The SSAJM

THE SCHOLARLY SOCIETY OF AMERICA:
JOURNAL OF MEDICINE

VOL. 4
ISSUE 1
ISSN: 2996- 587X

Table of Contents

1. Table of Contents
2. Acknowledgements
3. Executive Board
4. Reviewers
7. Meet an Editor

Commentaries:

8. Looking Back

Image of the Issue:

10. Planetary Polyp

Abstracts:

13. Colitis or Cancer? A Rare Case of Colonic MALT Lymphoma Revealed Through Biopsy
16. Metastatic Ovarian Cancer to the Common Bile Duct: A Case Report
19. Black Esophagus: A Case Report
24. A Case of Malignant Ampullary Ulceration
31. From Eye to Gut: A Melanotic Journey
35. A “MacGiveric” Myotomy for a Gastric Bypass Conundrum

Acknowledgements

On behalf of the SSAJM Executive Team, I would like to extend our deepest gratitude to all our readers, authors, and scholars who continue to support, engage with, and contribute to this journal. Your curiosity, dedication, and commitment to lifelong learning are the driving forces that keep this journal alive and thriving.

Research is more than the pursuit of answers—it is the collective effort to push the boundaries of knowledge, challenge assumptions, and improve the world around us. Every submission, every article read, and every idea shared strengthens the foundation upon which science and medicine progress. Your contributions fuel innovation, inspire future scholarship, and play a vital role in advancing society.

This year has brought its share of challenges and unexpected hardships. Yet, through perseverance, collaboration, and an unwavering devotion to scholarship, we have overcome these obstacles. It is because of your continued support and resilience that we are proud to present this next issue.

Thank you for believing in the power of research, for sharing your work with us, and for helping us continue this mission. We look forward to growing, learning, and discovering alongside you in the issues ahead.
The team would also like to express our heartfelt appreciation to Dr. Edwin Mogda, whose fearless leadership and remarkable technological expertise continue to elevate our journal. His innovation, dedication, and steady guidance remain cornerstones of our success.

We also express our gratitude to Dr. Tejas Nikumbh, whose fun, engaging approach—and the calm wisdom he brings from his parenting skills—have helped keep the journal organized, balanced, and always moving forward.

A special acknowledgment goes to Dr. Amninder Singh, whose wealth of statistical knowledge has been invaluable. Having recently matched into a Cardiology fellowship, he embodies excellence and will undoubtedly become an exceptional Cardiologist.

We are deeply thankful for Nathan Cardona, the true glue of our journal team. His dependability, coordination, and behind-the-scenes effort ensure that every part of the process comes together seamlessly.

Finally, we recognize Dr. Keerthy Gopalakrishnan, a remarkable physician whose professionalism is matched only by her extraordinary personality. Her presence and contributions add depth, warmth, and excellence to our work.

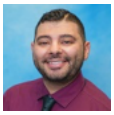
Together, this team represents the spirit, dedication, and collaborative strength that make our journal possible.

Sincerely,

Peter Iskander, MD

Peter Iskander, MD
Co-Founder & Chief Editor

Executive Board



Peter Iskander, MD, HBSc:

Born and raised in Toronto, Ontario, Dr. Iskander completed his medical degree at AUA before joining The Wright Center for GME for his Internal Medicine residency. During that time, he acted as Chief of Scholarly Activity for his program, where he helped establish the SSAJM with his team to promote research and publications for all residents. He is currently a 2nd-year GI Fellow at the same institution. In his free time, he loves to cook/bake and rarely passes up the opportunity for a game of volleyball!

Edwin Mogaka, MD, PhD:

Dr. Edwin Mogaka is a dedicated physician and currently practices at Lehigh Valley Health Network, where he is recognized for his clinical excellence, thoughtful communication, and collaborative approach to healthcare delivery. He is an alumnus of The Wright Center Family Medicine Residency Program, Class of 2024.

In addition to his clinical and academic pursuits, Dr. Mogaka is a founding member of SSAJM, underscoring his commitment to advancing global health scholarship, medical education, and equitable access to high-quality medical knowledge.

Amninder Singh, MD:

Dr. Amninder Singh is a Cardiology Fellow at The Wright Center for GME. He brings with him a wealth of experience, having served in the Indian military and acting as a reviewer for various medical journals. Dr. Singh is passionate about clinical research in Cardiology and actively encourages fellow residents to get involved. He is a founding member of the Editorial Board at SSAJM.

Tejas Nikumbh, MD:

Dr. Tejas Nikumbh is a physician and academic with training from Grant Medical College in Mumbai, India—one of the country's oldest and largest medical institutions. Early exposure to a high-volume government hospital sparked a strong interest in research and evidence-based medicine. He is currently the Chief Resident for Research and Scholarly Activity at The Wright Center for GME in Scranton, Pennsylvania, where he enjoys mentoring residents and advancing scholarly work. He plans to pursue a career as an Academic Hospitalist, with long-term goals centered on clinical excellence, leadership, and mentorship. Outside of medicine, he values time with his family, enjoys long drives, and kayaking as a way to unwind.

Keerthy Gopalakrishnan, MD:

Dr. Keerthy Gopalakrishnan is an internal medicine resident at The Wright Center for GME, with one year of research fellowship training at the Mayo Clinic. Her research interests focus on the application of artificial intelligence in medicine, particularly in the development of diagnostic tools. She is passionate about interlinking technology with patient care to improve healthcare outcomes for both patients and physicians. During her residency, she has served as a scholarly leader, supporting peers in academic development, and is an active member of the research committee within her program. Her work has been published in several peer-reviewed journals and presented at multiple national and international conferences.

Nathan Cardona, MS:

Nathan Cardona is a graduate of the University of Scranton with a background in Occupational Therapy. Nathan serves as the Director of Scholarly Activity, Institutional Research, and IRB Administration at The Wright Center for GME and has a passion for a wide range of medical research activities.

Reviewers

CARDIOLOGY:

Dr. Samir Pancholy, MD, MSCAI, FAHA, FACC, FACP:

Dr. Pancholy completed his Internal Medicine residency at SUNY Stony Brook and his general and interventional cardiology fellowship at Presbyterian Medical Center in Philadelphia. He is a certified specialist in advanced heart failure and transplant cardiology. He currently serves as the Program Director for the Cardiology Fellowship at The Wright Center for Graduate Medical Education. He is also a faculty member at Geisinger School of Medicine and the director of Cardiac Catheterization at Wilkes-Barre VA Hospital. Over his distinguished career, he has 70+ US patents, led multiple RCTs, and is one of a select few honored as a Master of SCAI for innovation and advancement in Interventional Cardiology.

Dr. Tapan Buch, MD:

Dr. Buch completed his residency and Cardiology training at The Wright Center for Graduate Medical Education. He is an Adult Non-invasive cardiologist in the Northwell Health system, where he focuses on prevention and lifestyle modifications, cardiac imaging, vascular/venous disease, and education. He has a particular interest in advancing the integration of emerging cardiac technology.

GERIATRICS:

Dr. Edward Dzielak, MD:

Dr. Edward J. Dzielak is dual board-certified in Internal and Geriatric Medicine. He currently serves as the Program Director for the Geriatrics fellowship at The Wright Center for Graduate Medical Education. He earned his medical degree from the Philadelphia College of Osteopathic Medicine medical school and is also a graduate of the Scranton Temple Residency Program (now The Wright Center).

Dr. Tanureet Kochar, MD:

Born and raised in India, Dr. Tanureet Kochar moved to the United States, where she completed her Internal Medicine residency at the Charleston area Medical Center in Charleston, West Virginia. She went on to pursue further training in Geriatrics and Sleep Medicine at the Detroit Medical Center in Michigan! She is a current faculty member with The Wright Center for Graduate Medical Education. Her interests include dementia care, elder abuse, home care for the elderly, and improving the quality of life in older adults. Aside from working, she enjoys dancing, cooking, and all aspects of being a mom.

Dr. Nirali Patel, MD:

Dr. Nirali Patel is a board-certified Internal Medicine and board-eligible geriatric physician. She earned her medical degree from the Medical University of Lublin, Poland, and completed her Internal Medicine residency and Geriatrics Fellowship training at The Wright Center for Graduate Medical Education. She is a current Associate Program Director of the Geriatrics Fellowship Program and core faculty of the Internal Medicine Residency Program.

INFECTIOUS DISEASE:

Dr. Gary Decker, MD:

Dr. Gary Decker is a board-certified Infectious Disease specialist who completed his Fellowship training at Georgetown University in Washington, DC. With over 30 years of wealth of knowledge and experience, he helps in the management and treatment of various diseases in the Scranton region of Pennsylvania.

