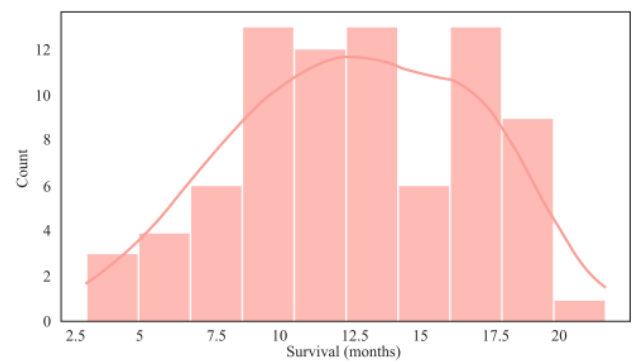

Figure-II: Distribution of Survival time of study populations

Below are correlation tables analyzing the relationship between demographic/tumor characteristics and key outcomes of esophageal stenting in 80 patients with inoperable esophageal carcinoma. Statistical significance was determined using chi-square tests (Fisher's exact test for small cell counts).

Table III: Distribution of patients according to factors associated with successful dysphagia relief.

(Success = Dysphagia Score ≤1 at 1 month post-stent; n=72/80, 90%)

Factor	Group	Success Rate	p-value
Age	<60 years (n=30)	93.3% (28/30)	0.42
	60–70 years (n=35)	88.6% (31/35)	
	>70 years (n=15)	86.7% (13/15)	
Sex	Male (n=48)	91.7% (44/48)	0.47
	Female (n=32)	87.5% (28/32)	
Tumor Location	Lower (n=44)	97.7% (43/44)	**<0.001**
	Mid (n=30)	90.0% (27/30)	
	Upper (n=6)	33.3% (2/6)	
Histopathology	SCC (n=52)	90.4% (47/52)	0.85
	Adenocarcinoma (n=28)	89.3% (25/28)	

Table IV: Distribution of patients according to factors associated with complications of esophageal stenting:

(Any complication; n=28/80, 35%)

Factor	Group	Complication Rate	p-value
Age	<60 years (n=30)	26.7% (8/30)	0.18
	60–70 years (n=35)	34.3% (12/35)	
	>70 years (n=15)	53.3% (8/15)	
Sex	Male (n=48)	33.3% (16/48)	0.60
	Female (n=32)	37.5% (12/32)	
Tumor Location	Lower (n=44)	22.7% (10/44)	**<0.001**
	Mid (n=30)	40.0% (12/30)	
	Upper (n=6)	100% (6/6)	
Histopathology	SCC (n=52)	36.5% (19/52)	0.62
	Adenocarcinoma (n=28)	32.1% (9/28)	

Table V: Distribution of patients according to Factors Associated with Survival >6 Months:

(n=45/80, 56.3%)

Factor	Group	>6-Month Survival	p-value
Age	<60 years (n=30)	73.3% (22/30)	**0.03**
	60–70 years (n=35)	51.4% (18/35)	
	>70 years (n=15)	33.3% (5/15)	
Sex	Male (n=48)	62.5% (30/48)	0.10
	Female (n=32)	46.9% (15/32)	
Tumor Location	Lower (n=44)	59.1% (26/44)	0.45
	Mid (n=30)	56.7% (17/30)	
	Upper (n=6)	33.3% (2/6)	
Histopathology	SCC (n=52)	51.9% (27/52)	0.28
	Adenocarcinoma (n=28)	64.3% (18/28)	

Discussions:

This multicenter retrospective analysis from Northern Bangladesh reinforces that self-expanding metal stents (SEMS) offer rapid, clinically meaningful relief of malignant dysphagia in patients with inoperable esophageal carcinoma, with an acceptable safety profile in a resource-constrained setting. The magnitude and speed of symptom improvement observed here-mean dysphagia score declining from 3.2 to 1.1 at 1 week and 1.0 at 1 month, with 90% achieving a score ≤1-mirrors the well-established efficacy of SEMS for swift palliation, which has consistently surpassed alternative modalities in terms of rapid swallowing restoration.16-18 The absence of procedure-related mortality and the manageable complication profile further align with contemporary guideline expectations for palliative stenting in advanced disease.[18-19]

Our cohort's immediate functional gains are congruent with prior studies reporting substantial short-term dysphagia relief after SEMS placement, typically within days and sustained at early follow-up.[16,17] The observed complication spectrum-chest pain (20%), migration (8.75%), stent blockage from food/ingrowth (7.5%), and bleeding requiring intervention (3.75%)-falls within the ranges reported across large series and guidelines, where chest pain is the most frequent early adverse event and migration and obstruction represent the leading causes of late dysfunction.18,20,21 The mean time to complication onset of approximately two months is consistent with the typical trajectory of late events such as overgrowth, ingrowth (for partially or uncovered designs), and food bolus impaction.[17]

Median overall survival of 192 days (~6.3 months) corresponds to survival expectations for patients selected for palliative stenting, who frequently harbor advanced locoregional disease and systemic spread at presentation. Multiple cohorts and systematic reviews have documented median survivals in the 3-7 month range following palliative SEMS, underscoring that the primary therapeutic aim is quality-of-life improvement rather than survival extension.15,16 In this context, the proportion surviving beyond six months (56%) suggests durable clinical benefit for a meaningful subset.

The predominance of squamous cell carcinoma (65%) and lower to mid-esophageal involvement mirrors regional epidemiology across South Asia, where SCC remains prevalent and often presents late with profound dysphagia and weight loss. (17-22) Adenocarcinoma comprised 35%, consistent with a growing global burden but still secondary to SCC in many Asian settings.

The proportion achieving near-normal swallowing by 1 month (≤1 in 90%) compares favorably with reports citing 70–90% early dysphagia response after covered SEMS insertion.[17,18] The statistical robustness of improvement ($p<0.001p<0.001$) is in line with the large effect sizes typically seen when transitioning from liquid-only diets to solids after stent deployment. Migration rates of 7–15% are commonly reported for covered SEMS, influenced by tumor location, stent design (anti-migration features, flares), and adjuvant therapies; our rate (8.75%) sits within that band.[17, 22] Obstruction from food or tumor tissue (7.5%) is also concordant with literature values of roughly 5–20% over follow-up, and bleeding requiring intervention typically ranges from 1–8%.[17,19] Notably, no procedure-related mortality was observed, reflecting the low peri-procedural risk reported in experienced centers.[19]

Although formal QoL instruments were not reported here, prior randomized and observational data show stenting rapidly improves swallowing-related QoL domains, while radiotherapy (external beam or brachytherapy) may offer superior longer-term dysphagia control at the expense of slower onset and different toxicity profiles.[16,17] In many low-resource contexts, SEMS provides an immediately impactful, pragmatic option.

The dominant early complaint of chest pain, often self-limited yet occasionally requiring analgesia beyond 48 hours, is well recognized and may reflect stent radial force, tumor stretch, and esophageal spasm.[17] Migration risk is heightened in short strictures, the lower esophagus, and fully covered stents; mitigation strategies include selecting appropriate length and diameter, ensuring adequate tumor overlap (typically ≥2 cm proximally and distally), and, where available, choosing designs with anti-migration features.[18] Stent obstruction is frequently preventable with dietary counseling-small bites, thorough chewing, avoidance of fibrous meats and sticky foods-and can be managed endoscopically by clearance, dilation for overgrowth, or stent-in-stent when needed.[17,19] The low bleeding rate here is reassuring; vigilance remains warranted in friable SCCs, anticoagulated patients, and when concomitant radiotherapy is used.[19]

Conclusion:

This larger retrospective analysis confirms that SEMS placement is a highly effective and safe palliative modality for managing malignant dysphagia in patients with inoperable esophageal carcinoma in Northern Bangladesh. It provides rapid and significant improvement in swallowing ability for the vast majority of patients, thereby enhancing quality of life. While complications occur in a significant minority (35%), they are generally manageable. The procedure carries a low immediate risk, making it a valuable option in resource-constrained settings. The survival data underscores the importance of effective palliation in this patient population with limited life expectancy.

Conflict of Interest:

There is no conflict of interest of any authors in this study.

Acknowledgement:

We are grateful to all endoscopy assistants and stent suppliers related to this study.

Referencess:

1. Sung H, Ferlay J, Siegel RL, Laversanne M, Soerjomataram I, Jemal A et al. Global cancer statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA Cancer J Clin.* 2021;71(3):209-249.

2. International Agency for Research on Cancer (IARC). Bangladesh. Globocan 2022 [Internet]. Lyon: IARC; 2022 [cited 2025 Jun 10]. Available from: https://gco.iarc.fr/today/data/factsheets/populations/50-bangladesh-fact-sheets.pdf

3. Biswas PK, Rahman MM, Sultana S, Hoque MM, Ali MS, Chowdhury MR, et al. Stage migration and survival deficit in Bangladeshi esophageal cancer patients. *Asian Pac J Cancer Prev.* 2022;23(8):2701-2707.

4. Abnet CC, Arnold M, Wei WQ. Epidemiology of esophageal squamous cell carcinoma. *Gastroenterology.* 2018;154(2):360-373.

5. Coleman HG, Xie SH, Lagergren J. The epidemiology of esophageal adenocarcinoma. *Lancet Gastroenterol Hepatol.* 2017;2(1):37-46.

6. Rahman MA, Hossain MS, Akter S, Kabir M, Karim MR, Alam MS, et al. Changing histopathological spectrum of esophageal cancer in Bangladesh. *J Gastroenterol Hepatol Res.* 2023;12(1):45-52.

7. Gupta V, Vashistha N, Jagati A, Kannan S, Garg H. Malnutrition in inoperable esophageal cancer: a neglected prognostic factor. *Clin Nutr* ESPEN. 2021;45:136-142.

8. Martin L, Lagergren P, Pilling M, Lagergren J. Risk of aspiration pneumonia after diagnosis of esophageal cancer in a population based cohort study. *Ann Surg Oncol.* 2020;27(8):2642-2651.

9. Lagergren P, Johar A, Ness Jensen E. Health related quality of life in esophageal cancer. *JAMA Surg.* 2023;158(3):e225341.

10. Spaander MCW, Baron TH, Siersema PD, Fuccio L, Schumacher B, Escorsell À. Esophageal stenting for benign and malignant disease: European Society of Gastrointestinal Endoscopy (ESGE) Guideline – update 2023. *Endoscopy.* 2023;55(5):523-542.

11. Homs MYV, Steyerberg EW, Eijkenboom WMH, Tilanus HW, Stalpers LJA, Bartelsman JFWM, et al. Single dose brachytherapy versus metal stent placement for the palliation of dysphagia from oesophageal cancer: multicentre randomised trial. *Lancet.* 2004;364(9444):1497-1504.

12. Yusuf TE, van Dijkum EJ, Bruno MJ, Fockens P. Cost effectiveness of palliative therapies for oesophageal cancer in low and middle income countries. *World J Gastroenterol.* 2021;27(36):6080-6091.

13. World Health Organization. Bangladesh cancer country profile 2024 [Internet]. Geneva: WHO; 2024 [cited 2025 Aug 19]. Available from: https://www.who.int/publications/m/item/cancer *BGD* 2024

14. Ahmed S, Islam MS, Hossain MI. Barriers to cancer therapy in Bangladesh. *Cancer Control.* 2022;29:1-10.

15. Bangladesh Gastroenterology Society. National endoscopy workforce survey 2024. Dhaka: *BGS Publications;* 2024.

16. Homs MY, Steyerberg EW, Eijkenboom WM. Single-dose brachytherapy versus metal stent placement for the palliation of dysphagia from oesophageal cancer: multicentre randomised trial. *Lancet.* 2004;364(9444):1497 1504. doi:10.1016/S0140-6736(04)17272-17273

17. Siersema PD. Palliative treatment of esophageal carcinoma. *Gastrointest Endosc Clin N Am.* 2010;20(1):37 52. doi:10.1016/j.giec.2009.08.010

18. Siersema PD. Esophageal stenting in clinical practice: an overview. *Nat Rev Gastroenterol Hepatol.* 2015;12(3):173-182. doi:10.1038/nrgastro.2015.3

19. Spaander MCW, Baron TH, Siersema PD. Esophageal stenting for benign and malignant disease: European Society of Gastrointestinal Endoscopy (ESGE) Clinical Guideline. *Endoscopy.* 2021;53(7):751 762. doi:10.1055/a 1401-2901

20. ASGE Technology Committee; Adler DG, Baron TH. Enteral stents. *Gastrointest Endosc.* 2013;78(3):391-403. doi:10.1016/-j.gie.2013.04.197

21. Vakil N, Morris AI. Self-expanding metallic stents for palliation of malignant dysphagia: impact on quality of life. *Gastrointest Endosc.* 1998;47(6):532-538. doi:10.1016/S0016-5107(98)70246-1

22. Siewert JR, Ott K. Are squamous and adenocarcinomas of the esophagus the same disease? *Semin Radiat* Oncol. 2001;11(1):1-12. doi:10.1053/srao.2001.20711

Original Article

Post-ERCP pancreatitis in the Gastroenterology department of BMU

Md. Tasmir Arefin,[1] Dewan Saifuddin Ahmed,[2] Md. Masudur Rahman Khan,[3] Md. Razibul Alam,[4] Chanchal Kumar Ghosh,[5] Mohammad Shoaib Chowdhury,[6] Md. Zahidur Rahman,[7] A.B.M. Safiullah,[8] Mohammad Asadur Rahman,[9]

Abstract

Background: Endoscopic retrograde cholangiopancreatography (ERCP) is an advanced endoscopic procedure used to treat pancreaticobiliary diseases. However, it poses significant risks, with post-ERCP pancreatitis (PEP) being the most common consequence, occurring in about 10.2% of cases.

Objective: To assess the frequency and severity of post-ERCP pancreatitis.

Materials & Methods: This prospective observational study was conducted among consecutive patients who underwent ERCP in the Department of Gastroenterology at Bangladesh Medical University (BMU), Dhaka, from December 2023 to February 2025.

Results: A total of 272 participants were evaluated. The average age was 51.2 years, with 47.1% of participants male and 52.9% female. The most common indications of ERCP were choledocholithiasis (49.6%), cholangiocarcinoma (12.1%) and papillary stenosis (10.7%). Overall, 8.8% of participants developed post-ERCP pancreatitis (PEP), 7% had mild PEP, and 1.8% had moderate PEP. No participant developed any severe PEP.

Authors:

Md. Tasmir Arefin, MBBS, MD (Gastroenterology)
Department of Gastroenterology, Bangladesh Medical University.
Email: arefintasmir@gmail.com

Dewan Saifuddin Ahmed, MBBS, FCPS (Medicine), MD Gastroenterology) Professor and Chairman, Department of Gastroenterology, Bangladesh Medical University.
Email: saifgastro@gmail.com

Md. Masudur Rahman Khan, MBBS, MD (Gastroenterology)
Professor, Department of Gastroenterology, Bangladesh Medical University.
Email: masudgastro64@gmail.com

Md. Razibul Alam, MBBS, MD (Gastroenterology)
Professor, Department of Gastroenterology, Bangladesh Medical University.
Email: Razibulalam73@gmail.com

Chanchal Kumar Ghosh, MBBS, FCPS (Medicine), MD (Gastroenterology) Professor, Department of Gastroenterology, Bangladesh Medical University, Email: dr.chanchalghosh@gmail.com

Mohammad Shoaib Chowdhury, MBBS, MD (Gastroenterology)
Associate Professor, Department of Gastroenterology, Bangladesh Medical University, Email: drshoaibchowdhury@gmail.com

Md. Zahidur Rahman, MBBS, MD (Gastroenterology)
Associate Professor, Department of Gastroenterology, Bangladesh Medical University, Email: zahidgastro@gmail.com

A.B.M. Safiullah, MBBS, MD (Gastroenterology)
Associate Professor, Department of Gastroenterology, Bangladesh Medical University, Email: gastrosafi67@gmail.com

Mohammad Asadur Rahman, MBBS, MD (Gastroenterology)
Associate Professor, Department of Gastroenterology, Bangladesh Medical University, Email: dr.asad74@gmail.com

Affiliations:
Department of Gastroenterology, Bangladesh Medical University, Shahbag, Dhaka, Bangladesh

Corresponding Author:
Md. Tasmir Arefin, MBBS, MD (Gastroenterology),
Department of Gastroenterology, Bangladesh Medical University, Shahbag, Dhaka, Bangladesh.
Phone: +8801683740271

Introduction:

ERCP is an endoscopic procedure performed with a side-view scope. Since its introduction in 1968, ERCP has become an important procedure for identifying and treating numerous pancreaticobiliary diseases.

Due to advancements in alternative imaging modalities, such as magnetic resonance imaging and/or MRCP and EUS, ERCP has changed from a diagnostic procedure to a therapeutic one, mainly for stone clearance, stricture dilation, and placing stents to relieve blockage in the pancreatic and bile ducts. Common adverse events of ERCP are pancreatitis, cholangitis, cholecystitis, bleeding, and perforation. Post-ERCP pancreatitis is the most common major complication of ERCP.[1]

PEP is clinical evidence of pancreatitis following ERCP, with a three-fold increase in serum amylase after 24 hours, necessitating hospitalization or an extended hospital stay.[2] After that, Freeman added new or deteriorating abdominal pain as a new characteristic to define PEP and suggested allowing lipase as a substitute for amylase.[3] According to the European Society of Gastrointestinal Endoscopy guideline 2020, PEP is characterised by new or deteriorating abdominal pain accompanied by raised pancreatic enzymes (lipase or amylase greater than three times the normal level after 24 hours) and extending a scheduled hospital stay or necessitating hospitalization following ERCP.[4]

The pathophysiology of PEP is a complex concept that is not fully understood. This includes a variety of insults caused by papillary instrumentation, hydrostatic injury from the overfilling of the pancreatic duct with contrast material, and mechanical, chemical, thermal, enzymatic, allergic, and microbiological factors. These factors lead to a series of events that cause premature intracellular activation of pancreatic proteolytic enzymes, autodigestion, and the creation of inflammatory cytokines that have an impact on the body both locally and systemically.[5]

The severity of PEP is classified as mild, moderate, and severe. According to "cotton criteria", Mild, moderate and severe post-ERCP pancreatitis are defined as requiring hospitalization for 2-3, 4-10 and >10 days, respectively, after ERCP or the development of any complication (necrosis or pseudocyst) or requirement for any intervention (surgery or drainage).2

Methods

This prospective observational study was conducted in the Department of Gastroenterology, Bangladesh Medical University (BMU), Dhaka, during the period from December 2023 to February 2025. Patients who met standard indications for ERCP were included in the study. Exclusion criteria were pancreatitis before ERCP.

A total of 291 patients underwent ERCP in the department of gastroenterology, during the study period. 19 patients were excluded due to pancreatitis before ERCP. 272 participants were finally enrolled in the study.

Abdominal pain 24 hours following ERCP and a three-fold or higher increase in serum amylase or lipase were considered indicators of pancreatitis. The severity of pancreatitis was defined based on the number of hospitalized days following ERCP as mild (<4 days), moderate (4 to 10 days), or severe (>10 days).[6]

Before performing ERCP, baseline serum amylase and lipase levels were measured in the patients who presented with abdominal pain clinically consistent with acute pancreatitis. All the participants obtained standard hydration and preprocedural rectal indomethacin. After ERCP, participants were assessed clinically for new or worsened abdominal pain compatible with acute pancreatitis. Serum lipase and amylase levels were measured 24 hours after ERCP. The number of days required to stay at the hospital after ERCP was also recorded.

Results:

Most participants were between 55 and 64 years (25.37%), and females (52.9%) were predominant over males (47.1%). Table I presents the demographic distribution (by age and gender) of the participants.

Table I: Age and Gender distribution of the participants (n=272)

Variables	Number of participants (n)	Percentage (%)
Age (Mean±SD)	51.2±7.8	
Age groups(years)		
<25	8	2.94
25-34	26	9.56
35-44	61	22.42
45-54	57	20.96
55-64	69	25.37
>64	51	18.75
Gender		
Male	128	47.1
Female	144	52.9

Table II: Indications of ERCP among the participants (n=272)

Indications	Number of participants (n)	Percentage (%)
Choledocholithiasis	135	49.63
Cholangiocarcinoma	33	12.13
Papillary stenosis	29	10.66
Benign biliary stricture	24	8.82
Ampullary carcinoma	23	8.46
Carcinoma gallbladder with biliary infiltration	13	4.78
Carcinoma head of the Pancreas	10	3.68
Biliary ascariasis	5	1.83
Total	**272**	**100**

Table II shows, the most common indication of ERCP was choledocholithiasis (49.63%).

Table III: Distribution of participants based on the development of post-ERCP pancreatitis.

Post -ERCP pancreatitis	Number of participants (n)	Percentage (%)
Yes	24	8.8
No	248	91.2
Total	**272**	**100**

Table III shows that among all the participants, 8.8% (24 out of 272) developed post-ERCP pancreatitis

Table IV: Distribution of participants according to the severity of post-ERCP pancreatitis (n=272)

Severity of post-ERCP pancreatitis	Number of participants (n)	Percentage (%)
Mild	19	7
Moderate	5	1.8
Severe	0	0
Total	**24**	**8.8**

Table IV shows the severity of post-ERCP pancreatitis among all the participants. As a whole, 7% (19 out of 272) had mild and 1.8% (05 out of 272) had moderate PEP. No participant developed any severe PEP.

Table V: Age, Gender and indication-based distribution of participants developing post-ERCP pancreatitis (n=24)

Age groups (years)	n (%)	Gender	n (%)	Indication	n (%)
<25	2 (25%)	Male	9 (7%)	Choledocholithiasis	9 (6.7%)
25 -34	7 (27%)	Female	15 (10.4%)	Cholangiocarcinoma	6 (18.2%)
35-44	4 (6.6%)			Papillary stenosis	4 (13.8%)
45-54	3 (5.3%)			Benign biliary stricture	1 (4.2%)
55-64	5 (7.2%)			Ampullary carcinoma	2 (8.7%)
>64	3 (5.9%)			Carcinoma gallbladder with biliary infiltration	2 (15.4%)

Table V shows that the rate of developing PEP was higher in patients between 25-34 years of age, female participants, and in patients with cholangiocarcinoma.

Discussion:

ERCP is one of the most technically demanding and high-risk procedures. The most common complication of ERCP is acute pancreatitis, which is a major cause of morbidity and mortality. A comprehensive review of one hundred eight RCTs from 1977 to 2012, including 13,296 patients, revealed that the rate of PEP was 9.7%. PEP was 5.7% for mild, 2.6% for moderate, and .5% for severe. Two thousand three hundred forty-five high-risk participants revealed PEP as 14.7%. Mild, moderate, severe PEP and PEP-related mortality were 8.6%, 3.9%, .8%, and .2%, respectively. In North American RCTs, the rate of PEP was 13%, but in European and Asian RCTs, it was 8.4% and 9.9%, respectively.[7]

In 2017, a study was performed in the BMU, including 95 participants. The mean age was 49.74 years. 58.9% patients underwent ERCP due to choledecholithiasis. Post-ERCP pancreatitis occurred in 9.4% patients.[8]

In this research, most participants (25.4%) were in the age group of 55-64 years. Gender distribution was not much different, with 47.1% males and 52.9% females. Nearly half of the participants underwent ERCP due to choledocholithiasis, 135 out of 272; 49.6%. Other indications were cholangiocarcinoma (12.13%), papillary stenosis (10.66%), benign biliary stricture (8.82%), ampullary carcinoma (8.46%), carcinoma gallbladder with biliary infiltration (4.78%), carcinoma head of the Pancreas (3.68%), biliary ascariasis (1.83%). Post-ERCP pancreatitis developed in 19 out of 24, 8.8% of participants overall. Severity analysis revealed that 19 out of 24, 7% had mild, and 05 out of 24, 1.8% had moderate PEP. No participant developed any severe PEP. The majority of people experiencing PEP were under 35 years of age, female, and cholangiocarcinoma was the primary indication.

Conclusion:

In comparison to other endoscopic procedures, ERCP is still linked to a very high rate of complications, especially post-ERCP pancreatitis, even with advancements in technique and equipment. Most of these issues fall into the mild to moderate severity category.

Conflict of Interest:

There is no conflict of interest of any authors in this study.

Acknowledgements:

We would like to extend our heartfelt gratitude to the study participants.

References:

1. Chandrasekhara V, Khashab MA, Muthusamy VR, Acosta RD, Agrawal D, Bruining DH, Eloubeidi MA, Fanelli RD, Faulx AL, Gurudu SR, Kothari S. Adverse events associated with ERCP. *Gastrointestinal endoscopy.* 2017 Jan 1;85(1):32-47.

2. Cotton PB, Lehman G, Vennes J, Geenen JE, Russell RC, Meyers WC, Liguory C, Nickl N. Endoscopic sphincterotomy complications and their management: an attempt at consensus. *Gastrointestinal endoscopy.* 1991 May 1;37(3):383-393.

3. Freeman ML, Nelson DB. Complications of endoscopic biliary sphincterotomy. *New England Journal of Medicine.* 1997 Mar 27;336(13):963-964.

4. Dumonceau JM, Kapral C, Aabakken L, Papanikolaou IS, Tringali A, Vanbiervliet G, Beyna T, Dinis-Ribeiro M, Hritz I, Mariani A, Paspatis G. ERCP-related adverse events: European Society of Gastrointestinal Endoscopy (ESGE) guideline. *Endoscopy.* 2020 Feb;52(02):127-149.

5. Freeman ML. Adverse outcomes of ERCP. *Gastrointestinal Endoscopy.* 2002 Dec 1;56(6):S273-282.

6. Alavinejad P, Tran NP, Eslami O, Shaarawy OE, Hormati A, Seiedian SS, Parsi A, Ahmed MH, Behl NS, Abravesh AA, Tran QT. Oral N-Acetyl cysteine versus rectal indomethacin for prevention of post ERCP pancreatitis: a multicenter multinational randomized controlled trial. *Arquivos de gastroenterologia*. 2022 Nov 14;59:508-512.

7. Kochar B, Akshintala VS, Afghani E, Elmunzer BJ, Kim KJ, Lennon AM, Khashab MA, Kalloo AN, Singh VK. Incidence, severity, and mortality of post-ERCP pancreatitis: a systematic review by using randomized, controlled trials. *Gastrointestinal Endoscopy.* 2015 Jan 1;81(1):143-149.

8. Habib MR, Ahmed F, Gain G, Hasan R, Ishaque SM, Ahmed DS. Patterns of post-endoscopic retrograde cholangiopancreatography (ERCP) complications. *Bangladesh Medical Journal.* 2021;50(2):35-39.

Original Article

Video Capsule Endoscopy To Detect Small Bowel Lesions- Experience From A Single Centre

Dr. Md. Musab Khalil,[1] Dr. Subrata Podder Chayan,[2] Dr. Md. Moshfiqur Rahman Chowdhury,[3] Dr. Md. Yasir Arafat,[4] Dr. Abu Sayeed,[5] Dr. Imteaz Ahmed Dipon,[6]

Abstract

Background: Capsule endoscopy has been a ground breaking tool since its inception to diagnose small bowel lesions. Video capsule endoscopy stared operating in Bangladesh few years back. The objective of the study is to find out the indications, findings and diagnostic yields of different indications used in video capsule endoscopy.