Dr. Mary Louise Decker, MD:

Dr. Mary Louise Decker is a board-certified physician currently serving as an Infectious Disease Medical Director for The Wright Center for Community Health. Her duties include managing The Wright Center's Ryan White HIV and Infectious Disease/Hepatitis-C Clinic. She completed her residency in internal medicine and her fellowship at Georgetown University School of Medicine, Washington, D.C.

Dr. Pragya Dhaubhadel, MD:

Dr. Dhaubhadel is a board-certified and fellowship-trained specialist in Infectious Diseases. Her clinical and research interests include antimicrobial stewardship, Hepatitis-C virus infection, and HIV. She earned her medical degree from Lady Hardinge Medical College in India and then moved to the United States, where she completed her residency and a fellowship in infectious diseases at Harlem Hospital Center.

INTERNAL MEDICINE:

Dr. Milos Babic, MD:

Dr. Babic is a board-certified Internal Medicine physician practicing primary care and hospitalist medicine in Scranton, PA. After completing his medical degree from the University of Belgrade Medical School in Serbia, he traveled to NYC to undergo residency training at the Icahn School of Medicine at Mount Sinai, where he served as Chief Resident.

Dr. Douglas Klamp, MD:

Dr. Douglas Klamp is a board-certified Internal Medicine physician and the current program director of the Internal Medicine Residency Program at The Wright Center for Graduate Medical Education. Dr. Klamp earned his bachelor's degree in biology from Pennsylvania State University and graduated from Johns Hopkins School of Medicine. He completed his residency in primary care Internal Medicine at Alameda County General Hospital in Oakland, California.

Dr. Erin McFadden, MD:

Dr. McFadden is a board-certified Internal Medicine physician in Scranton, PA. She completed her medical degree at Temple University School of Medicine. She currently serves as a medical director at The Wright Center for Community Health.

Dr. Roop Parlapalli, MD:

After obtaining his medical degree from Sri Venkateswara Medical College, Dr. Parlapalli went on to complete his residency at The Wright Center for Graduate Medical Education in Internal Medicine. He currently works as an academic hospitalist at the Geisinger Community Medical Center in Scranton, PA, where he is involved in teaching medical students and residents. He is also an active member of multiple medical societies, including, but not limited to, ACP, SHM, and SGIM. His fields of interest entail clinical reasoning, medical education, and hospital medicine.

LIFESTYLE MEDICINE / NUTRITION:

Walter Wanas, RDN:

Walter is a registered and licensed dietitian in Scranton, PA. He currently serves as the Director of Lifestyle Modification and Preventive Medicine for The Wright Center for Community Health. He is a graduate of Pennsylvania State University and Marywood University's dietetic certification program. Walter works closely with fellow Wright Center care teams to help address and improve long-term care and reverse chronic medical conditions.

NEUROSURGERY / NEURO-ONCOLOGY:

Dr. Michael Karsy, MD, PhD, MSc:

Dr. Karsy is a neurosurgeon who has a special interest in cranial skull base and spine neurosurgeries. He obtained his medical degree from the New York Medical College and completed a Neurosurgery residency at the University of Utah. He went on to further his education to pursue a Fellowship in Minimally Invasive and open skull base surgery at Jefferson Medical Center. He also serves as an assistant professor of neurosurgery at Drexel University College of Medicine in Philadelphia, PA.

NEPHROLOGY:

Dr. John Prior, DO:

After completing his residency at the Philadelphia College of Osteopathic Medicine, Dr. Prior went on to obtain a Fellowship in Nephrology from Hahnemann University Hospital. He currently provides quality care in the greater Scranton area of Pennsylvania.

PULMONARY / CRITICAL CARE:

Dr. Nikul Patel, MD

Dr. Patel is a graduate of Albert Einstein Medical Center in Philadelphia, where he completed his residency and went further to pursue a Fellowship in Critical Care and Pulmonary Medicine. He currently helps serves in the Scranton region.

RHEUMATOLOGY:

Dr. Nevena Barjaktarovic, MD

Dr. Barjaktarovic is a dual board-certified physician in Internal Medicine and Rheumatology, providing diagnosis and treatment for a wide range of rheumatic and inflammatory conditions. She obtained her medical degree at Belgrade University, Serbia, and completed her residency in Internal Medicine at Icahn School of Medicine. She went further to pursue her Fellowship in Rheumatology at Albert Einstein College of Medicine-Montefiore Medical Center in the Bronx.

Dr. Tejas Nikumbh

Meet an Editor

Where did you complete your education?

I graduated from Grant Medical College in Mumbai, India. It is one of the oldest and largest medical colleges in the country, established back in 1845. Training in such a massive, high-volume government hospital gave me exposure to a vast range of medical conditions early on. This was also where I first got involved in research as a medical student, realizing early that I wanted to understand the evidence behind clinical practice.

Current pursuits and ruture aspirations?

I am currently serving as the Chief Resident for Research and Scholarly Activity at The Wright Center for GME in Scranton, PA. It's a role I really enjoy because it lets me combine my own interest in research with mentoring other residents. After graduation, I plan to work as an Academic Hospitalist. My long-term goal is to strike a balance between clinical care, leadership, and mentorship.

What got you into research?

Initially, it was just about learning the process. But over the years, especially after practicing as a surgeon and then transitioning to medicine, my perspective changed. I realized that clinical experience alone has limits. Research is the tool we use to question whether 'the way we've always done it' is actually the best way. It keeps me humble and ensures I am constantly looking for better answers for my patients.

Favorite hobbies!

To be honest, my favorite hobby right now is simply being a dad. I prioritize cherishing the simple moments with my family. I also enjoy going on long drives and kayaking once in a while to unwind.

Any Career Advice for Our Readers?

Persevere and don't be afraid to pivot. My path wasn't linear—moving from surgery to internal medicine taught me a lot about adaptability. Never underestimate the power of mentorship, and remember that it is good to step out of your comfort zone. That is usually where the real growth happens.

LOOKING BACK

The ray of sunlight gently announces its presence through the window, timidly inviting itself into my room. Asking to be a companion of my solitude with a meekness only the morning light can be capable of. A light not yet burdened by the demands of the day that lies ahead. With silence, it cast a soft shadow on the few pill containers by the window. I humbly accepted the invitation from the sun into my small fifth-story room overlooking the river.
The sun felt the need to warm the coldness that radiated from those bottles, and the darkness of the half-drawn curtains was not a deterrent to this noble mission. Outside, all I could hear was a disconnected symphony of sounds from monitors trying - and failing - to harmonize the passionate yelling of my neighbor. He was remembering the days when he had served in the war, not so long ago. Or maybe it is now?

My tired eyes had seen the sun rise and set on eight and a half decades of existence. Its beauty and ugliness intertwined into one inseparable narrative, littered with laughter and tears as numerous as sand on the beach. I remember the losses for which time didn't stop when I wanted it to, and when it raced by, the happy moments that I had wished to last forever.

My gaze caught my reflection in the window, and I saw the marks of the stories that I have lived through on the body that has been my home and constant companion since the day of my birth. My deep-set brown eyes overflowed with tears of joy and sadness on more days than I can count. The eyes looking back now have gained wisdom through the trials of life more than they ever wished to have gained. A life of experience that has come at a price. These eyes have also witnessed the life stories of many with their triumphs and tragedies alike.

But also in those eyes, I see courage to have made it through decades of uncertainty. In the window's reflection, the woman looking back at me starts to smile as she remembers the little joys of the past that have brought her to where she is now. The moments of tender touch and heartfelt tears that once made her feel alive now give her strength to stand tall today. She looks back at that reflection with pride, knowing how many battles she has fought and won.

As the day slowly draws to a close, the shy, timid sunlight that welcomed the morning retreats, making way for the evening to unfold. It slowly bids the day farewell, knowing of its inevitable return the next morning. The hustle of feet rushing from one place to the next begins to quiet with reluctance, knowing the many tasks that have been left unfinished with the fading daylight. Conversations left unsaid, documents left unwritten, and emotions not yet processed by the hearts that bear them. The one constant in the background is the coldness of the fluorescent lights, becoming more pronounced as day fades into night.

Just outside my door, I overhear a conversation between two young resident doctors thankful to have made it through another stressful day in the ICU and afraid of what the darkness of the night may bring. Their voices, shrouded in hesitation and worry, were barely masked by the neutrality of their words. They prefer to face the setting sun in my window than to see each other's eyes and acknowledge the uneasiness that lies between them. They gaze longingly at the sun, aware that they need its warmth and companionship now more than at any other time of day.

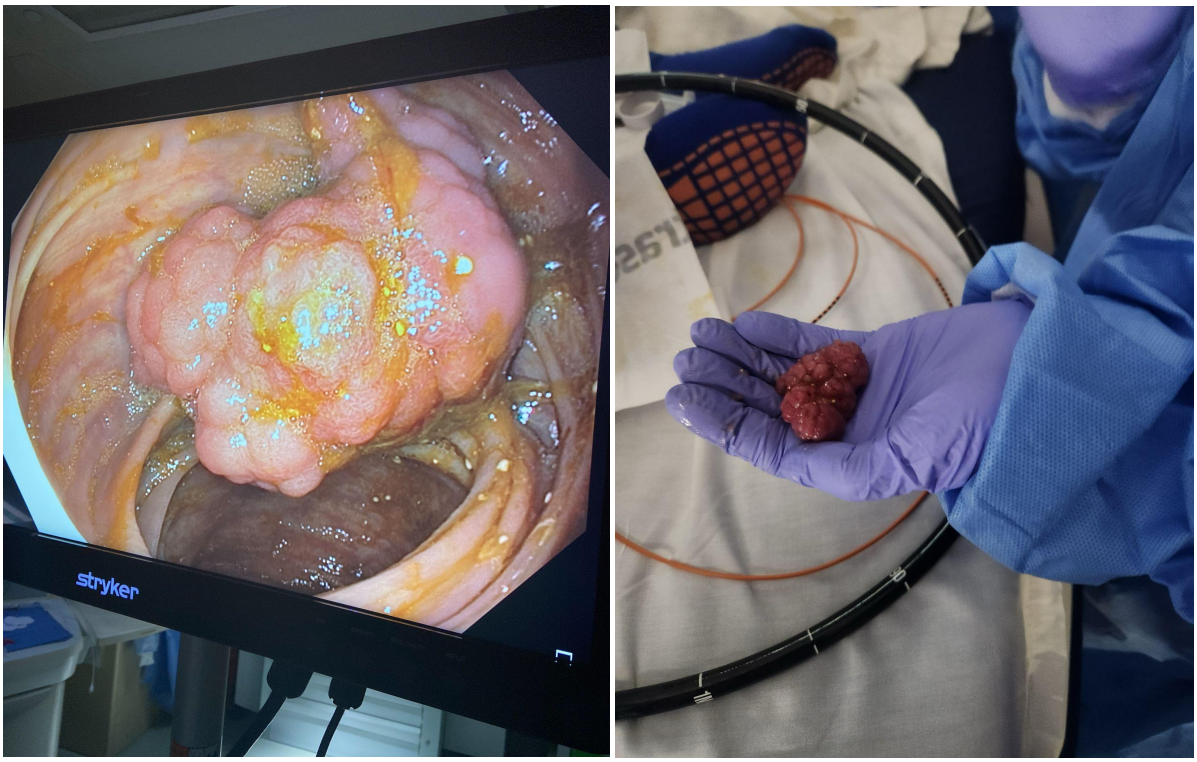
A few minutes pass, and their reflections become more evident on the hospital's cold glass windows as the darkness settles in. Each resident takes a few moments to familiarize themselves with this new reflection of who they are. A reflection of a person that they did not know this morning. The passing day has taught them new ideas and events that have now become a permanent part of their memory and who they are. The acknowledgement of the many changes that one day can bring begins to settle, and a new reality unfolds. They slowly begin to smile as the evening sky boldly reveals its deep purple hues, allowing them to momentarily forget the endless words and thoughts that have floated through their minds today. The beauty of the evening sky was a moment needed by all of us to remember that outside these walls, there is light.

Dr. Monica Ghebrial
Internal Medicine PGY-2
Department of Internal Medicine
University of Manitoba

Planetary Polyp

Siefeen-Ghattas, Mariez MS [1]; Abdelmalek, Karim [2]; Tahir, Hafsa MD [3], Kumar, Sunny MBBS [4]; Iskander, Stephen [5]

1. Lake Erie College of Osteopathic Medicine, Elmira, NY
2. St. George's University, West Indies, Grenada
3. Providence Alaska Medical Center, Anchorage, Alaska
4. The Wright Center for GME, Scranton, PA
5. York University, Ontario, Canada



What is a Polyp?

Colorectal polyps are abnormal tissue growths that protrude from the lining (mucosa) of the colon or rectum into the intestinal lumen. Polyps are classified morphologically based on their size, shape, color, and histopathology, particularly in relation to their malignancy potential and associated cancer risk [1]. Globally, colorectal polyps are the second leading cause of cancer-related mortality [2], with a rising incidence among males and individuals over the age of 45 [3].

The most commonly observed pathway for colorectal cancer (CRC) development involves genetic mutations that drive abnormal cell proliferation. This includes the adenoma-carcinoma sequence, characterized by mutations in the APC tumor suppressor gene, and the serrated polyp-carcinoma sequence, involving MLH1 promoter hypermethylation that silences tumor suppressor genes [4]. Due to the potential for malignant transformation of various polyp types, colonoscopy with polypectomy remains the primary strategy for CRC prevention [5].

Types of Polyps:

Tubular adenomas, which include all non-serrated lesions, vary in risk depending on their size and histological features. Adenomas measuring at least 10 mm in diameter or exhibiting tubulo-villous or villous architecture, or high-grade dysplasia, are considered high-risk for neoplastic progression. In such cases, repeat colonoscopy is recommended within three years. In the absence of high-risk features, the recommended follow-up interval depends on the number of adenomas identified. For individuals with one to two tubular adenomas, a surveillance colonoscopy is advised after seven to ten years. For those with three to four adenomas, a follow-up interval of three to five years is recommended. When five to ten adenomas are present, the risk of neoplasia increases, warranting surveillance at three years. If more than ten adenomas are found, a hereditary polyposis syndrome is likely, and a repeat colonoscopy is advised within one year. One study reported that individuals with more than ten adenomas had approximately a 25% chance of developing advanced neoplasia within four years [6].

Sessile serrated polyps (SSPs) are considered high-risk when they are 10 mm or larger or demonstrate dysplasia, and in such cases, a three-year follow-up colonoscopy is recommended. When these features are absent, surveillance intervals vary based on polyp burden. For one to two SSPs, a five- to ten-year follow-up interval is advised. If three to four SSPs are detected, colonoscopy should be repeated in three to five years, while five to ten SSPs warrant surveillance within three years.

Traditional serrated adenomas (TSAs) carry a higher neoplastic risk than tubular adenomas. Due to their malignant potential, the recommended follow-up after identification is a repeat colonoscopy in three years.

Hyperplastic polyps, particularly those smaller than 10 mm, are generally benign and may be reported as part of a normal colonoscopy. However, if a hyperplastic polyp is 10 mm or larger, a three- to five-year follow-up colonoscopy is recommended due to the elevated neoplasia risk associated with large serrated lesions. The presence of more than 20 hyperplastic polyps may indicate serrated polyposis syndrome, a high-risk condition requiring individualized follow-up strategies. Conversely, individuals with fewer than 20 hyperplastic polyps, all smaller than 10 mm, are considered low risk for neoplasia [7].

For individuals with a high-risk family history, surveillance recommendations are more aggressive. Colonoscopy every five years is advised for those with either colorectal cancer or an advanced adenoma in two first-degree relatives diagnosed at any age, or in one first-degree relative diagnosed before the age of 60. Screening should begin at age 40, or ten years before the earliest diagnosis in the family—whichever occurs first. If a first-degree relative was diagnosed at age 60 or older, colonoscopy should commence at age 40, with follow-up according to average-risk screening protocols. In cases where a second-degree relative has been diagnosed with colorectal cancer or an advanced polyp, standard average-risk CRC screening recommendations should be followed.

Guidelines for Polyp Follow-Up:

Findings from a screening colonoscopy play a crucial role in determining an individual's future risk of colorectal cancer and guiding the timing of repeat examinations. The U.S. Multi-Society Task Force has established updated recommendations for surveillance intervals following initial screening colonoscopy in individuals at average risk. In the case of a normal colonoscopy, which may include the presence of hyperplastic polyps smaller than 10 mm but no other significant abnormalities, the Task Force

recommends a follow-up colonoscopy in 10 years. Although direct evidence supporting this interval is limited, the recommendation is based on the low likelihood of progression to colorectal cancer in the absence of high-risk findings [8].