Patients and Methods: This cross sectional study was conducted at the inpatient department of medical Gastroenterology of National Gastroliver institute and hospital, Mohakhali, Dhaka from January 2024 till September 2025.

Results: Out of 26 patients, 23 patients had undergone video capsule endoscpy for evaluation of small bowel bleeding. Rest of them were for suspected small bowel Crohns' disease. The diagnostic yields of small bowel bleeding could be detected in 47.8% of the cases and evidence of Chrohn's disease was seen in 60% of the cases. One patient had capsule retention due to presence of stricture which was removed surgically.

Conclusions: Video capsule endoscopy is a safe and effective procedure to detect small bowel pathology. Suspected Small bowel bleeding is the most common indication.

Key Words:
Small Bowel Ulcers, Small Bowel Bleeding, Capsule Retention

Authors:
Dr. Md. Musab Khalil, Medical officer
National Gastroliver institute and Hospital, Mohakhali, Dhaka

Dr. Subrata Podder Chayan, Junior consultant
Upazilla Health Complex, Sonaimuri,Noakahali

Dr. Md. Moshfiqur Rahman Chowdhury, Assistant Professor
National Gastroliver institute and Hospital, Mohakhali, Dhaka

Dr. Md. Yasir Arafat, Assistant Professor
National Gastroliver institute and Hospital, Mohakhali, Dhaka

Dr. Abu Sayeed, Medical Officer
National Gastroliver institute and Hospital, Mohakahli, Dhakar.

Dr. Imteaz Ahmed Dipon, Assistant Professor
National Gastroliver institute and Hospital, Mohakhali, Dhaka

Corresponding Author:
Dr. Md. Musab Khalil, Medical officer
National Gastroliver institute and Hospital, Mohakhali, Dhaka
Email: evandmc@gmail.com, Mobile: +8801818462121

Introduction:

Capsule Endoscopy is a noninvasive technology to diagnose small bowel diseases. Capsule endoscopy was first introduced in mid 1990s by Gavriel Iddan.[1] Since its inception, video capsule has revolutionized small bowel imaging by providing complete visualization of small bowel mucosal surfaces.[2] Capsule endoscopy is the investigation of choice for detecting small bowel disorders such as suspected small bowel bleeding, suspected Crohn's disease, celiac disease, polyposis syndromes etc.[3] The diagnostic yield, compared to other technologies, varies from 38-87 % depending on the indications.[2] Suboptimal visualization of mucosa, inability to acquire tissue and inability to provide therapeutic benefit as well as the cost of the capsule, have been the major limitation of video capsule endoscopy. Despite the limitation, video capsule endoscopy has provided superior efficacy in comparison to small bowel follow through and comparable efficacy to device assisted enteroscopy to evaluate small bowel disorders. Video capsule endoscopy has been introduced in Bangladesh recently. However, there is no published data so far regarding the video capsule endoscopies. Therefore, we are going to publish our study regarding the findings of capsule endoscopy.

Materials and Methods:

This cross sectional study was conducted at the inpatient department of medical Gastroenterology of National Gastroliver institute and hospital, Mohakhali, Dhaka from January 2024 till September 2025. Upto twenty six video capsule endscopy data has been collected. Pillcam VCE was used to perform VCEs.

All adult patients, who were willing to participate in the study and who underwent VCE since 2024 January was included in the study. Before performing VCE , we have done CT enterography or MR enterography. If the patient had clinical features of small bowel obstruction; ie visible peristalsis; abdominal pain and vomiting along with constipation or if the patient had radiological presence of small bowel narrowing with proximal dilatation, were excluded from the study.

Patients were given 500 ml of mannitol solution along with 500 ml of plain water, in split doses for bowel clearance. After ensuring adequate bowel clearance, capsule was allowed to be ingested by the patient. Patient was kept under observation till the next morning for ensuring evacuation of the capsule. Clear fluid was allowed two hours after the capsule ingestion while light meal was allowed 4 hours after capsule ingestion.

Capsule was performed by three endoscopists who have experience in performing enteroscopies. CE findings were labeled as per international Delphi consensus on nomenclature and description of SB vascular lesions.[4] The vascular lesions have beed described according to Saurin classification and Yano- Yamamoto vascular lesion classification.[5]

All data were entered in standardized format in spreadsheets using Microsoft excel. Continuous variables were expressed as mean and standard deviation or median and interquartile range wherever appropriate. Categorical variables were expressed as a percentage. Categorical variables were compared using Chisquare test or Fisher exact test wherever appropriate.

Continuous variables were compared using student's t test or Mann- Whitney tests wherever appropriate. $P < 0.05$ was considered as statistically significant. The SPSS version 25 (IBM Corp., Armonk, New York, United States) was used for statistical analysis.

Results:

We analyzed 26 patients data who had underwent Video Capsule Endoscopy (VCE). Median age of the participants was 57.50 (SD ± 18.261 years). Four of the patients were smokers. The major indications were suspected small bowel bleeding (23 patients) while rest of the patients had VCE due to suspected Crohn's disease. The diagnostic yield for small bowel bleeding was 47.82% while the diagnostic yield for crohns' disease was 60%. Among the 26 VCE examinations, 16 (61.5%) had lesions in their examinations while no lesion could be found amongst the rest. Among the lesions, small bowel ulcers were present in 25% patients. Vascular ectasias were present in 18.8%. Erosions were present in 12.5 % patients as well.

Table I: Locations of VCE lesions

VCE Lesion Location	Number of Patients (n)
Duodenum	4
Jejunum	12
Ileum	11

Table II: VCE Findings

VCE Findings	Frequency	Percentage (%)
Erosions	2	7.7
Ulcerations	4	15.4
Nodularities	1	3.8
Stricture	1	3.8
Vascular Ectasia	3	11.5
Ulcerated Strictures	1	3.8
Others	3	11.5

Discussion:

Capsule endoscopy has become an essential investigation to detect small bowel diseases. It isa a relatively safe and comfortable procedure having few contraindications namely bowel obstruction, strictures as well as fistulas.[6] Small bowel bleeding has been the most common indication for VCE worldwide that coincides with our indications for VCE as well.[3] The diagnostic yield for suspected small bowel bleeding of our study was 47.82% . study done at one of largest centres in India showed similar scenario as well.[3] The capsule retention rate is 1% which is also consistent with other studies as well.[1] Video Capsule endoscopy has proven to be an important diagnostic tool to detect Crohn's Disease as well. It has been superior in diagnosing suspected small bowel crohns disease than other diagnostic modalities such as imaging, enteroscopies, barium follow through as well.[7] The diagnostic yield for Crohns' disease varies from 52% to 71% which are comparable to our studies as well.[8]

The present study has some limitations. Sample size is small to bring into any completion. It is a single centre study. Also follow up of patients could not be done to reach a final diagnosis.

Conclusion:

High diagnostic yield, relatively safe and tolerable procedure have made Video capsule endoscopy is the procedure of choice for evaluating small bowel diseases. Yet, inability to capture biopsies is one of the major limitations to reach into a final diagnosis. Further samples are needed to improve the diagnostic yields of different small bowel lesions. Video capsule endoscopy will definitely help to diagnose seemingly undiagnosable small bowel diseases in future diagnosable and thereby will help to reduce patients sufferings and save lives.

Conflict of Interest:

There is no conflict of interest of any authors in this study.

Acknowledgements:

We would like to extend our heartfelt gratitude to the study participants.

References:

1. Song HJ, Shim KN. Current status and future perspectives of capsule endoscopy. Vol. 14, Intestinal Research. *Korean Association for the Study of Intestinal Diseases;* 2016. p. 21–29.

2. Pennazio M, Spada C, Eliakim R, Keuchel M, May A, Mulder CJ, et al. Small-bowel capsule endoscopy and device-assisted enteroscopy for diagnosis and treatment of small-bowel disorders: European Society of Gastrointestinal Endoscopy (ESGE) Clinical Guideline. Vol. 47, Endoscopy. *Georg Thieme Verlag;* 2015. p. 352–376.

3. Singla N, Inavolu P, Jagtap N, Singh AP, Kalapala R, Memon SF, et al. Small Bowel Capsule Endoscopy: Experience from a single large tertiary care centre. *Endosc Int Open.* 2023 Jun;11(06):E623–628.

4. Leenhardt R, Li C, Koulaouzidis A, Cavallaro F, Cholet F, Eliakim R, et al. Nomenclature and semantic description of vascular lesions in small bowel capsule endoscopy: an international Delphi consensus statement. *Endosc Int Open.* 2019 Mar;07(03):E372–379.

5. Yano T, Yamamoto H, Sunada K, Miyata T, Iwamoto M, Hayashi Y, et al. Endoscopic classification of vascular lesions of the small intestine (with videos). *Gastrointest Endosc* [Internet]. 2008 Jan 1 [cited 2025 Sep 20];67(1):169–172. Available from: https://www.sciencedirect.com/science/article/abs/pii/S0016510707024972

6. Goenka MK, Majumder S, Kumar S, Sethy PK, Goenka U. Single center experience of capsule endoscopy in patients with obscure gastrointestinal bleeding. *World J Gastroenterol.* 2011;17(6):774–778.

7. Li F, Gurudu SR, De Petris G, Sharma VK, Shiff AD, Heigh RI, et al. Retention of the capsule endoscope: a single-center experience of 1000 capsule endoscopy procedures. *Gastrointest Endosc* [Internet]. 2008 Jul 1 [cited 2025 Sep 21];68(1):174–180. Available from:https://www.sciencedirect.com/science/article/abs/pii/S0016510708002691

8. Hara AK, Leighton JA, Heigh RI, Sharma VK, Silva AC, De Petris G, et al. Crohn Disease of the Small Bowel: Preliminary Comparison among CT Enterography, Capsule Endoscopy, Small-Bowel Follow-through, and Ileoscopy1. https://doi.org/101148/radiol2381050296 [Internet]. 2006 Jan 1 [cited 2025 Sep 21];238(1):128–134. Available from: https://pubs.rsna.org/doi/10.1148/radiol.2381050296

Case Report

Unusual presentation of Sheehan's Syndrome with recurrent vomiting: A case report

Dr. Monisha Israt Jahan,[1] Dr. Shiplu Basak,[2] Dr. Mohammad Enamul Karim[3]

Abstract

Sheehan's syndrome is often a sequela of massive postpartum hemorrhage in resource-poor developing countries where blood loss during delivery is often neglected. The diagnosis of this rare yet fatal disease is often delayed because the symptoms are vague, insidious in nature and partial deficiencies are often difficult to determine. We report the case of a 30-year-old multiparous female with recurrent vomiting and severe hyponatremia. This report highlights the subtle manifestations of Sheehan's syndrome to help clinicians establish a prompt diagnosis which can play role in improving the quality of life of the patient while also saving her from impending adrenal crisis.

Keywords:
Sheehan's syndrome, Hormones, Hypopituitarism, Hypotension, Hypovolemic shock, Postpartum Hemorrhage, Adrenal insufficiency.

Authors:
Dr. Monisha Israt Jahan, MBBS, FCPS (Final Part), MRCP UK (Part 2) FCPS Trainee, Department of Medical Gastroenterology
National Gastroliver Institute and Hospital, Mohakhali, Dhaka

Dr. Shiplu Basak, MBBS, MD, Assistant Professor,
Department of Medical Gastroenterology
National Gastroliver Institute and Hospital, Mohakhali, Dhaka

Dr. Mohammad Enamul Karim, MBBS, FCPS, MD
Associate Professor, Department of Medical Gastroenterology
National Gastroliver Institute and Hospital, Mohakhali, Dhaka

Correspondence to:
Dr. Monisha Israt Jahan, MBBS, FCPS (Final Part), MRCP UK (Part 2) FCPS Trainee, Department of Medical Gastroenterology
National Gastroliver Institute and Hospital, Mohakhali, Dhaka
Email: monishaisratjahan@gmail.com, Phone: +8801799900536

Introduction:

Sheehan's syndrome is hypopituitarism due to postpartum ischemic necrosis of the pituitary gland. It was first described in 1937 by Sheehan.[1] It is reported that Sheehan's syndrome accounts for 0.5% of all known cases of hypopituitarism in females.[2] The disease is deemed "rare" in industrialized nations, but in developing nations, due to a lack of access to sophisticated medical procedures, skilled professionals, and medical resources, which contributes to a higher prevalence of postpartum hemorrhage and subsequent Sheehan's syndrome, it is reported to occur in 5 out of every 100,000 births.[3,4] The rate is much higher in developing countries, with a prevalence of 3.1% in a state in India where more than half of the affected individuals had home deliveries.[5] The underlying mechanism leading to Sheehan's syndrome is the infarction of the physiologically enlarged anterior pituitary lobe (due to hyperplasia of prolactin-secreting cells resulting from elevated estrogen secretion) and secondary to the compression of the blood vessels supplying the gland by the enlarged gland itself or due to grossly impaired blood supply during intrapartum or postpartum events, which is therefore highly vulnerable to ischemia in the setting of hypotension and hemorrhage during delivery. Apart from adenohypophysal necrosis, other etiologies are noted: autoimmunity, tumoral, immunological, iatrogenic, traumatic, infectious and genetic.[6] Pituitary dysfunction, which slowly progresses, is thought to be due to an inflammatory or autoimmune process triggered by released antigens during pituitary necrosis and autoantibodies have been detected in patients with Sheehan syndrome, but not consistently.[7] Sheehan's syndrome can present during the postpartum period or several months or even years following delivery. A study in France showed a delay of 9 ± 9.7 years in the diagnosis of Sheehan's syndrome,[8] and a longer delay of 20.37 ± 8.34 years was noted in developing countries.[9] Women with Sheehan's syndrome have varying degrees of hypopituitarism, ranging from panhypopituitarism to only selective anterior pituitary deficiencies even sometimes in posterior pituitary hormones deficiency, causing diabetes insipidus.[10,11] The most common initial symptoms are agalactia and/or amenorrhea. Uncommonly, it can present as an emergency condition like adrenal crisis, myxedema coma, hypoglycemia, and hyponatremia precipitating by an infection or surgery.[12] Differential diagnoses include pituitary adenoma and lymphocytic hypophysitis. There may be a long delay to diagnosis; even over a decade because symptoms are often vague and pituitary dysfunction progresses gradually. We describe a case of a patient with chronic presentation of Sheehan's syndrome 6 years after the obstetric event and with no clear precipitating event.