References:

1. Meseeha M, Attia M. Colon Polyps [Internet]. PubMed. Treasure Island (FL): StatPearls Publishing; 2020. Available from: https://www.ncbi.nlm.nih.gov/books/NBK430761/
2. Sullivan BA, Noujaim M, Roper J. Cause, Epidemiology, and Histology of Polyps and Pathways to Colorectal Cancer. Gastrointestinal Endoscopy Clinics of North America. 2022 Feb;32(2):177–94.8
3. Definition & Facts for Colon Polyps [Internet]. National Institute of Diabetes and Digestive and Kidney Diseases. NIDDK - National Institute of Diabetes and Digestive and Kidney Diseases; 2025 [cited 2025 Mar 31]. Available from: https://www.niddk.nih.gov/health-information/digestive-diseases/colon-polyps/definition-facts/?utm_source=chatgpt.com#what
4. Tang J, Lam GT, Brooks RD, Miles M, Useckaite Z, Johnson IRD, et al. Exploring the role of sporadic BRAF and KRAS mutations during colorectal cancer pathogenesis: A spotlight on the contribution of the endosome-lysosome system. Cancer Letters. 2024 Mar;585:216639.
5. Patel VV, Bechara R, Rai M. Dysplasia and Malignancy in Colonic Polyps: Preparing for a Resect and Discard Strategy in Canada. JGH Open. 2025 Mar;9(3).
6. Am Fam Physician. 2021;103(5):314-316
7. Gupta S, Lieberman D, Anderson JC, Burke CA, Dominitz JA, Kaltenbach T, Robertson DJ, Shaukat A, Syngal S, Rex DK. Recommendations for Follow-Up After Colonoscopy and Polypectomy: A Consensus Update by the US Multi-Society Task Force on Colorectal Cancer. Am J Gastroenterol. 2020 Mar;115(3):415-434. doi: 10.14309/ajg.0000000000000544. PMID: 32039982; PMCID: PMC7393611.
8. Shaukat A, Kahi CJ, Burke CA, Rabeneck L, Sauer BG, Rex DK. ACG Clinical Guidelines: Colorectal Cancer Screening 2021. Am J Gastroenterol. 2021 Mar 1;116(3):458-479. doi: 10.14309/ajg.0000000000001122. PMID: 33657038.

Colitis or Cancer? A Rare Case of Colonic MALT Lymphoma Revealed Through Biopsy

Awad, Monica MS [1]; Shaker, Ramez MS [2]; Samaan, Bishoy MS [3]; Awad, Youana BHSc [4]; Mekhaeil, Selena [4]; Salama, Carol MS [5]; Siefeen-Ghattas, Mariez MS [6]; Sunny, Kumar MBBS [7]

1. University of Galway School of Medicine, Galway, Ireland
2. University College Cork School of Medicine, Cork, Ireland
3. Saint George's University School of Medicine, Grenada, West Indies
4. Wilfrid Laurier University, Waterloo, ON
5. University of Ottawa School of Medicine, Ottawa, ON
6. Lake Erie College of Medicine, Elmira, NY
7. The Wright Center for GME, Scranton, PA

Abstract:

Colonic mucosa-associated lymphoid tissue (MALT) lymphoma is an uncommon subtype of extranodal marginal zone non-Hodgkin lymphoma that may clinically and endoscopically mimic inflammatory bowel disease (IBD), leading to significant diagnostic delays. Although gastric MALT lymphoma is well characterized, colonic involvement is rare and accounts for only a small proportion of cases worldwide. We present an expanded case of a 77-year-old male who presented with chronic diarrhea, profound weight loss, and colonic inflammation initially misinterpreted as ulcerative colitis. Histopathologic evaluation ultimately demonstrated a monoclonal lambda-restricted B-cell infiltrate consistent with low-grade MALT lymphoma. Staging with PET/CT revealed widespread low-grade lymphadenopathy consistent with Ann Arbor Stage III disease. This report underscores the importance of thorough diagnostic evaluation when endoscopic findings resemble IBD, particularly in older adults, and highlights current evidence-based approaches to diagnosis, staging, and management of colonic MALT lymphoma [1,4].

Introduction:

Mucosa-associated lymphoid tissue (MALT) lymphoma is an indolent, extranodal B-cell lymphoma arising from the marginal zone of mucosal lymphoid tissue. It accounts for approximately 8% of all non-Hodgkin lymphomas, with the stomach representing the most common site due to chronic Helicobacter pylori–related antigenic stimulation [2]. Colonic MALT lymphoma, however, is exceedingly rare, comprising only 2–3% of all MALT lymphomas and less than 0.5% of all gastrointestinal lymphomas [3]. The pathogenesis often involves chronic antigenic stimulation, autoimmune conditions, or underlying inflammatory states, although many colonic cases remain idiopathic. Clinically, colonic MALT lymphoma may present with nonspecific symptoms such as diarrhea, abdominal discomfort, hematochezia, or weight loss—features that often overlap with ulcerative colitis and Crohn's disease [5]. Endoscopic findings similarly lack specificity and may include mucosal ulcerations, nodularity, friability, or diffuse inflammation. As a result, misdiagnosis is common.

Accurate diagnosis requires a combination of histopathology, immunophenotyping, molecular studies, and cross-sectional imaging. The hallmark histologic finding is a dense infiltrate of small-to-medium B

cells demonstrating light-chain restriction and positivity for markers such as CD20, CD79a, and BCL-2, with negative expression of CD5, CD10, and cyclin D1, distinguishing MALT lymphoma from mantle cell lymphoma, follicular lymphoma, and chronic lymphocytic leukemia [3,6]. Given its rarity and potential for delayed recognition, increased clinical awareness is essential.

Case Presentation:

A 77-year-old man with a medical history significant for prostate cancer, type 2 diabetes mellitus, hyperlipidemia, Barrett's esophagus, and previously documented rectosigmoid MALT lymphoma presented with a several-month history of worsening fatigue, dehydration, confusion, and diarrhea occurring 4–5 times daily, including nocturnal episodes. He also reported a 40-pound unintentional weight loss. Laboratory evaluation revealed mild anemia, hypoalbuminemia, and elevated inflammatory markers. CT imaging of the abdomen demonstrated diffuse colonic wall thickening and mesenteric lymphadenopathy. Infectious workup, including Clostridioides difficile PCR, was negative.

Colonoscopy showed severe, continuous colonic inflammation characterized by ulcerations, friability, and erythema, initially suggesting ulcerative colitis. Empiric therapy with mesalamine and prednisone was initiated. However, biopsies obtained from multiple colonic segments demonstrated focal acute cryptitis with dense, atypical lymphoid infiltrates involving the lamina propria. Immunohistochemistry revealed B-cell markers CD20+, CD79a+, BCL-2+ with lambda light-chain restriction and absence of CD5, CD10, BCL-1 (cyclin D1), and BCL-6—findings consistent with low-grade MALT lymphoma [4]. The Ki-67 proliferation index was low (~10%), further supporting an indolent lymphoma.

Flow cytometry identified a monoclonal B-cell population consistent with marginal zone lymphoma. Additional testing for MYD88 L265P mutation was negative, making lymphoplasmacytic lymphoma less likely. PET/CT demonstrated mild-to-moderate hypermetabolic lymphadenopathy across mesenteric, retroperitoneal, and thoracic nodes, compatible with Ann Arbor Stage III disease. The patient was referred to hematology/oncology and initiated on rituximab-based therapy, with consideration for bendamustine depending on therapeutic response.

Discussion:

Colonic MALT lymphoma represents a diagnostic challenge because clinical and endoscopic presentations often mimic more common inflammatory conditions such as ulcerative colitis. The patient's presentation—chronic diarrhea, nocturnal symptoms, significant weight loss, and diffuse colitis—strongly resembled IBD, leading to initial misclassification. Such overlap has been described in several case series and reviews, where MALT lymphoma presented as multifocal ulcerations or diffuse mucosal inflammation indistinguishable from IBD [5,7].

Histopathologic evaluation remains the cornerstone of diagnosis. The presence of small atypical B cells infiltrating the lamina propria, along with immunophenotypic findings (CD20+, CD79a+, CD5–, CD10–, cyclin D1–), helps distinguish MALT lymphoma from other B-cell neoplasms [3,6]. Light-chain restriction further confirms clonality. In some cases, molecular studies demonstrating rearrangement of the MALT1 gene or API2-MALT1 fusion may assist diagnosis, although such findings are less common in colonic cases [2].

Staging with PET/CT is essential because extranodal marginal zone lymphoma can involve multiple mucosal sites, the lymphatic system, and bone marrow. Disseminated disease, as in this patient, does not preclude favorable outcomes due to the generally indolent course of MALT lymphoma.