Case Presentation:

A 30-year-lady with recurrent vomiting, generalized fatigability and chronic severe hyponatremia of unclear origin sought care at our gastroenterology outdoor department. She was hospitalized for further evaluation and treatment. During her hospital stay, physical examination raised some concerns. The patient exhibited generalized skin pallor suggesting true hypopigmentation with mild anemia (Figure 1). Her skin appeared dry; she had fine wrinkling around the mouth and eyes and her hair was thin. Initial laboratory evaluations showed anemia, with a hemoglobin level of 10.30 g/dL, random blood sugar at 3.5 mmol/L and severe hyponatremia, with a level of 109 mmol/L and potassium level at 3.10 mmol/L. All of the following tests were within normal ranges or had negative results: basic metabolic panel, lipase level, creatinine level, liver function test, urinalysis and imaging like abdominal ultrasound, upper GI Endoscopy.

To evaluate recurrent hyponatremia, further tests were given which fulfilled the diagnostic criteria of SIADH with urinary sodium 76 mmol/L, blood osmolality 236.14 mOsmol/Kg, urine osmolality 573 mOsmol/Kg. She continued to have orthostatic symptoms even after fluid resuscitation. Given the absence of an alternative cause of her symptoms, additional endocrine testing was undertaken (Table 1). Her TSH was 1.27 mIU/L, and free triiodothyronine (T3) and free thyroxine (T4) levels were 1.12pg/ml and 0.24ng/dl respectively. A 9 am cortisol level was 8.73 mcg/dL, and adrenocorticotropic hormone (ACTH) level was 15.93 pg/ml necessitating for short synachthen test which confirmed a diagnosis of adrenal insufficiency (cortisol after 30 min and 60 min were 10.20 mcg/dl and 14.70 mcg/dl). Her prolactin level was inappropriately normal at 75.64 mcIU/L. Her level of Luteinizing hormone was low at 2.64 mIU/L with Follicular stimulating hormone at 8.93 mIU/L, indicating gonadotropin hormone deficiency. Magnetic resonance imaging of the brain was consistent with partial empty sella (Figure 2).

Further inquiries during her treatment revealed that she had experienced a severe postpartum hemorrhage following the delivery of her last child 6 years prior, with an estimated blood loss of 700 mL and required two units of blood transfusion. She had delivered at home, and bleeding subsided without intervention. After childbirth, she had difficulty in lactating but her menstrual cycle was regular. She was having OCP since her childbirth; that's why having withdrawal bleeding mimicking regular menstrual cycle and flow. This was actually masking her amenorrhea with altered picture of gonadotropin deficiency. She also reported symptoms of anhedonia and light-headedness. Over the years, her primary care practitioners had evaluated her condition by TSH levels, which had remained in the normal range.

The patient was diagnosed with Sheehan's syndrome, which was associated with PRL deficiency, gonadotropin deficiency, adrenal insufficiency and secondary hypothyroidism. The patient was rehydrated with normal saline (NS), and standard dosage of oral contraceptives, prednisolone and thyroxin were started following consultation with the endocrinology department. She was initially given hydrocortisone, 5 mg in the morning and 2.5 mg in the evening, followed by levothyroxine supplementation, 75 mcg daily started 3 days later. She was strictly instructed on the nature of her illness and to take these medications for the rest of her life. Her orthostatic symptoms got resolved and significant improvement was noted following the commencement of hormone replacement.

Discussion:

The diagnosis of Sheehan's syndrome is determined by the patient's history and physical examination, and later confirmed by laboratory tests. Failure to lactate is often a common initial complaint.[13] Many of them also report amenorrhea after delivery.[14] The diagnosis of Sheehan's syndrome is not made until several years later in certain cases, when the features of hypopituitarism gradually become apparent in a woman who had postpartum bleeding.[15] Patient presents with varied symptoms depending on the specific hormone deficiencies. Growth hormone deficiency causes fatigue, decreased quality of life, and weight loss. Prolactin deficiency can cause lactation failure. Gonadotropin deficiency will often cause amenorrhea, loss of libido or genital hair loss. Corticotrophin deficiency can result in generalized fatigue, weakness, hypoglycemia, dizziness or vomiting. Symptoms of secondary hypothyroidism are clinically similar to primary hypothyroidism while having low triiodothyronine and thyroxine levels, with low or even inappropriately normal thyroid-stimulating hormone levels. Diagnosis of panhypopituitarism is straightforward, but partial deficiencies are often difficult to determine.[16] Our patient developed chronic Sheehan's syndrome, which include clinical manifestations of growth hormone deficiency, prolactin deficiency, gonadotropin deficiency and partial involvement of adrenocorticotropic and thyroid stimulating hormone. The French study found that the delay in diagnosis in patients presenting with hypothyroidism was 8.1 ± 8.5 years and in those presenting with acute adrenal insufficiency was 10.6 ± 9.4 years.[8] In our patient, the first clue to her diagnosis was her lactational failure and postpartum haemorrhage, and the next clue was the manifestation of symptoms of adrenal insufficiency in subtle ways with fatigue and anorexia which progressed to dizziness, nausea, and recurrent vomiting, all of which were unfortunately missed as findings in making a diagnosis. This can be attributed to a lack of awareness, especially given that patients with panhypopituitarism present with nonspecific symptoms, coupled with a lack of a thorough history and physical examination required to diagnose a rare disease. Laboratory tests can reveal many other abnormalities, including hyponatremia. This is the most common electrolyte imbalance, occurring in 33–69% of cases.[17,18] As a presenting manifestation of Sheehan's syndrome, severe hyponatremia causing altered level of consciousness has rarely been described in the literature. This might be due to the slow evolution of the disease into its chronic form.[19] Punwell and colleagues found mild to severe hyponatremia in 9 of 13 patients with Sheehan's syndrome.[20] The pathology behind hyponatremia in Sheehan's syndrome is still open to debate. Cases of severe hyponatremia, with serum sodium levels below 125 mmol/L, developing 16 years after postpartum haemorrhage have been reported. SIADH may be responsible for hyponatremia in Sheehan's syndrome.[12,22] Inappropriate secretion of ADH is known to occur in states of adrenocorticotropin deficiency. Animal experiments and clinical observations suggest that glucocorticoids tonically inhibit the secretion of ADH. A sudden loss or decrease in the inhibitory control may lead to rapid serum elevations of ADH. Another potential mechanism for the elevation in ADH is the uncontrolled release of the hormone from the posterior hypophysis in the setting of ischemia.[23] In our patient's case, we found hypo-osmolar hyponatremia with euvolemia, and increased urinary sodium. Low serum osmolality and elevated urine osmolality suggested SIADH. There are several other possible mechanisms by which hypopituitarism can result in hyponatremia. Hypothyroidism can cause decreased free-water clearance and subsequently hyponatremia occurs. Glucocorticoid deficiency can also cause decreased free-water clearance, independent of ADH. The potassium level in these situations is normal, because adrenal production of aldosterone is independent of pituitary. In this case, the initial hypokalemia noted could be due to gastrointestinal loss following recurrent vomiting. The patient's sodium level was subsequently normalized with commencement of hydrocortisone, and potassium was corrected with KT syrup. Anemia is a well-recognized feature of hypopituitarism. Gokalp et al. recently reported hematological abnormalities in 65 patients with Sheehan's syndrome, 80% of whom presented with anemia, compared with 25% of controls.

Many hormonal deficiencies, including hypothyroidism, adrenal insufficiency, and gonadal hormonal deficiency, can explain normochromic anemia in hypopituitarism.[24] Dynamic pituitary MRI may reveal different features depending on the stage of the disease. While early scans are not usually helpful for diagnosis, they may demonstrate a non-hemorrhagic enlargement of the pituitary gland, leading to its subsequent involution, and late scans typically show an empty sella. A secondary empty sella is considered a classical finding of Sheehan's syndrome.[25] Treatment of young female with hypopituitarism usually includes replacement of hydrocortisone first and then replacement of thyroid-stimulating hormone and estrogen with or without progesterone, depending on having a uterus. Hydrocortisone is replaced first because thyroxin therapy can exacerbate glucocorticoid deficiency and induce life threatening adrenal crisis.[14,26]

Figure I: Facial features showing fine wrinkling, skin hypopigmentation.

The standard dose of hydrocortisone is 20 mg/day for an adult (15 mg every morning and 5 mg every evening). Both gonadotropin and thyroxin replacement are titrated to each individual. Replacement of growth hormone is necessary in children with hypopituitarism but controversial in adults. Some people with severe growth hormone deficiency may derive great benefit from replacement, but standard recommendations are not available.[27] For our patient, since the clinical symptoms were caused by combination of multiple pituitary hormone deficiencies, the diagnosis was made based on the presence of specific hormone deficiency symptoms, an established obstetric history, and lowered basal hormone levels such as prolactin, luteinizing hormone, cortisol and free thyroid hormones.

Figure II: Dynamic pituitary MRI showing partial empty sella

The investigation of choice was MRI of sella and parasellar region, which revealed a partial empty sella turcica following pituitary atrophy. We replaced relevant hormones considering her age and fertility desire.

Table: Results of endocrine evaluation

Laboratory Test	Result	Reference range, units	Interpretation
TSH	1.27	0.30-4.80 mIU/L	Inappropriately normal
Free T3	1.12	2.20-4.20 pg/ml	Low
Free T4	0.24	0.79-1.70 ng/dl	Low
9am cortisol	8.73	>15 mcg/dL	Inconclusive
ACTH	15.93	5.00-46.00 pg/mL	Low normal
Luteinizing hormone (LH)	2.64	8.70-76.30 mIU/L	Low
Follicular stimulating hormone (FSH)	8.93	3.40-33.40 mIU/L	Low normal
Prolactin	75.64	59.00-619.00 mcIU/L	Inappropriately normal
Short synacthen test (cortisol after 30min and 60 min)	10.20 14.70	>18.1 mcg/dl	Positive

Conclusion:

A high index of suspicion for Sheehan's syndrome by primary care physicians is warranted in patients with a bad obstetric history of intrapartum or postpartum hemorrhage. Signs of adenohypophysal insufficiency are often delayed and subtle leading to the diagnosis being easily missed. In some cases, the pituitary necrosis is partial and the syndrome can present in atypical and incomplete forms further complicating the diagnostic pathway. Awareness among clinicians is crucial so that such cases are not overlooked, especially in developing nations, where home delivery is still common and obstetric care is limited.

Conflict of Interest:

There is no conflict of interest of any authors in this study.

Acknowledgement:

We would like to acknowledge Department of Endocrinology, Bangladesh Medical University. We would like to offer special thanks to our patient for her cooperation in allowing us to use her information and MRI pictures for the case report.

References:

1. Kovacs K. Sheehan syndrome. *Lancet.* 2003;361(9356):520–522.

2. Qadri MI, Mushtaq MB, Qazi I, Yousuf S, Rashid A. Sheehan's syndrome presenting as major depressive disorder. *Iran J Med Sci.* 2015;40(1):73.

3. Schury MP, Adigun R. Sheehan syndrome. Treasure Island: *StatPearls Publishing;* 2019.

4. Shivaprasad C. Sheehan's syndrome: newer advances. *Indian J Endocrinol Metab.* 2011;15(Suppl3): S203.

5. Karaca Z, Laway BA, Dokmetas HS, Atmaca H, Kelestimur F. Sheehan syndrome. *Nat Rev Dis Primers.* 2016;2(1):1–15.

6. Perraudin V, Lefebvre H, Kuhn JM. Insuffisance antéhypophysaire. *Encycl Méd chi (Paris), Endocrinologie-Nutrition* 10-019-A-10. 1992:1–12.

7. Goswami R, Kochupillai N, Crock PA, Pituitary autoimmunity in patients with Sheehan's syndrome: *J Clin Endocrinol Metab,* 2002; 87(9); 4137-4141

8. Alwani R, Schmit Jongbloed L, De Jong F, van der Lely A-J, De Herder W, Feelders R. Differentiating between Cushing's disease and pseudo-Cushing's syndrome: comparison of four tests. *Eur J Endocrinol.* 2014;170(4):477–486.

9. Gokalp D, Alpagat G, Tuzcu A, Bahceci M, Tuzcu S, Yakut F, et al. Four decades without diagnosis: Sheehan's syndrome, a retrospective analysis. *Gynecol Endocrinol.* 2016;32(11):904–907.

10. Dizerega G, Kletzky OA, Mishell DR Jr. Diagnosis of Sheehan's syndrome using a sequential pituitary stimulation test. *Am J Obstet Gynecol.* 1978;132(4):348–353.

11. Lakhdar A, McLaren E, Dauda N, McKay E, Rubin P. Pituitary failure from Sheehan's syndrome in the puerperium. Two case reports. *Br J Obstetr Gynaecol.* 1987;94(10):998–999.

12. Pokharel S, Jha S, Maskey D, Shrestha B, Poudel P, Dhital B. A case report on Sheehan's syndrome. *J Chitwan Med College.* 2013;3(2):49–50.

13. Rajasekaran S, Vanderpump M, Baldeweg S, Drake W, Reddy N, Lanyon M, et al. UK guidelines for the management of pituitary apoplexy. *Clin Endocrinol.* 2011;74(1):9–20.