Management typically depends on disease localization and symptom severity. Localized gastric MALT lymphoma may respond to H. pylori eradication alone, but colonic lesions generally require systemic therapy. Rituximab monotherapy or rituximab combined with alkylating agents such as bendamustine demonstrates high response rates with acceptable toxicity profiles [8]. Radiotherapy may be considered for localized disease, though multifocal colonic involvement commonly necessitates systemic treatment. Prognosis is generally favorable, with 5-year survival rates exceeding 80% for most MALT lymphomas despite the potential for disseminated disease [2,8].

Conclusion:

This case highlights the importance of considering lymphoid malignancy in older adults presenting with atypical or treatment-refractory colitis. Because colonic MALT lymphoma is rare and may mimic IBD, definitive diagnosis relies on histopathology, immunophenotyping, and comprehensive staging. Early recognition ensures timely oncologic therapy and prevents delays caused by inappropriate treatment for presumed inflammatory disease. Continued awareness and reporting of colonic MALT lymphoma are essential to improve diagnostic accuracy and optimize patient outcomes.

References:

1. Abbas, H., Niazi, M., and Makker, J. “Mucosa-Associated Lymphoid Tissue (MALT) Lymphoma of the Colon: A Case Report and a Literature Review.” American Journal of Case Reports, 2017.
2. Filip, P. V., et al. “MALT Lymphoma: Epidemiology, Clinical Diagnosis and Treatment.” Journal of Medicine and Life, 2018.
3. Jeon, M. K., et al. “Endoscopic Features and Clinical Outcomes of Colorectal MALT Lymphoma.” Gastrointestinal Endoscopy, 2018.
4. Silva, V. M., et al. “Multifocal Colonic Ulcerations: An Unusual Presentation of MALT Lymphoma.” GE – Portuguese Journal of Gastroenterology, 2022.
5. Lee, Y. G., et al. “Multiple MALT Lymphoma of the Colon Identified as Simple Mucosal Discoloration.” Journal of Korean Medical Science, 2005.
6. Zucca, E., et al. “Marginal Zone Lymphomas: ESMO Clinical Practice Guidelines.” Annals of Oncology, 2020.
7. Zullo, A., et al. “Gastrointestinal Lymphoma: The Evolving Role of Endoscopy.” World Journal of Gastroenterology, 2012.
8. Raderer, M., and Kiesewetter, B. “Management of Extranodal Marginal Zone Lymphoma of MALT Type.” British Journal of Haematology, 2021.

Metastatic Ovarian Cancer to the Common Bile Duct: A Case Report

Iskander, Andrew MB, BCh, BAO [1]; Salama, Carol MS [2]; Phlamon, Juliana MS [3]; Rofaeel, Karen MS [4]; Awad, Monica MS [5]; Kumar, Sunny MBBS [6]

1. Western University, London, Ontario
2. University of Ottawa, School of Medicine, Ontario, Canada
3. University College Dublin, School of Medicine, Dublin, Ireland
4. University of British Columbia, Faculty of Medicine, Vancouver, Canada
5. University of Galway, School of Medicine, Galway, Ireland
6. The Wright Center for GME, Scranton, PA

Abstract:

This case report details the clinical course of an 81-year-old female with a history of ovarian cancer treated with chemotherapy, who subsequently developed metastatic disease involving the pancreatic head and distal common bile duct (CBD), leading to malignant biliary obstruction. Despite initial intervention with endoscopic retrograde cholangiopancreatography (ERCP) and stent placement, the patient experienced disease progression, presenting with symptoms indicative of advanced disease and systemic involvement. This report underscores the challenges in managing metastatic ovarian cancer with atypical metastatic patterns and highlights the importance of a multidisciplinary approach in palliative care.

Introduction:

Ovarian cancer is known for its tendency to spread within the peritoneal cavity, leading to a range of complications. However, distant metastases, such as those involving the pancreas and bile ducts, remain rare. When malignant biliary obstruction arises in the context of ovarian cancer, it presents unique diagnostic and treatment challenges. This case underscores the complexity involved in managing such conditions and highlights the importance of personalized patient care strategies.

Clinical Course:

An 81-year-old female with a history of ovarian cancer treated with chemotherapy presented one month following her last chemotherapy dose with painless jaundice and weight loss. Laboratory tests revealed hyponatremia (129 mmol/L), hypokalemia (3.1 mmol/L), alkaline phosphatase (737 U/L), AST (83 U/L), ALT (90 U/L), and bilirubin (14.2 mg/dL). CT chest was notable for worsening bilateral pleural effusion, multiple lung nodules, and an ill-defined lesion in the pancreatic head and distal CBD. Dilated GB wall thickening without obvious cholelithiasis, pericholecystic fluid, and dilated intra/extrahepatic ducts were also reported. ERCP was performed with CBD and pancreatic duct stent placement. Biopsy of these lesions confirmed metastatic adenocarcinoma in the periampullary region, consistent with ovarian cancer.

Subsequently, the patient presented back to the hospital with 15lb weight gain, upper extremity edema, abdominal distension, and worsening dyspnea. Laboratory values showed worsening leukocytosis (15

cells/µL), hyponatremia (127mmol/L), and further elevation of liver enzymes. Repeat CT imaging confirmed peri-splenic ascites, anasarca, patent stents, and resolution of previous biliary dilation. A multidisciplinary discussion was had with the patient and family regarding possible interventions/treatment, along with the risks/benefits of each; a collaborative decision was made at that time to pursue hospice care.

Discussion:

Ovarian Cancer Overview:

Ovarian cancer originates in the ovaries, fallopian tubes, or peritoneum and is often asymptomatic in early stages. Common symptoms include abdominal distension, pain, bloating, early satiety, urinary urgency, vaginal bleeding, etc [1]. Metastatic ovarian cancer typically spreads via peritoneal dissemination, lymphatics, or hematogenous routes, with common sites including the peritoneum, liver, lungs, and bowel [2]. Bile duct involvement, however, is exceedingly rare, with only a few reported cases in the literature [3].

Mechanisms of Metastasis to the Biliary System:

Metastatic spread to the biliary system is uncommon but can occur through direct invasion, lymphatic spread, or hematogenous dissemination [4]. In this case, the presence of a pancreatic head lesion and distal CBD involvement suggests lymphatic or hematogenous spread. Malignant biliary obstruction typically presents with painless jaundice, pruritus, dark urine, pale stools, and abnormal liver function tests, symptoms of which were noted in our patient [5].

Imaging and Diagnosis:

CT, MRI, and MRCP are essential for characterizing biliary obstruction and detecting metastatic lesions [6]. In this patient, CT findings demonstrated an ill-defined pancreatic head lesion, biliary dilation, GB wall thickening, and ascites, supporting a diagnosis of malignant biliary obstruction secondary to metastatic ovarian cancer. ERCP is an advanced endoscopic intervention with the capability of addressing malignant and benign biliary obstructions. [7]

Management Challenges and Prognosis:

Biliary stenting via ERCP is a standard palliative intervention for malignant biliary obstruction. Advancement of disease, however, often necessitates further interventions such as percutaneous transhepatic biliary drainage (PTBD) or permanent metal stenting [8]. Prognosis for metastatic ovarian cancer is poor, especially if there is biliary involvement, with an estimated 30% 5-year survival [3]

Conclusion:

This case report highlights a rare presentation of metastatic ovarian cancer involving the pancreatic head and distal CBD. Although ERCP provided initial symptom relief, disease progression underscored the challenges of managing metastatic biliary obstruction. Early recognition, personalized treatment plans, and comprehensive palliative care remain critical in improving patient outcomes. Future research should further investigate the mechanisms of biliary metastasis and novel therapeutic strategies for this rare manifestation of ovarian cancer.