14. Ojb AL. Pituitary disorders. Drug treatment options. *Drugs.* 2000; 59:93–106.

15. Sert M, Tetiker T, Kirim S, Kocak M. Clinical report of 28 patients with Sheehan's syndrome. *Endocr J.* 2003;50(3):297–301.

16 Robalo R, Pedroso C, Agapito A, Borges A. Acute Sheehan's syndrome presenting as central diabetes insipidus. Case Rep. 2012;2012:bcr2012007022.

17. Anfuso S, Patrelli TS, Soncini E, Chiodera P, Fadda G, Nardelli G. A case report of Sheehan's syndrome with acute onset, hyponatremia and severe anemia. *Acta Biomed.* 2009;80(1):73–76.

18. Huang Y-Y, Ting M-K, Hsu B-S, Tsai J-S. Demonstration of reserved anterior pituitary function among patients with amenorrhea after postpartum hemorrhage. *Gynecol Endocrinol.* 2000;14(2):99–104.

19. Rajput R, Bhansali A, Khandelwal N, Dutta P, Bhadada S. Hyponatremic encephalopathy: A rare presenting manifestation of chronic Sheehan's syndrome. *J Indian Acad Clin Med.* 2009;10(1&2):72-74.

20. Purnell DC, Randall RV, Rynearson EH. Post-partum pituitary insufficiency: (Sheehan's's syndrome): Review of 18 cases. *Mayo Clin Proc.* 1964;39:321-331.

21. Bethune JE, Nelson DH. Hyponatremia in hypopituitarism. *N Engl J Med.*1965;272:771-776.

22. Şilfeler DB, Çelik M, Gökçe C, Balcı A, Dolapçıoğlu KS, Okyay AG. Sheehan's syndrome with recurrent hyponatremia and anemia: A case report. *East J Med.* 2014;19:33-37.

23. Martinez FJ, Lash RW. Endocrinologic and metabolic complications in the intensive care unit. *Clin Chest Med.* 1999;20(2):401-21. PMID: 10386264.

24. Gokalp D, Tuzzcu A, Bahceci M, Arikan S, Bahceci S, Pasa S. Sheehan's syndrome as a rare cause of anemia secondary to hypopituitarism. *Ann Hematol.* 2009;88(5):405-410.

25. Dash R, Gupta V, Suri S. Sheehan's syndrome: clinical profile, pituitary hormone responses and computed sellar tomography. *Aust N Z J Med.* 1993;23(1):26–31.

26. Lamberts S, De Herder W, van der Lely A-J. Pituitary insufficiency. *Lancet.* 1998;352(9122):127–134.

27. Davies J, Obuobie K, Smith J, Rees D, Furlong A, Davies N, et al. A therapeutic trial of growth hormone in hypopituitary adults and its influence upon continued prescription by general practitioners. *Clin Endocrinol.* 2000;52(3):295–303.

Case Report

Granulomatous Proctitis Mimicking Rectal Carcinoma: A Case of Isolated Rectal Tuberculosis in a Young Female

Abdur Rahman,[1] Mahabub Rahman,[2] Prof. Dr. Dewan Saifuddin Ahmed[3]

Abstract

Rectal tuberculosis (TB) is a rare manifestation of extrapulmonary tuberculosis (TB), and it can clinically and endoscopically mimic malignancy. We are here reporting the case of a 22-year-old female who presented with rectal bleeding, altered bowel habits, and systemic symptoms. Initial colonoscopic findings suggested rectal carcinoma, but histopathology confirmed the diagnosis of tuberculosis. The patient was treated successfully with anti-tubercular therapy (ATT), leading to complete symptomatic and endoscopic resolution. A high index of suspicion in endemic regions and early recognition of such atypical manifestations is crucial to prevent unnecessary interventions and to initiate appropriate therapy

Authors:
Abdur Rahman, HMO, Mugda Medical College Hospital, Dhaka
Email: ars.shawon0321@gmail.com

Mahabub Rahman, MBBS, FCPS, MD (Gastro), MD (Internal Medicine USA), Fellowship (Geriatric Internal Medicine)
Kaiser Permanent, Santa Clarita medical office

Prof. Dr. Dewan Saifuddin Ahmed, Professor and Chairman, Dept. of Gastroenterology, Bangladesh Medical University

Correspondence to:
Abdur Rahman, HMO, Mugda Medical College Hospital, Dhaka
Email: ars.shawon0321@gmail.com, Mobile: +8801913400036

Introduction:

Gastrointestinal tuberculosis is an uncommon presentation of gastrointestinal *Mycobacterium tuberculosis* infection. The rectal involvement is particularly the rarest. Nonspecific clinical presentation of Gastrointestinal TB can mimic malignancies, leading to diagnostic dilemmas.[1,2] That's why a high index of suspicion, early recognition, and appropriate management are crucial for favorable outcomes.

Case Presentation:

A 22-year-old female presented with a 6-month history of intermittent per-rectal bleeding along with occasional nocturnal urgency to defecate, which disrupted her sleep. Associated symptoms included mild unintended weight loss, malaise, and a continuous low-grade feverish feeling. There was no significant past medical history. She also denied any prior pulmonary symptoms or contact with tuberculosis patients. Physical examination was unremarkable except for significant pallor.

Laboratory investigations revealed moderately low hemoglobin and an elevated erythrocyte sedimentation rate (ESR). HIV testing was negative.

A colonoscopy was performed, which revealed an ulceroproliferative friable growth with contact bleeding in the rectum, raising suspicion for rectal carcinoma. The initial biopsy from the lesion demonstrated granulation tissue with fibrinopurulent exudate, which contains mixed acute and chronic inflammatory cells but no evidence of malignancy.

A repeat colonoscopic biopsy was conducted, which showed granulomatous inflammation with chronic inflammatory cells, consistent with tuberculosis. Acid-fast bacilli (AFB) staining was inconclusive, but clinical and histological characteristics favored a diagnosis of rectal tuberculosis. The patient was started on standard 4 drugs (isoniazid, rifampicin, pyrazinamide, ethambutol) anti-tubercular therapy. Over the following months, her symptoms gradually improved. Rectal bleeding stopped, nocturnal urgency resolved, and her general well-being improved.

After three months of treatment, a follow-up sigmoidoscopy revealed resolution of the growth with no residual lesion

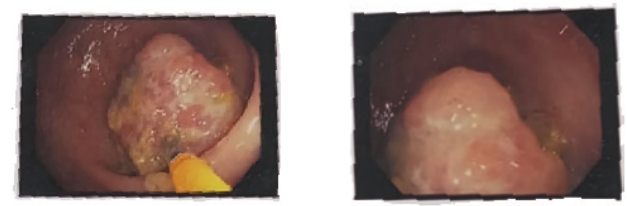

Figure I: Initial colonoscopic image showing an ulceroproliferative growth in the rectum with mucosal irregularity and friability, initially suggestive of rectal carcinoma.

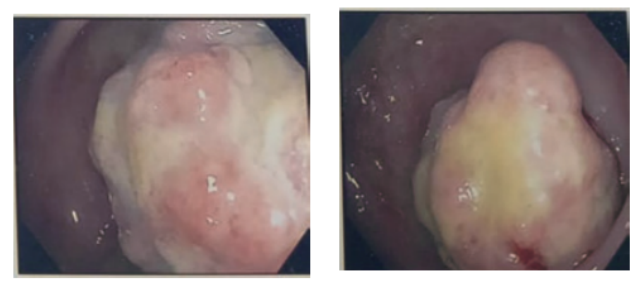

Figure II: Repeat colonoscopy showing persistent rectal lesion with similar morphology.

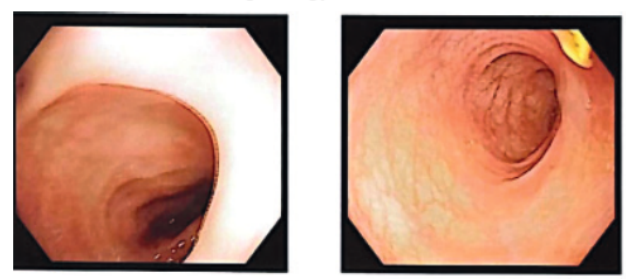

Figure III: Follow-up sigmoidoscopy after 3 months of anti-tubercular therapy demonstrating complete mucosal healing with no residual lesion.

Discussion:

Rectal tuberculosis is an uncommon but important differential diagnosis for isolated rectal masses, especially in young individuals from TB-endemic areas.[1,3] The clinical presentation of both diseases can closely mimic, leading to potential misdiagnosis and unnecessary interventions.

In addition to the nonspecific nature of the symptoms and the submucosal location of lesions, it often makes the diagnosis more challenging and lowers the diagnostic yield from endoscopic biopsies.[2,4] In our case, the initial biopsy was inconclusive, emphasizing the importance of repeat biopsies and thorough histopathological evaluation. Histopathological confirmation remains the gold standard for diagnosis, with granulomas and caseation being hallmark features.

Gastrointestinal tuberculosis can involve any part of the digestive tract, with the ileocecal junction being the most common. Isolated involvement of the rectum is rareand might be due to direct extension or hematogenous spread. The symptoms are usually nonspecific in nature and include, but are not limited to, abdominal fullness, nighttime urge to defecate, per rectal gross or occult bleeding, and other systemic features like fever and weight loss.[5]

Anti-tubercular drugs remain the mainstay of therapy. Our patient's dramatic clinical and endoscopic response to anti-tubercular drugs further confirms the diagnosis and highlights the significance of considering tuberculosis in the differential diagnosis of rectal lesions.

A careful follow-up with repeat colonoscopic evaluation is recommended to assess mucosal healing and to exclude any residual pathology.

Acknowledgement:

The authors would like to express their sincere gratitude to the patient and hospital authority for their assistance and co-operation.

Conclusion:

Rectal tuberculosis, though rare, should be included in the differential diagnosis for rectal bleeding with mass lesions, particularly in previously healthy young patients from endemic regions where TB is common. A high index of suspicion, appropriate biopsy, and histopathology are essential for timely diagnosis and cure. This case showed repeat biopsies are often needed to ensure accurate diagnosis. This case highlights the potential for TB to mimic rectal cancer and the importance of considering it as in atypical presentations.

References:

1. Eraksoy H. Gastrointestinal and Abdominal Tuberculosis. *Gastroenterol Clin North Am.* 2021 Jun;50(2):341-360. doi: 10.1016/j.gtc.2021.02.004. PMID:34024445.

2. Raghuvanshi V, Padwale VB. Gastrointestinal tuberculosis: clinical image. *Pan Afr Med J.* 2023 May 25;45:60. doi: 10.11604/pamj.2023.45.60.39839. PMID:37637398; PMCID: PMC10460097.

3. Al-Zanbagi AB, Shariff MK. Gastrointestinal tuberculosis: A systematic review of epidemiology, presentation, diagnosis and treatment. *Saudi J Gastroenterol.* 2021 Sep-Oct;27(5):261-274. doi: 10.4103/sjg.sjg_148_21. PMID: 34213424; PMCID: PMC8555774.4. Sheer TA, Coyle WJ. Gastrointestinal tuberculosis. *Curr Gastroenterol* Rep. 2003 Aug;5(4):273-278. doi: 10.1007/s11894-003-0063-1. PMID: 12864956.

5. Siddiqui WT, Quentzel H. Gastrointestinal tuberculosis: A mimicker of many. *Travel Med Infect Dis.* 2022 Mar-Apr;46:102272. doi:10.1016/j.tmaid.2022.102272. Epub 2022 Feb 3. PMID: 35123067

Case Report

Duodenal Dieulafoy's lesion- challenges in diagnosis: A Case report

Syeda Nur-E- Jannat,[1] Md. Raisul Arafat Riad,[2] Mohammad Shohidul Islam,[3] Md. Kamal Hossain Mozumdar[4]

Abstract

Dieulafoy's lesion is a rare but potentially life-threatening cause of upper gastrointestinal (GI) bleeding, typically presenting with severe haematemesis, melaena, or haematochezia without prior symptoms, often with hemodynamic instability requiring transfusion of multiple units of packed erythrocytes. Diagnosis is frequently challenging due to its subtle endoscopic appearance and obscure location of the lesion. Early endoscopy during a bleeding episode is essential for an accurate diagnosis and sometimes multiple endoscopies are needed to establish the diagnosis. Here, we present a case of a 51-year-old female with multiple comorbidities, including end-stage renal disease (ESRD) on maintenance hemodialysis and ischemic heart disease, who presented with persistent melaena for one month. After extensive evaluation, she was diagnosed with Dieulafoy's lesion in the duodenum. Prompt endoscopic management stabilized her condition. This case highlights the diagnostic challenges and clinical importance of recognizing Dieulafoy's lesion in patients with complex medical histories.

Authors:
Syeda Nur-E- Jannat
Associate professor (Gastroenterology),
National Gastroliver Institute & Hospital

Md. Raisul Arafat Riad
FCPS Part 2 Trainee, National Gastroliver Institute & Hospital

Mohammad Shohidul Islam
Assistant professor (Gastroenterology),
National Gastroliver Institute & Hospital

Md. Kamal Hossain Mozumdar
FCPS Part 2 Trainee, National Gastroliver Institute & Hospital

Corresponding Author
Syeda Nur-E- Jannat
MBBS (DMC), FCPS (Medicine), MD (Gastroenterology)
Associate professor (Gastroenterology),
National Gastroliver Institute & Hospital,
Email: jannatdmc@yahoo.com, Phone: +8801819442631

Introduction:

Dieulafoy's lesion (DL) was first reported by Gallard in 1884, but was named after a French surgeon, Georges Dieulafoy, who described the lesion more precisely in 1898.1,2 It is defined anatomically as a dilated (1-3 mm), aberrant, submucosal artery that erodes the overlying GI mucosa in the absence of an underlying ulcer, aneurysm, or intrinsic mural abnormality.3 It accounts for 1–2% of acute GI bleeding cases.[4,5] Patients are typically asymptomatic before presenting with acute, profuse GI bleeding, often recurrent, which can manifest as hematemesis, melena or hematochezia with haemodynamic instability.[6,7] About 75% of lesions are located in the stomach, usually within 6 cm of the gastroesophageal junction along the gastric lesser curve.8 The duodenum is the second most common site for a Dieulafoy's lesion (14–18% of cases), and the majority of these lesions are located in the duodenal bulb (53%), followed by the third part of the duodenum (29%) and the junction of the first and second parts of the duodenum (18%).[9,10] The diagnosis and management of bleeding DLs rely primarily on endoscopy. Given its rarity and difficulty in diagnosis - especially in patients with significant comorbidities such as ESRD and ischemic heart disease - early diagnosis and timely endoscopic intervention are crucial.

Case Presentation:

A 51-year-old woman, a housewife from Savar, Dhaka, presented with a history of black, tarry, foul-smelling stools (melaena) 4-5 episodes per day for the last 4 weeks. She also complained of extreme fatigue and breathlessness on exertion for the last 15 days. She had no history of abdominal pain, heartburn, hematemesis, or any other bleeding manifestation like haemoptysis, epistaxis, or easy bruising. No history of jaundice, pedal edema, altered level of consciousness, joint pain, oral ulceration, skin rash, fever, cough, contact with a known TB patient, weight loss, or recent history of taking NSAIDs, steroids, or iron-containing drugs. Her appetite was normal. With all the above complaints, she got admitted herself to the National Gastroliver Institute & Hospital (NGLIH), Dhaka for better management.

She was known to be hypertensive, non-diabetic and diagnosed with CKD of unknown etiology in 1997. She had a renal transplant in 2009, followed by immunosuppressive therapy (steroids, cyclosporine, and azathioprine) and was doing well until 2021. Since then, her renal function deteriorated, requiring hemodialysis (HD) and she discontinued all immunosuppressant medication. Now, she was on maintenance hemodialysis (MHD) three times per week and on erythropoietin once in each cycle of HD. She was a known case of ischemic heart disease (IHD) with PTCA & stenting to the LCX in 2021 and was taking aspirin and clopidogrel. She used to take regular anti-hypertensive medication with good control of BP. She required blood transfusions every 2-3 months interval after starting HD since 2023.

Initially, she was admitted to a local hospital, where she was resuscitated with IV fluid, injection ceftriaxone 1 gm twice a day, 9 units of blood transfusion for melaena, sucralfate (1 gm every 6 hours), and injection proton pump inhibitor (8 mg/hour) infusion for the last 7 days without any significant improvement in bleeding. She was started on heparin-free HD and dual antiplatelet therapy was also stopped for the last 1 month.

She had no family history of liver disease, renal disease, cardiac disease, or coagulation disorder. She denied any history of tobacco, alcohol or betel nut intake. She had three abortions, and her menstrual cycle was normal. She was vaccinated for HBV and COVID-19.

On examination, she appeared ill, moderately anemic, non-icteric, had a cushingoid appearance, generalized hyperpigmentation, and leukonychia. There was also a left-arm arteriovenous fistula, but no stigmata of chronic liver disease (CLD), no edema, no lymphadenopathy, and JVP was not raised. Vitals were within normal limits.
Abdominal examination revealed a scar mark in the right iliac fossa (RIF) and hypogastric region, mild hepatomegaly and a transplanted kidney in the RIF. No splenomegaly, no ascites. Cardiovascular examination detected a pansystolic murmur all over the precordium. Other systemic examinations revealed normal findings.

Investigations:

Her hemoglobin level was repeatedly low, ranging from 6.4 to 7.4 gm/dL with blood transfusion indicating ongoing bleeding; the total platelet count was also low, ranging from 90,000/µL to 77,000/µL. Serum creatinine ranged between 4.8 to 8.8 mg/dL with MHD. Liver function tests, prothrombin time with INR, APTT, FDP and D-Dimer were within normal limit. Iron profile was consistent with anemia of chronic disease. Viral markers, HBsAg and anti-HBc (total) were negative, but anti-HCV was positive with HCV RNA negative.USG of the whole abdomen revealed a graft kidney in the right iliac fossa, with normal liver echotexture, no intra-abdominal lymphadenopathy and no ascites. Doppler USG of the hepatobiliary-pancreatic vessels revealed normal vascular flow patterns. Chest X-ray showed mild cardiomegaly, and echocardiogram revealed a good ejection fraction (70%).

She underwent an upper GI endoscopy outside the hospital before admission, which showed antral polypoid swelling and clotted blood in the stomach, with fresh oozing of blood in the bulb, postbulbar area and second part of the duodenum onward, without any specific bleeding source in the duodenum or stomach. After admission to NGLIH, we repeated upper GI endoscopy twice with adequate water jet irrigation, flushing and suction, with the same findings of fresh and altered blood in the stomach and duodenum, without identifying any specific point of active bleeding.

As bleeding continued, after resuscitation and adequate counseling, under anesthesia, we attempted a 4th upper GI endoscopy. After rinsing with water and aspiration, a specific source of oozing blood from the floor close to the lateral wall near the second part of the duodenum could be identified (Figure 1). Due to peristalsis and pulsation, it was difficult to exactly localize the bleeding point. So, with the aim of better visualization of the lateral wall and floor of the second part of the duodenum, we introduced a pediatric colonoscope, with adequate suction and water insufflation. Micro pulsatile bleeding from a bleeding vessel without any ulceration could be identified at the floor of the junction of the first and second part of the duodenum. Adrenaline (1:10,000 dilution) was injected at the base, followed by argon plasma coagulation (APC) applied to the bleeding vessel (Figure 2). After two days, her melaena stopped, her vital status remained stable, and her hemoglobin also rose to 10 gm/dL. No surgical or angiographic intervention was required. Subsequently, the patient was discharged from the hospital to home.

Thus, our final diagnosis was actively bleeding Dieulafoy's lesion at the junction of the first and second parts of the duodenum.

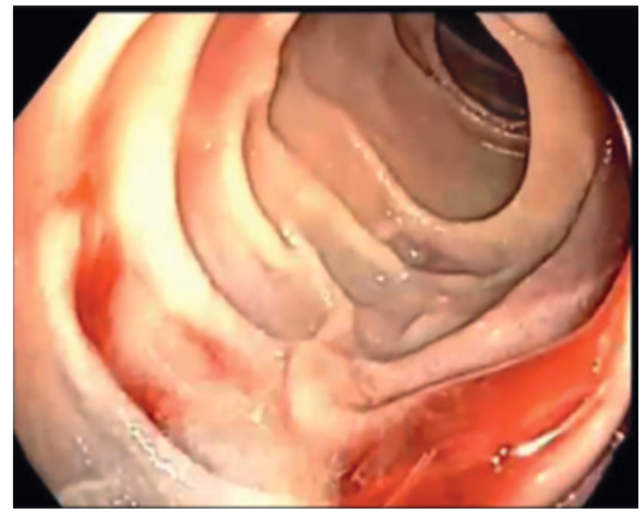

Figure I: Shows fresh blood in infero-lateral wall of duodenum.

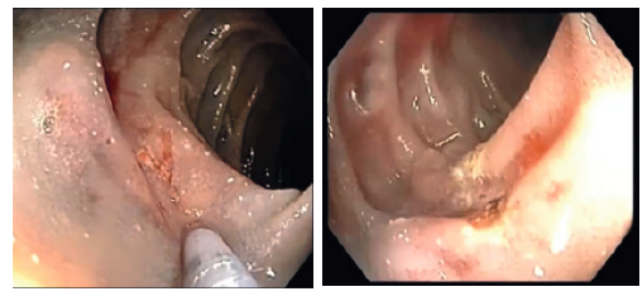

Figure II: haemostasis with injection adrenalin (1:10,000 dilution) followed by Argon Plasma Coagulation (APC)

Discussion:

Dieulafoy's lesion (DL) is an uncommon but important cause of recurrent GI bleeding. The condition is often underdiagnosed due to its small mucosal defect, obscure location of the lesion, and intermittent bleeding.[5,11] We diagnosed duodenal DL as a cause of recurrent melaena in a woman in her sixth decade of life. DL can occur at any age but is most common in the elderly, typically in the fifth or sixth decade of life.[5,12,13] GI bleeding from DL is frequently linked with comorbid conditions, including cardiovascular disease, chronic kidney disease, hypertension, peptic ulcer disease, diabetes mellitus and long-term use of medications such as nonsteroidal anti-inflammatory drugs and anticoagulants.[14,15] In this patient, ESRD with hemodialysis and prior dual antiplatelet therapy likely contributed to her bleeding risk.

Our patient required endoscopy four times to establish a diagnosis of duodenal DL, although initial endoscopy is diagnostic in approximately 70% of cases, with about 6% of patients requiring three or more attempts. Initial endoscopy may be compromised by factors such as excessive blood, subtle lesions obscured by folds or gastric contents, surrounding normal mucosa, blood clots adhering to the site or pooled blood from significant hemorrhage.[5,8] Moreover, DL located in the periampullary area and second part of the duodenum is more difficult to diagnose, as they cannot be seen or treated by a forward-viewing endoscope.[16,17]

Lateral-view endoscopy can visualize the periampullary region better. We used a pediatric colonoscope, as it has additional insertion length and a favorable 5- to 6-o'clock orientation of the working channel, which provided an advantage for better visualization of the lateral and posterior walls and distal duodenum to identify the bleeding point source.[18] Endoscopies performed within the first 12 hours have a high success rate for diagnosing DLs because of their capability to pinpoint the bleed location.[19]

Endoscopic hemostasis by different procedures, like regional injection, thermal techniques, and mechanical methods, is the main modality of treatment, with surgery reserved for refractory cases in patients with uncontrollable bleeding, alongside endoscopic and angiographic embolization methods.[5,20,21] The choice of therapeutic technique will depend on the clinical presentation, lesion site, and available surgical and endoscopic expertise. Endoscopic injection therapy using vasoconstrictors like adrenaline, sclerosants (ethanol, polidocanol) or N-butyl 2-cyanoacrylate is a simple and cost-effective method but has a high risk of rebleeding when used alone. Thermal therapy includes contact methods (bipolar, heater probe), which are effective but carry a risk of transmural injury, and non-contact argon plasma coagulation (APC), which is safer but mainly useful for superficial lesions. Mechanical therapies, such as hemoclips and band ligation, are preferred for achieving hemostasis but can be challenging due to duodenal angulation. Band ligation has a lower risk of perforation but may cause rebleeding ulcers.[13,19,22] Combining endoscopic therapies, such as injection therapy followed by thermal or mechanical interventions, has proven to be superior to single-modal approaches, with permanent hemostasis achieved in 95% of patients.3 In our case, a combination of APC with adrenaline injection was used to successfully secure hemostasis.
The risk of rebleeding from DL ranges from 9% to 40%,[5,21] for which regular follow-up for at least 6 months is recommended. Mortality from bleeding due to this lesion is 9%–13%.[8]

Conclusion:

Although rare, Dieulafoy's lesion should be considered an important etiology of unexplained, life-threatening upper GI bleeding, particularly in an elderly patient with multiple comorbidities. The localization of Dieulafoy's lesion in the duodenum can be more difficult. Increased awareness and careful, early endoscopic evaluation following the bleeding episode are key to accurate diagnosis and prompt therapeutic intervention. Endoscopic hemostasis is crucial for successful management, a favorable outcome and reducing the need for invasive surgical interventions.

References:

1. Gallard MT. Aneurysme miliaires de l'estomach, donnant lieu a des hematemese mortelles. *Bull Soc Med Hop Paris* 1884; 1: 84-91

2. Dieulafoy G. Exulceratio simplex. L'intervention chirurgicale dans les hematemeses foudroyantes consecutives a l'exulceration simple de l'estomac. *Bull Acad Med* 1898; 49: 49-84

3.Lee YT, Walmsley RS, Leong RW, Sung JJ. Dieulafoy's lesion. *Gastrointest Endosc* 2003; 58: 236-243 [PMID: 12872092] https:// doi. org/ 10. 1067/ mge. 2003. 328.

4. Malik T. F. and Anjum F., "Dieulafoy's Lesion Causing Gastrointestinal Bleeding," in StatPearls *(StatPearls Publishing, 2023).*

5. Baxter M, Aly EH. Dieulafoy's lesion: current trends in diagnosis and management. *Ann R Coll Surg Engl.* 2010;92(7):548–554. https:// doi. org/ 10. 1308/ 00358 8410X 12699 66390 5311.

6. Luis LF, Sreenarasimhaiah J, Jiang Tang S. Localization, efficacy of therapy, and outcomes of Dieulafoy lesions of the GI tract – The UT Southwestern GI Bleed Team experience. *Gastrointest Endosc.* 2008; 67: AB 87

7. Lopez-Arce G, Zepeda-Gomez S, Chavez-Tapia NC, et al. Upper gastrointestinal Dieulafoy's lesions and endoscopic treatment: first report from a Mexican centre. *Therap Adv Gastroenterol.* 2008;1(2):97–101

8. Nojkov B, Cappell MS. Gastrointestinal bleeding from Dieulafoy's lesion: Clinical presentation, endoscopic findings, and endoscopic therapy. *World J Gastrointest Endosc* 2015; 7(4): 295-307. https:// doi. org/ 10. 4253/ wjge. v7. i4. 295.

9. Chaer RA, Helton W.S. Dieulafoy's disease *J. Am. Coll. Surg.*2003 Feb1;96 (2):290-296.

10. Rana SS, Bhasin DK, Gupta R, Yadav TD, Gupta V, Singh K. Periampullary Dieulafoy's lesion: an unusual cause of gastrointestinal bleeding. *JOP.*2010;5 (11) :266-269.