References:

1. National Cancer Institute, no date, 'Definition of ovary - NCI Dictionary of Cancer Terms', *National Cancer Institute*. Available at: https://www.cancer.gov/publications/dictionaries/cancer-terms/def/ovary [Accessed 1 March 2025].
2. Lengyel E. Ovarian cancer development and metastasis. Am J Pathol. 2010 Sep;177(3):1053-64. doi: 10.2353/ajpath.2010.100105. Epub 2010 Jul 22. PMID: 20651229; PMCID: PMC2928939.
3. Shijo M, Fukase K, Ohtsuka H, Ariake K, Masuda K, Ishida M, Mizuma M, Nakagawa K, Hayashi H, Morikawa T, Motoi F, Naitoh T, Unno M. Metastasis of ovarian cancer to the bile duct: a case report. Surg Case Rep. 2019 Jun 20;5(1):100. doi: 10.1186/s40792-019-0659-9. PMID: 31222668; PMCID: PMC6586734.
4. Halkia E, Spiliotis J, Sugarbaker P. Diagnosis and management of peritoneal metastases from ovarian cancer. Gastroenterol Res Pract. 2012;2012:541842. doi: 10.1155/2012/541842. Epub 2012 Jul 19. PMID: 22888339; PMCID: PMC3408715.
5. Coucke EM, Akbar H, Kahloon A, Lopez PP. Biliary Obstruction. 2022 Nov 26. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan–. PMID: 30969520.
6. Olthof SC, Othman A, Clasen S, Schraml C, Nikolaou K, Bongers M. Imaging of Cholangiocarcinoma. Visc Med. 2016 Dec;32(6):402-410. doi: 10.1159/000453009. Epub 2016 Dec 6. PMID: 28229074; PMCID: PMC5290452.
7. Cacaci M, De Maio F, Matteo MV, Posteraro B, Di Vito M, Menchinelli G, Tringali A, Monzo FR, Torelli R, Costamagna G, Spada C, Bugli F, Sanguinetti M, Boskoski I. Pilot study on cultural and metagenomic analysis of bile and biliary stentslead to unveiling the key players in stent occlusion. Sci Rep. 2024 Feb 9;14(1):3344. doi: 10.1038/s41598-024-51480-2. PMID: 38336904; PMCID: PMC10858256.
8. Nikolić I, Radić J, Petreš A, Djurić A, Protić M, Litavski J, Popović M, Kolarov-Bjelobrk I, Dragin S, Popović L. The Clinical Benefit of Percutaneous Transhepatic Biliary Drainage for Malignant Biliary Tract Obstruction. Cancers (Basel). 2022 Sep 26;14(19):4673. doi: 10.3390/cancers14194673. PMID: 36230596; PMCID: PMC9563508.

Black Esophagus: A Case Report

Samaan, Bishoy [1]; Tahir, Hafsa MD [2]; Mikhaiel, Elarea MS [3]; Fahim, Mark MS [4]; Maher, Miray BHSc [5]; Awad, Youana BHSc [6]; Kumar, Sunny MBBS [7]

1. Saint George's University School of Medicine, Grenada, West Indies
2. Providence Alaska Medical Center, Anchorage, Alaska
3. University of Central Lancashire, Preston, England
4. American University of the Caribbean, St. Maarten, Lesser Antilles
5. Temerty Faculty of Medicine, Toronto, ON
6. Wilfrid Laurier University, Waterloo, Ontario
7. The Wright Center for GME, Scranton, PA

Abstract:

Acute esophageal necrosis (AEN), or "black esophagus," is a rare and life-threatening condition characterized by diffuse circumferential black discoloration of the esophageal mucosa. This case report discusses a 71-year-old male with multiple comorbidities who presented with gastrointestinal bleeding and was subsequently diagnosed with AEN. The report highlights the clinical presentation, diagnostic findings, management strategies, and a review of relevant literature.

Introduction:

AEN is an uncommon clinical entity with an estimated prevalence ranging from 0.01% to 0.2% in autopsy studies [1]. It is characterized by a striking black appearance of the esophageal mucosa on endoscopy, typically involving the distal esophagus and abruptly stopping at the gastroesophageal junction [1]. The etiology is multifactorial, often involving a combination of ischemic insult, gastric acid injury, and systemic conditions leading to hemodynamic compromise [2]. The mortality rate of AEN is high, with one study approximating mortality at 30%. However, 60% of affected patients experience favorable outcomes with appropriate treatment [3]. AEN predominantly affects elderly males with multiple comorbidities [1]. Risk factors include type 2 diabetes, hypertension, alcoholism, and chronic kidney and liver disease [2].

Case Presentation:

A 71-year-old male presented to the emergency department with symptoms of gastrointestinal bleeding and a fall resulting in a head injury. Medical history remarkable for atrial fibrillation, coronary artery disease, congestive heart failure, aortic and mitral prosthetic valves, a pacemaker, chronic obstructive pulmonary disease, cirrhosis, renal disease on hemodialysis, and epilepsy. Medications included aspirin, carvedilol, Depakote, digoxin, divalproex sodium, furosemide, valsartan, and warfarin. Vitals were remarkable for a temperature of 95.1°F, heart rate of 60 bpm, respiratory rate of 20, blood pressure of 118/53 mmHg, and oxygen saturation of 96% on 4L via nasal cannula. He arrived confused and hypoglycemic, for which both improved following IV dextrose administration and red blood cell transfusion. The patient reported experiencing hematemesis, vomiting, hematochezia for three weeks, and black tarry stools for three months (which continued through most of the hospital course). Additionally, he had significant bodily weakness and a history of recurrent falls. Hemoglobin was 10.1g/dL on arrival, and this continued to drop daily. Further labs can be noted in Table 1. He was maintained on IV fluid resuscitation and IV proton pump inhibitor (PPI). The patient underwent esophagoduodenoscopy (EGD), which was remarkable for distal esophageal ulcerations and patches of black mucosa, concerning for necrosis, which was biopsied (Figure 1). Pathology was notable for necro-

inflammatory debris without intact esophageal mucosa. By day 4, there were no further reports of tarry dark stool, and Hgb had stabilized around 8g/dL. He was transfused 1U PRBC (secondary to cardiac history and transitioned to PO PPI. By day 6, he was feeling symptomatically better, his vitals remained stable, and he was cleared for discharge to rehab due to weakness. He was recommended to follow up in the clinic in 1 week for further evaluation and discussion of the need for a repeat endoscopy.

Investigations:

- *Laboratory Tests:*
  - Elevated white blood cell count
  - Renal dysfunction
  - Abnormal liver function tests
  - Elevated INR
- *Upper Endoscopy with Biopsy:*
  - Identified necro-inflammatory debris and ulcerative esophageal mucosa
- *Transthoracic Echocardiography:*
  - Mitral valve stenosis with paravalvular regurgitation
  - Reserved left ventricular systolic function
- *Liver and Gallbladder Sonogram:*
  - Benign findings with no obvious pathology
- *CT Imaging:*
  - CT Head showed cerebral atrophy with chronic microvascular ischemic changes
  - CT Chest/Abdomen revealed multiple rib fractures and pancreatic atrophy with fatty infiltrates

Table 1: Table depicting the trend of various labs from arrival to day 6 when the patient was cleared for discharge.

Lab Type	Arrival	Day 2	Day 3	Day 4	Day 5	Day 6
WBC (10^3/µL)	17.00	15.30	10.70	7.00	7.70	10.70
Hgb (g/dL)	10.1	9.2	8.5	7.9	8.1	9.2
Platelets (10^6/µL)	228	153	133	111	107	118
Sodium (mmol/L)	138	140	138	139	139	135
Potassium (mmol/L)	4.2	4.2	4.0	3.8	4.1	4.1
BUN (mg/dL)	64	68	43	27	17	14