11. Schmulewitz N, Baillie J. Dieulafoy lesions: a review of 6 years of experience at a tertiary referral center. *Am J Gastroenterol.* 2001;96(6):1688–1694.

12. Shewaye AB, Berhane KA, Fanta AD, Adem A. Bleeding duodenal Dieulafoy's lesion in a pediatric patient from Ethiopia: A Case Report. Clinical Case Reports, 2025 Aug; 13(8):e70743 https://doi.org/10.1002/ccr3.70743

13. Di Nardo G, Esposito G, Mauro A, et al. Dieulafoy lesion: two pediatric case reports. *Italian Journal of Pediatrics.*2020;46(1) doi:10.1186/s13052-020-0814-8.

14. Saleh R, Lucerna A, Espinosa J, Scali V. Dieulafoy lesion: the little known sleeping giant of gastrointestinal bleeds. *American journal of Emergency Medicine*.2016 Dec1; 34(12):2464.e3–5

15. Kasapidis P, Georgopoulos P, Delis V, Balatsos V, Konstantinidis A, Skandalis N. Endoscopic management and long term follow- up of Dieulafoy's lesions in the upper GI tract. *Gastrointestinal Endoscopy.* 2002 Apr;55(4): 527–531.

16. Lee WS, Cho SB, Park SY, et al. Succesful side-viewing endoscopic hemoclipping for Dieulafoy-like lesion at the brim of a periampullary diverticulum. *BMC Gastroenterol.* 2010;10:24–29.

17. Pollack R, Lipsky H, Goldberg RI. Duodenal Dieulafoy's lesion. *Gas trointest Endosc.* 1993;39(6):820–822.

18. Michael J. Bourke,1,2 Simon K. Lo,3 Ross C. D. Buerlein,4 and Koushik K. Das AGA Clinical Practice Update on Nonampullary Duodenal Lesions: Expert Review. *Gastroenterology* 2025;168:169–175.

19. Yılmaz T, Kozan R. Duodenal and jejunal Dieulafoy's Le sions: optimal management, *Clinical and Experimental Gastroenterology*.2017;10: 275–283.

20. López JDM, Hernández CAT, Gómez DO, Návar AD, Ortiz CAB, Reséndiz BF, et al. Duodenal dieulafoy injury: case report. *Int Surg J.*2024;11:104-106.

21. Jeon HK, Kim GH. Endoscopic management of Dieulafoy's Lesion. *Clinical Endoscopy.* 2015;48(2):112–120.

22. Inayat F, Amjad W, Hussain Q, Hurairah A. Dieulafoy's Lesion of the Duodenum: a comparative review of 37 cases. *BMJ* Case Reports. 2018 Feb22; bcr-2017-223246

Review Article

High-Resolution Manometry in Esophageal Motility Disorders: Latest Clinical Evidence and the Impact of Chicago Classification Version 4.0

Mohammad Shohidul Islam

Abstract

High-resolution manometry (HRM) has fundamentally transformed the diagnosis and management of esophageal motility disorders (EMDs) by providing detailed, spatiotemporal pressure topography and offers a superior evaluation of esophageal function compared to conventional manometry. HRM interpretation is standardized by the Chicago Classification (CC). The clinical landscape has evolved significantly with the introduction of CC version 4.0 (CCv4.0) and complementary technologies. This review article provides an updated synthesis of HRM in clinical practice, incorporating the latest evidence. It discusses key changes introduced in CCv4.0, such as the expanded protocol with provocative maneuvers (Multiple Rapid Swallows and Rapid Drink Challenge) and a refined diagnostic framework for achalasia, esophagogastric junction outflow obstruction (EGJOO), and peristaltic abnormalities. The article also explores the critical pre-operative role of HRM before anti-reflux surgery and its utility in evaluating refractory GERD symptoms. Furthermore, it addresses the growing importance of combining HRM with adjunct technologies like FLIP, barium radiography to enhance diagnostic confidence, especially in challenging cases. Finally, the review outlines current limitations, such as symptom correlation for minor disorders, and discusses future perspectives in the field.

Keywords: High-resolution manometry, Chicago Classification 4.0, esophageal motility disorders, achalasia, EGJ outflow obstruction, provocative maneuvers, functional lumen imaging probe.

Author & Corresponding to:
Mohammad Shohidul Islam
Assistant Professor (Gastroenterology),
National Gastroliver Institute and Hospital, Mohakhali, Dhaka.
Email: msironyk56@gmail.com, Mobile: +8801818711523

Introduction:

High-resolution manometry (HRM) has become the gold standard for assessing esophageal motor function since its clinical introduction.[1] Unlike conventional manometry, which relies on a few widely spaced pressure sensors, HRM utilizes a high density of pressure transducers to generate a continuous, color-coded pressure map, known as esophageal pressure topography (EPT). This enhanced resolution has enabled a more precise characterization of esophageal motility, leading to improved diagnostic accuracy and the identification of distinct EMD phenotypes.[2] The interpretation of HRM studies is standardized by the Chicago Classification (CC), a hierarchical framework that has been regularly updated by international experts to incorporate new research findings.[3] The latest iteration, CCv4.0 was published in 2021 and marks a paradigm shift from a purely metric-based classification to a more integrated, clinically-oriented diagnostic approach (Figure-I).[4]

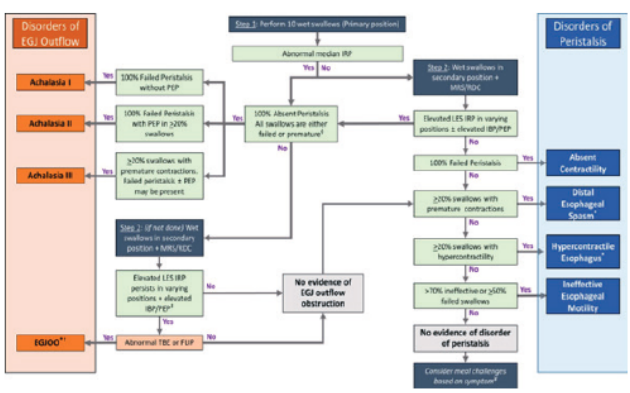

Figure I: Chicago Classification 4.0 Hierarchical Classification Scheme[19]

This update introduces a more robust protocol, emphasizing provocative maneuvers and clinical correlation, which is crucial for translating manometric findings into meaningful clinical decisions.[5] This review article aims to provide a comprehensive overview of the latest clinical evidence related to HRM. We will discuss the key technical aspects of HRM, detail the major changes in CCv4.0 and their clinical implications, and explore the role of HRM in diagnosing specific EMDs. Furthermore, we will examine the integration of complementary technologies and address the current limitations and future perspectives of HRM in gastroenterology.[6]

2. The Impact of Chicago Classification Version 4.0

The Chicago Classification (CC) v4.0 was developed to address several shortcomings of earlier versions, particularly regarding the need for greater diagnostic certainty and clinical correlation.[3] The updated protocol (Figure-II) expands beyond the standard 10 supine liquid swallows to include additional maneuvers that provide more comprehensive physiological data.[7]

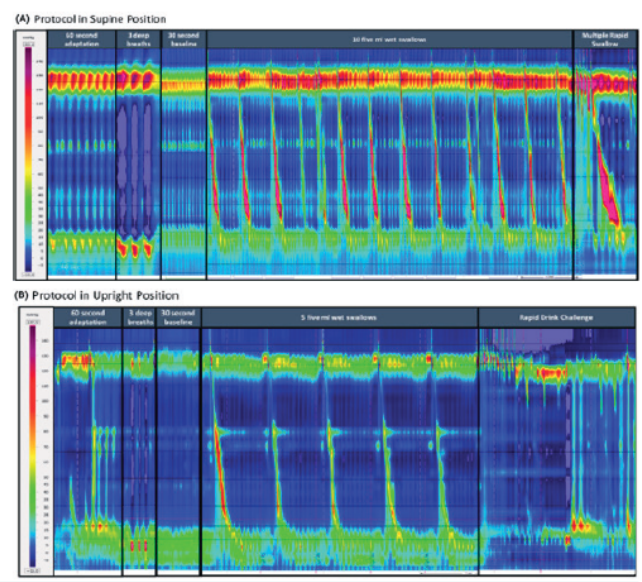

Figure II: High-resolution manometry images depicted the standard protocol.

A, The supine position includes a 60 second adaptation period, 3 deep breaths, 30 second baseline period, 10 five ml wet swallows and at least one multiple rapid swallow.

B, Position is changed to the upright position followed by a 60 second adaptation, 3 deep breaths, 30 second baseline period, 5 five ml wet swallows, and a rapid drink challenge [19]

2.1. Disorders of EGJ Outflow: Refining the Diagnosis of Achalasia

CCv4.0 has further refined the subtyping of achalasia, a disorder characterized by impaired LES relaxation and absent peristalsis (Figure-III). While the three classic subtypes (I, II and III) are retained, the diagnostic criteria have been tightened.

Type I (Classic): Requires a median integrated relaxation pressure (IRP) > upper limit of normal (ULN) and 100% failed peristalsis (Distal Latency [DL] > ULN but Distal Contractile Integral [DCI] < 100 mmHg•s•cm).

Type II (with Pan-esophageal Pressurization): Requires pan-esophageal pressurization in ≥20% of swallows, a more stringent threshold than the previous ≥2 swallows, to improve diagnostic consistency.[4,8]

Type III (Spastic): Defined by the presence of ≥20% premature contractions (DL < 4.5 s) with a median IRP > ULN.

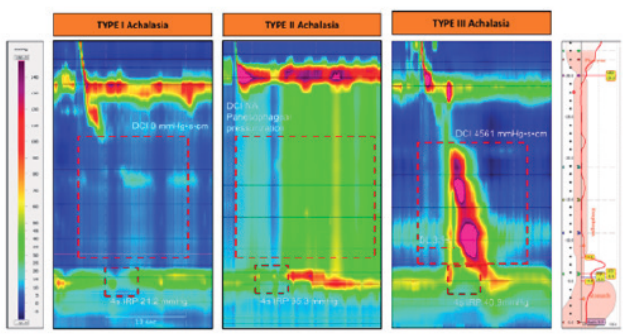

Figure III: Achalasia Subtypes. Type I Achalasia: integrated relaxation pressure (IRP) is elevated with failed peristalsis (DCI) <100 mmHg-s-cm), and without panesophageal pressurization. Type II Achalasia: IRP is elevated with failed peristalsis and panesophageal pressurization. Type III Achalasia: IRP is elevated with a normal DCI, and a reduced distal latency.[19]

2.2. Major Motility Disorders: Absent Contractility and its Specificity

The diagnosis of "absent contractility" is now reserved for instances where there is 100% failed peristalsis and a normal median IRP. Crucially, CCv4.0 explicitly links this diagnosis to the clinical context of systemic diseases, most notably scleroderma.4 This emphasizes that absent contractility is not an idiopathic condition but is strongly associated with connective tissue disorders, guiding appropriate patient evaluation and management.

2.3. The Introduction of the "Inconclusive" Category

Recognizing that not all manometric findings are clear-cut, CCv4.0 formally introduces an "Inconclusive" category. This applies to studies with borderline metrics (e.g., IRP just above the ULN without supporting evidence for EGJOO) or inconsistent patterns.4 This category prompts the clinician to seek additional information, often through provocative testing, rather than forcing a potentially incorrect diagnosis. (Figure-IV)

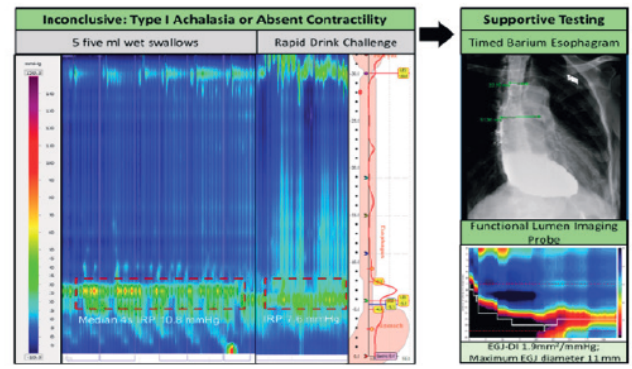

Figure IV: Inconclusive Diagnosis for Achalasia or Absent Contractility requires supportive Testing with timed barium esophagram and/or functional lumen imaging probe (FLIP). Here, the timed barium esophagram demonstrates a dilated distal esophagus with barium retention. On FLIP,the esophagogastric junction (EGJ) distensibility index (EGJ-DI) is reduced, maximal EGJ diameter is reduced and there is absent contractile response to distension.[19]

A major update is the introduction of **"EGJ Outflow Obstruction (EGJOO)"** as a distinct, potentially clinically relevant diagnosis, rather than a manometric finding of uncertain significance. CCv4.0 mandates that an elevated IRP must be accompanied by supporting evidence of obstruction, such as symptoms of dysphagia, evidence of retention on timed barium swallow, or a dilated esophagus on endoscopy.[4,9] This prevents over-diagnosis of inconsequential findings. (Figure-V)

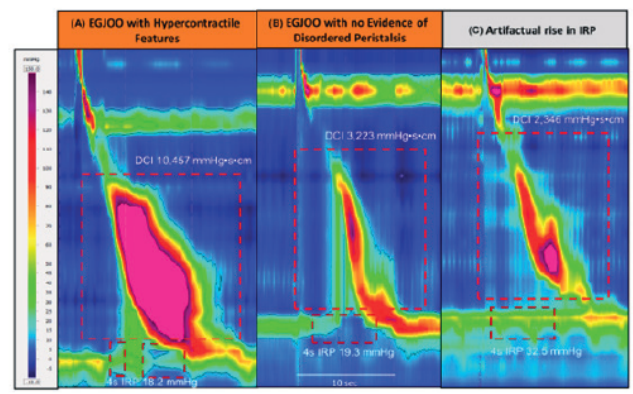

Figure V: EGJOO subtypes: A) EGJOO with hypercontractile features: IRP is elevated with intrabolus pressurization and hypercontractile swallow. B) EGJOO with no evidence of disordered peristalsis: IRP is elevated with normal contractile vigor. C) Manometric EGJOO related to artifactual rise in IRP: IRP is elevated in the absence of intrabolus pressurization and is likely associated with artifact.[19]

2.4. Enhanced Role of Provocative Maneuvers

CCv4.0 places greater emphasis on the use of provocative maneuvers during HRM to uncover latent motility abnormalities and assess esophageal reserve function.[10]

Multiple Rapid Swallow (MRS): This test assesses the integrity of neuromuscular inhibition and subsequent peristaltic augmentation. A normal response is inhibition during the swallows followed by a augmented contraction (post-MRS DCI > the mean single-swallow DCI). Its role in predicting outcomes following therapy for achalasia (e.g., peroral endoscopic myotomy, POEM) is an area of active research.[11]

Rapid Drink Challenge (RDC): Involves rapid ingestion of a liquid bolus (e.g., 200 mL). It is highly sensitive for detecting EGJ outflow obstruction, often eliciting a pan-esophageal pressurization in achalasia that may not be apparent on single water swallows.[12]

Standardized Test Meal: The use of a solid test meal (e.g., a 4-cm rice cake) during HRM can reproduce symptoms like dysphagia that are not present during liquid swallows. This "post-prandial HRM" can identify meal-induced abnormalities and is particularly useful in patients with symptoms out of proportion to standard HRM findings.[13]

3. Clinical Evidence and Impact on Management

The refinements in CCv4.0 are directly supported by clinical evidence that links manometric patterns to pathophysiology and treatment outcomes.