Creatinine (mg/dL)	2.1	2.0	1.7	1.3	1.2	1.1

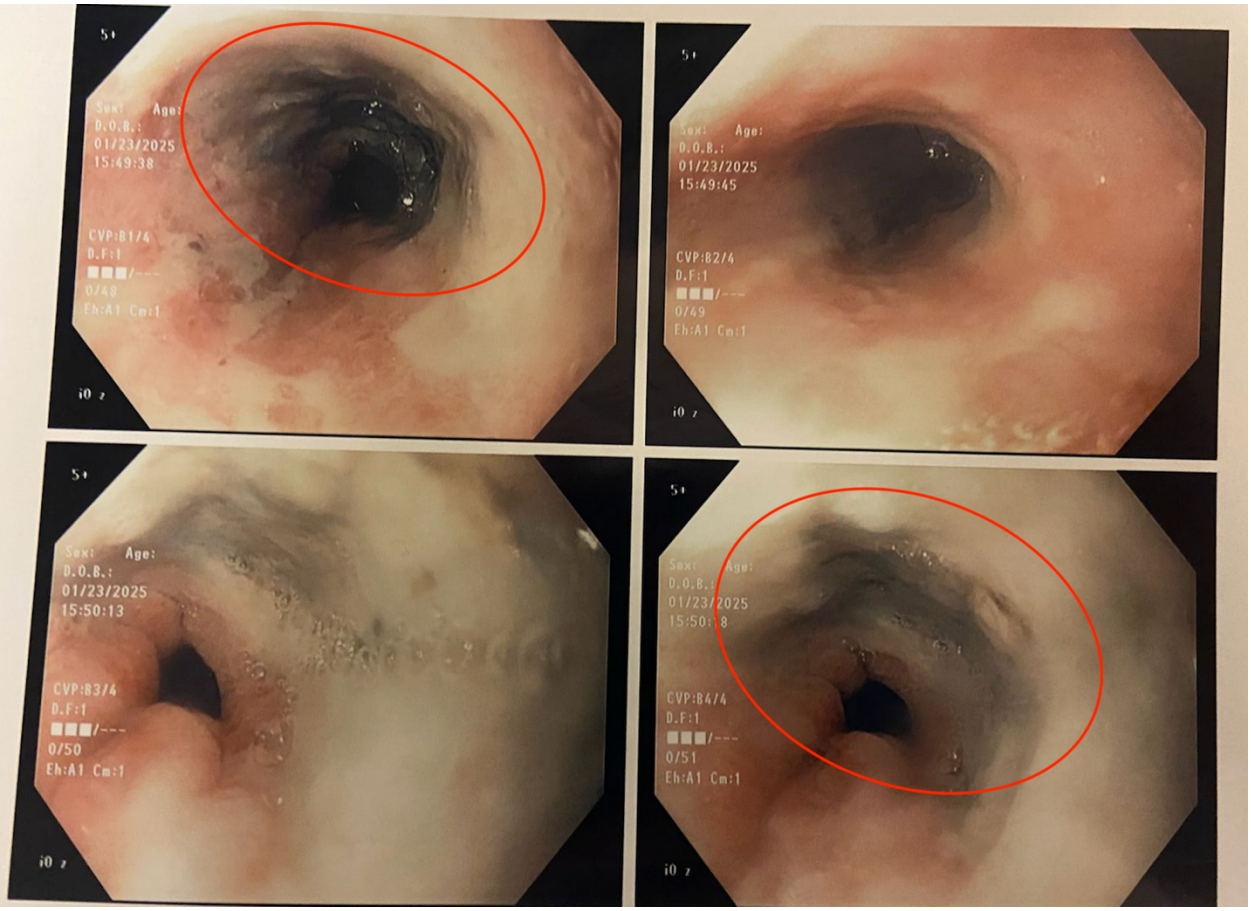


Figure 1: Endoscopic image with areas of visualized necrosis identified within the red circle.

Discussion:

AEN is a rare condition often associated with multiple comorbidities and systemic complications. The pathophysiology involves a combination of ischemia, gastric acid injury, and impaired mucosal defense mechanisms. Peak incidence occurs in the sixth decade of life and is more common in males with multiple comorbidities and poor nutritional status. Chronic alcohol use, illegal drug use, malignancy, solid organ transplantation, CAD, diabetes, hypertension, various infectious states causing immunocompromise, including HIV, herpes simplex virus, cytomegalovirus, and fungal infections, have been associated with diffuse AEN [4][5].

It primarily affects the distal esophagus due to its limited vascularization. Chronic microvascular ischemia can diminish perfusion, leading to intermittent low-flow states [5]. As the distal esophagus is considered a “watershed zone” compared to the remainder, as it receives blood flow from more distal branches, this makes it more susceptible to ischemic injury during these times of decreased perfusion [1] [6].

The classic presentation includes upper GI bleeding, hematemesis, coffee-ground emesis, melena, and dysphagia. Diagnosis is confirmed via esophagogastroduodenoscopy (EGD), which reveals characteristic black discoloration of the esophageal mucosa [8].

Histology is notable for necrotic changes, leukocytic infiltrate, and absence of squamous epithelium. Regeneration of damaged mucosa appears as whitish exudates in between the areas of black discoloration, giving it a “chessboard” like appearance [7]. The histopathology also helps distinguish AEN from other conditions, such as malignant melanoma, melanosis, coal deposition, etc.

The management of AEN includes symptomatic management and close monitoring for signs of hemodynamic compromise. Volume resuscitation and appropriate transfusion (to maintain hemoglobin > 7 g/dL and platelets at > 50,000/mm^3). Patients should initially be kept NPO for the first 24 hours with a slow progression of diet once more hemodynamically stable with less concern for bleeding/complications. Medical management includes gastric acid suppression with PPI and can include mucosal protection with sucralfate. Intravenous PPI is preferred initially (intermittent boluses vs infusion), which can later be changed to oral form [7]. Antimicrobial therapy is started for biopsy-positive esophageal cultures or in the presence of multinucleated giant cells or inclusion bodies. Nasogastric tubes should be avoided due to the risk of perforation. Bleeding should be rapidly controlled using submucosal epinephrine and some other form of hemostasis (i.e, self-expanding stents, clips, etc). Balloon tamponade should be avoided due to the risk of perforation. Perforations and strictures should be managed either surgically or with an expanding stent if surgery is not feasible (Chinta). Management can be summarized in Figure 2.

Management:

- **Supportive Care:**
  - Hgb transfusions for levels < 7g/dL (8 for those with cardiac comorbidities)
  - maintaining platelet numbers > 50,000/mm^3
  - V Fluid resuscitation,
- **Pharmacological Care:**
  - IV proton pump inhibitor infusion with transition to PO when stable
  - Sucralfate
  - Antibiotics (i.e. ceftriaxone)
  - Octreotide infusion if suspected variceal bleed
- **Nutritional Support:**
  - Maintain NPO initially, with slow advancement of diet
- **Anticoagulation Management:**
  - Can resume in the absence of active bleeding.
- **Lifestyle and Medications:**
  - Avoidance of NSAIDs, close INR monitoring.
- **Monitoring and Follow-up:**
  - Repeat laboratory tests and imaging to assess treatment effectiveness

Figure 2: Figure depicting overall management for acute eosinophilic necrosis.

The prognosis varies, with a mortality rate of 30-50%, largely depending on the patient’s underlying health conditions [9]. Complications such as esophageal perforation, strictures, and superinfection contribute to poor outcomes [8]. Effective management includes aggressive resuscitation, hemodynamic stabilization, gastric acid suppression, and infection control. At this time, there is no official consensus

on the timing of initial endoscopic intervention, but ideally, it should be performed within the first week [9]. Repeat endoscopic follow-up is recommended to monitor healing and prevent complications.

Conclusion:

AEN is a rare but life-threatening condition characterized by partial or complete circumferential blackening of the distal esophagus, primarily due to ischemic insult and systemic instability. Early recognition, aggressive resuscitation, and supportive care are critical in reducing mortality. Given the high association with comorbidities, a multidisciplinary approach is necessary for optimal patient outcomes.

References:

1. Gurvits, G. E. (2010, July 14). Black esophagus: Acute esophageal necrosis syndrome. World Journal of Gastroenterology. https://www.wjgnet.com/1007-9327/full/v16/i26/3219.htm
2. Richards, J. (2024, July 27). Esophageal necrosis. StatPearls [Internet]. https://www.ncbi.nlm.nih.gov/books/NBK572075/
3. Abdullah HM, Ullah W, Abdallah M, Khan U, Hurairah A, Atiq M. Clinical presentations, management, and outcomes of acute esophageal necrosis: a systemic review. Expert Rev Gastroenterol Hepatol. 2019 May;13(5):507-514. doi: 10.1080/17474124.2019.1601555. Epub 2019 Apr 16. PMID: 30933549.
4. Sheikh, Abu B, et al. "Acute Esophageal Necrosis: An in-Depth Review of Pathogenesis, Diagnosis and Management." *Journal of Community Hospital Internal Medicine Perspectives*, U.S. National Library of Medicine, 31 Jan. 2022, pmc.ncbi.nlm.nih.gov/articles/PMC9195118/.
5. Siddiqi A, Chaudhary FS, Naqvi HA, Saleh N, Farooqi R, Yousaf MN. Black esophagus: a syndrome of acute esophageal necrosis associated with active alcohol drinking. BMJ Open Gastroenterol. 2020 Aug;7(1):e000466. doi: 10.1136/bmjgast-2020-000466. PMID: 32788199; PMCID: PMC7422689.
6. Vancheri F, Longo G, Vancheri S, Henein M. Coronary microvascular dysfunction. Journal of clinical medicine. September 6, 2020. doi: 10.3390/jcm9092880. PMID: 32899944; PMCID: PMC7563453
7. Trad G, Sheikhan N, Ma J, Gheriani AG, Sagaslli A. Acute Esophageal Necrosis Syndrome (Black Esophagus): A Case Report of Rare Presentation. Cureus. 2022 Apr 19;14(4):e24276. doi: 10.7759/cureus.24276. PMID: 35607555; PMCID: PMC9123353.
8. Thomas M, Sostre Santiago V, Suhail FK, Polanco Serra G, Manocha D. The Black Esophagus. Cureus. 2021 Oct 11;13(10):e18655. doi: 10.7759/cureus.18655. PMID: 34790441; PMCID: PMC8583363.
9. Chinta S, Jyala A, Ghazanfar H, Makker J. Black Esophagus: A Rare Case of Acute Esophageal Necrosis. Cureus. 2024 Jan 21;16(1):e52660. doi: 10.7759/cureus.52660. PMID: 38380187; PMCID: PMC10878192.
10. Deliwala SS, Bala A, Haykal T, Elbedawi MM, Bachuwa G, Gurvits GE. Acute esophageal necrosis (GURVITS syndrome) presenting as Globus and altered phonation. American Journal of Case Reports. September 3, 2020. doi: 10.12659/AJCR.926019