Achalasia Subtyping and Treatment Selection: The prognostic value of achalasia subtyping is well-established. Type II achalasia has the best response to any therapy (pneumatic dilation, Heller myotomy, or POEM), while Type I may require more aggressive myotomy, and Type III, characterized by spasticity, often responds best to POEM due to its ability to extend the myotomy proximally.[8,14]

Clarifying EGJOO: The stricter criteria for EGJOO help distinguish true, clinically significant obstructions (e.g., from early achalasia, strictures, or eosinophilic esophagitis) from pseudo-obstructions caused by hiatal hernia or repetitive swallowing. This prevents unnecessary invasive procedures in patients with a benign manometric finding.[9]

Hypercontractile Esophagus and Distal Esophageal Spasm (DES): CCv4.0 maintains the distinction between hypercontractile esophagus (Jackhammer esophagus) and DES based on the presence of premature contractions. This is clinically relevant as the two disorders may respond differently to smooth muscle relaxants or neuromodulators.[4]

3.2 Evaluation of Refractory GERD and Non-cardiac Chest Pain: In patients with persistent symptoms despite optimal medical therapy, HRM can uncover underlying EMDs that mimic GERD or cause non-cardiac chest pain.[15]

Refractory GERD: HRM helps exclude achalasia and identifies disorders like IEM that may contribute to impaired acid clearance. It also characterizes EGJ morphology and contractility, which can have implications for reflux pathophysiology.[7]

Chest Pain: HRM can diagnose spastic EMDs like hypercontractile esophagus or distal esophageal spasm that may be responsible for non-cardiac chest pain.[5]

3.3 Pre-operative Assessment for Anti-reflux Surgery: HRM is an indispensable tool before fundoplication. It helps to:[16]

- Identify contraindications, such as achalasia or absent contractility, which would lead to post-operative dysphagia.
- Determine the integrity of peristalsis, which can influence the choice between a partial or a full fundoplication.
- Characterize the EGJ morphology, providing insights into the presence and type of hiatal hernia.

4. Complementary Technologies: HRM, FLIP, and HRIM: The diagnostic landscape is moving toward a multimodal approach, where HRM is often complemented by other technologies to enhance diagnostic confidence.[17]

4.1 Functional Lumen Imaging Probe (FLIP)

FLIP provides real-time information about the distensibility and contractility of the EGJ and esophageal body. This is particularly valuable in cases where HRM findings are inconclusive or discordant with patient symptoms. (Figure-IV) For example, FLIP can reveal impaired EGJ distensibility in some patients with EGJOO, providing objective confirmation of outflow obstruction.[16]

4.2 High-Resolution Impedance Manometry (HRIM)

HRIM combines pressure and impedance sensing, providing information on bolus transit in addition to pressure activity. This offers a more complete picture of esophageal function, as it can identify bolus clearance abnormalities even in cases with seemingly normal peristalsis on standard HRM.[17]

5. Limitations:

Despite its clear advantages, HRM is not without limitations. A significant challenge remains the poor correlation between minor manometric abnormalities (e.g., IEM) and clinical symptoms. This highlights the need for careful clinical assessment alongside manometric interpretation, a point underscored by CCv4.0.[18]

6. Acknowledgement:

The authors would like to express their sincere gratitude to the patient and hospital authority for their assistance and co-operation.

7. Conclusion

High-resolution manometry, interpreted through the robust framework of the Chicago Classification, remains the cornerstone of modern esophageal motility evaluation. The introduction of CCv4.0, with its expanded protocol and emphasis on clinical correlation, has further strengthened HRM's role in clinical practice. The integration of provocative maneuvers and complementary technologies like FLIP

and HRIM allows for a more comprehensive assessment of esophageal function, leading to more accurate diagnoses and better-informed treatment decisions. While challenges persist in fully correlating certain findings with patient symptoms, the evolution of HRM and its adjuncts promises to continue improving the diagnostic and therapeutic approach to esophageal motility disorders.

References:

1. Demeester SR, Jones L, Thompson P. High-resolution manometry and the treatment of achalasia. *World J Gastroenterol.* 2019;25(1):21-30.

2. Shaheen NJ, Kahrilas PJ. Diagnosis and management of achalasia. *Gastroenterology.* 2017;153(4):760-773.

3. Pandolfino JE, Gawron AJ, Zhang Q. Chicago Classification version 4.0: Innovations and updates in esophageal motility diagnosis. *Curr Gastroenterol Rep.* 2021;23(3):1-9.

4. Yadlapati R, Pandolfino JE, Fox MR, Bredenoord AJ, Kahrilas PJ. What is new in Chicago Classification version 4.0? *Neurogastroenterol Motil.* 2021 Jan;33(1):e14053.

5. Yadlapati R, Kahrilas PJ. High-resolution manometry and the Chicago classification: a review of its clinical significance. *Gastroenterol Clin North Am.* 2021;50(4):731-746.

6. Vela MF, Mellow M, Padilla D. High-resolution manometry and its clinical applications. *Am J Gastroenterol.* 2018;113(4):523-530.

7. Schwizer W, Fried M, Beglinger C. HRM in gastroesophageal reflux disease. *Am J Gastroenterol.* 2019;114(5):874-881.

8. Rohof WO, Salvador R, Annese V, Bruley des Varannes S, Chaussade S, Costantini M, et al. Outcomes of treatment for achalasia depend on manometric subtype. *Gastroenterology.* 2013 Apr;144(4):718-725.

9. Wang D, Patel A, Mello M, Shriver A, Gyawali CP. Esophagogastric junction outflow obstruction on high-resolution manometry: clinical and therapeutic implications. *Dis Esophagus.* 2020 Mar 16;33(3):doz086.

10. Sweis R, Anggiansah A, Wong T, Brady G, Fox M. Assessment of esophageal dysfunction and symptoms during and after a standardized test meal: development and clinical validation of a new methodology utilizing high-resolution manometry. *Neurogastroenterol Motil.* 2014 Mar;26(3):379-395.

11. Mari A, Abu Baker F, Pellicano R, Khoury T. Multiple Rapid Swallow (MRS) and the Role of the Chicago Classification in the Management of Esophageal Motility Disorders: A Systematic Review. *J Clin Med.* 2022 Jan 28;11(3):692.

12. Tolone S, de Cassan C, de Bortoli N, Roman S, Galeazzi F, Salvador R, et al. Esophagogastric junction morphology is associated with a positive impedance-pH monitoring in patients with GERD. *Neurogastroenterol Motil.* 2015 Sep;27(9):1175-1182

13. Xiao Y, Kahrilas PJ, Kwasny MJ, Roman S, Lin Z, Nicodème F, et al. High-resolution manometry correlates of ineffective esophageal motility. *Am J Gastroenterol.* 2012 Nov;107(11):1647-1654.

14. Kahrilas PJ, Katzka D, Richter JE. Clinical Practice Update: The Use of Per-Oral Endoscopic Myotomy in Achalasia: Expert Review and Best Practice Advice from the AGA Institute. *Gastroenterology.* 2017 Nov;153(5):1205-1211.

15. Ashby M, Mehta P. The role of HRM in pre-operative assessments for anti-reflux surgery. *Surg Endosc.* 2020;34(2):10-15.

16. Martin J, Hu K, Song H. The role of FLIP and HRIM in esophageal motility evaluation. *Gastroenterology.* 2021;160(6):2345-2354.

17. Thomas K, Kularatna M. Multimodal diagnostic approaches in HRM: A review. *J Clin Gastroenterol.* 2021;56(7):654-660.

18. Kahrilas PJ, Bredenoord AJ, Fox M. Gastroesophageal reflux disease and motility disorders. *Lancet Gastroenterol Hepatol.* 2021;6(2):56-65.

19. Yadlapati R, Kahrilas PJ, Fox MR, Bredenoord AJ, Gyawali CP, Roman S, et al. Esophageal motility disorders on high-resolution manometry: Chicago classification version 4.0. *Neurogastroenterol Motil.* 2020 Dec 29;32(1):e14058. doi: 10.1111/nmo.14058.

Letter to the Editor

Prospects of Endoscopic Ultrasound (EUS) in Bangladesh

Syeda Nur-E- Jannat

To
The Editor
Bangladesh Journal of Gastrointestinal & Liver Disease

Dear Sir,
Through the columns of your esteemed journal, I wish to highlight the growing prospects of Endoscopic Ultrasound (EUS) in Bangladesh- an emerging and indispensable advanced diagnostic and therapeutic modality that is now considered a cornerstone of modern gastrointestinal care worldwide.

Bangladesh bears a substantial burden of hepatobiliary, pancreatic, and upper gastrointestinal malignancies, many of which are diagnosed at advanced stages. EUS plays a pivotal role in early diagnosis, accurate staging, and tissue acquisition through fine-needle aspiration (FNA) and fine- needle biopsy (FNB), particularly for pancreatic malignancies, peripancreatic lymphadenopathy, subepithelial GI lesions, and rectal diseases. From a health-policy perspective, EUS significantly reduces reliance on invasive surgical diagnostics, thereby lowering procedure-related morbidity, hospital stay, and long-term healthcare expenditure.

Beyond diagnostics, therapeutic EUS has transformed the management of complex GI conditions. In cases of failed ERCP due to duodenal infiltration or hilar cholangiocarcinoma, EUS-guided biliary drainage offers a safe and effective alternative to percutaneous transhepatic biliary drainage (PTBD), which is associated with higher complication rates and patient discomfort. Similarly, EUS-guided gastroenterostomy provides a minimally invasive solution for gastric outlet obstruction in high-risk surgical patients, while EUS-guided radiofrequency ablation and celiac plexus neurolysis have significantly improved palliative care outcomes in pancreatic malignancies. These interventions align with global goals of minimally invasive, cost- effective and patient-centered care.

Despite its proven benefits, EUS services in Bangladesh remain severely limited, currently available at only three centers: the National Gastroliver Institute & Hospital (NGLIH), Bangladesh Medical University (BMU) and United Medical College Hospital. While this marks an encouraging start, it falls far short of national demand. Notably, NGLIH performed approximately 700 EUS procedures during 2024–2025, including diagnostic EUS, FNA/FNB, cystogastrostomy, and celiac plexus block—demonstrating both feasibility and growing clinical

need. However, advanced therapeutic EUS procedures are still not routinely practiced in Bangladesh due to high equipment costs, lack of trained manpower and absence of structured fellowship programs.

Recent exposure to international training programs, including participation in an advanced EUS workshop in Hyderabad, India - demonstrated that therapeutic EUS procedures such as biliary drainage, gastroenterostomy, radiofrequency ablation, and variceal coiling are achievable in resource-constrained settings with appropriate planning. This experience reinforced the belief that such procedures are feasible in Bangladesh with appropriate logistical support and structured training programs. Encouragingly, a recent EUS workshop organized by the Bangladesh Gastroenterology Society (BGS) at Bangladesh Medical University (BMU) conducted by an international faculty represent important steps toward capacity building.

From a national policy standpoint, the expansion of EUS services should be prioritized within tertiary and selected secondary healthcare facilities. This requires coordinated action involving government health authorities, professional societies, academic institutions and development partners to establishing structured EUS training and certification programs, developing centers of excellence with stepwise introduction of therapeutic EUS, strengthening cytopathology collaboration for EUS-guided tissue diagnosis and facilitating public-sector investment and public–private partnerships to reduce equipment and maintenance costs.

Wider adoption of EUS would reduce dependence on radiological interventions and palliative surgery, minimize the need for overseas referrals for advanced GI procedure, thereby conserving foreign currency and represent a significant advancement in interventional gastroenterology practice in the country. In this context, your esteemed journal can play a pivotal role in fostering scholarly discussion and awareness regarding the future of EUS in Bangladesh.

Sincerely,

Dr. Syeda Nur-E-Jannat
MBBS (DMC), FCPS (Medicine), MD (Gastroenterology) Associate professor (Gastroenterology),
National Gastroliver Institute & Hospital

Bangladesh Journal of Gastrointestinal and Liver Diseases

An Official Publication of the Bangladesh Gastroenterology Society

Bangladesh Gastroenterology Society

Scientific partner

ZISKA PHARMA