A Case of Malignant Ampullary Ulceration

Eskarous, Hany MD [1]; Iskander, Peter MD [1]; Daniel, Mina MD [1]; Khadijah, Sajid MD [1]; Farah, Kanith MD [2]; Aman, Ali MD [3]

1. The Wright Center for GME, Scranton, PA, 18505
2. J.W. Ruby Memorial Hospital, Morgantown, WV 26506
3. Wilkes-Barre General Hospital, Wilkes-Barre, PA, 18764

Abstract:

Ampullary carcinomas, as the name suggests, are rare tumors that are associated with the ampulla of Vater. Due to their location, symptoms may arise relatively faster than other similar malignancies. Because of this, they can typically be identified sooner, yielding better prognosis and survival outcomes. They are diagnosed via pathology; usually from biopsies obtained while performing EUS/ERCP. Management revolves around resection if able to, or chemo/radiation if deemed unresectable or evidence of metastasis is noted. We present a case of a female presenting with abdominal pain associated with jaundice and scleral icterus. The endoscopic evaluation noted ampullary ulceration with biopsies proven to be notable for moderately differentiated adenocarcinoma.

Introduction:

Papillary or ampullary carcinomas (ACs) are malignant tumors in the mucosa of the major duodenal papilla region, also known as the ampulla of Vater. ACs are rare, comprising only about 0.2% of gastrointestinal cancers [1,2]. It's reported that 90% of patient cases diagnosed are predominantly found in Caucasian males [1]. Due to the increasing usage of upper endoscopy for unrelated symptoms and screening of patients with familial adenomatous polyposis, it has been shown that there has been a significant increase in incidence rates of ACs in Western countries over the last few decades [2]. Due to the complexity of the ampulla's anatomy, pancreatic, duodenal, and common bile duct (CBD) tumors are often misdiagnosed as ampullary tumors [3]. Because of the location of ACs, characteristic symptoms such as jaundice and pain usually occur earlier when compared to other conditions, such as pancreatic cancers [4]. ACs generally have a better prognosis due to earlier detection, with a 5-year survival rate of 45% in patients undergoing resection [4]. Two main histological subtypes can be identified in ampullary cancers: intestinal and pancreaticobiliary [5–7]. Intestinal ampullary adenocarcinomas originate in the intestinal epithelium coating the ampulla, while pancreaticobiliary ampullary carcinomas originate in the epithelium of the distal CBD and pancreatic duct [7]. Pancreaticoduodenectomy is the mainstay of management and is usually followed by adjuvant chemo or chemo-radiation therapy [1,7].

Clinical Course:

A 33-year-old female with a past medical history of cesarean section presented to the hospital for episodes of light stool, dark urine, and abdominal pain. On exam, she was noted to have abdominal discomfort to palpation, jaundice, and scleral icterus. She was made NPO, started on IV fluids, and was given medication for pain and nausea. Labs were significant for elevated bilirubin and liver enzymes (Table 1). Gastroenterology (GI) was consulted, and she underwent Endoscopic Ultrasound (EUS) and Endoscopic retrograde cholangiopancreatography (ERCP) to evaluate her further for imaging significant

for dilated gallbladder (GB). She was found to have ampullary ulceration, for which biopsy was notable for adenocarcinoma on pathology (imaging, procedure details, and pathology findings to follow) (Figures 1-3). Her liver function tests were noted to increase post-procedure, but per the GI team, this was to be expected. The patient tolerated the procedure well and was advanced to a low-fat diet. She was discharged later in stable condition with recommendations for stent removal and repeat lab work to follow up on LFTs.

Table 1: Table depicting lab values resulting over hospital stay, with day 1 being the day of admission and day 2 being the day of discharge. The procedure occurred on day 6.

Marker	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
ALP	640	516	551	612			723
ALT	85	64	69	81			114
AST	112	85	96	103			98
Direct bilirubin			2.8				
Total bilirubin	6.7	4.8	4.6	5.1			4.79
WBC	8.1	11.2		10		6.8	7.7
Hgb	13	10.7		11.2		10.6	10.5
Hct	38.6	31.2		33.2		31.7	31.8

Imaging:

US of the abdomen:

GB distension with moderate hypoechoic sludge, an 8mm soft tissue echogenicity consistent with polyp, and mild GB wall thickening (up to 4.7mm) with some pericholecystic edema.

MRCP:

Marked intra/extrahepatic biliary ductal dilation. CBD measuring up to 3.2cm, tapering to 1.2 cm distally, proximal to the ampulla of Vater. GB distension, mild wall thickening/edema, sludge, and edema.

Procedures:

EGD:

Hill Class III hiatal hernia.

EUS:
Moderate intra-hepatic biliary dilation, a massively dilated CBD measuring 35.5 mm proximally and 21.5 mm distally, and a small amount of sludge noted in the bile duct. No stones were seen.

ERCP:
The ampulla was noted to be ulcerated with a significantly dilated intraduodenal portion of the CBD, which is massively dilated. A careful biliary sphincterotomy was carried out with no complications. No obvious stones were seen; balloon sweeps yielded only sludge. The ampullary orifice appeared to be ulcerated. It was unclear whether this was secondary to an impacted stone causing ulceration (due to obstruction) or a small ampullary carcinoma. Aggressive biopsies of the ulcerated ampullary area were taken to rule out cancer, mainly focusing on the biliary orifice. This was followed by placing 10 French 12 cm plastic stents in the bile duct.

Pathology:
Markedly atypical glands associated with inflammation and fibrosis. Immunohistochemical stains for pan-cytokeratin, CK7, and CEA were positive with a high Ki-67. Negative for CDX2, P53, CK20, and CA 19-9. Reports verified a superficially invasive and moderately differentiated adenocarcinoma with marked ulceration. Background adenoma with high-grade dysplasia was noted.

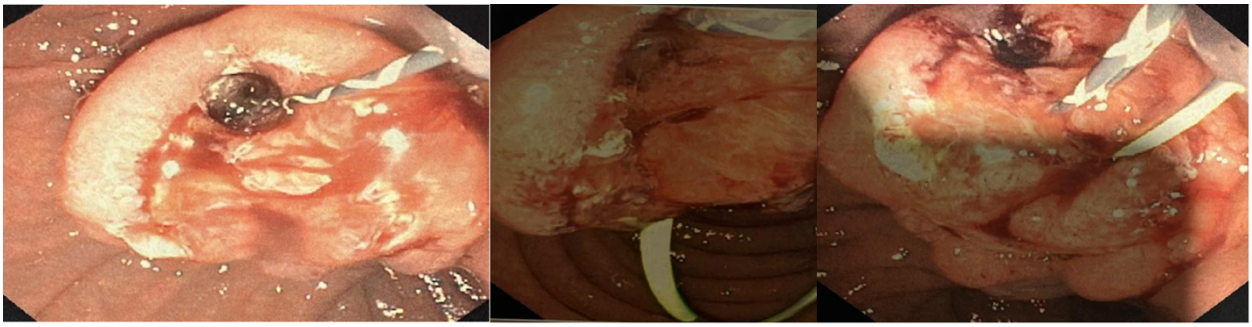


Figure 1: Endoscopic imaging revealing ulceration noted within the ampulla of Vater.

Discussion:

Several cancers exist in and around the ampulla, such as pancreatic ductal adenocarcinomas (PDACs), cancers of the duodenum, and ACs [1]. Ampullary or papillary carcinomas are tumors originating from the mucosa (epithelium) located in the major duodenal papilla, a complex area of bile and pancreatic ducts. This rare cancer comprises only 0.6-0.8% of digestive cancers, while it accounts for about 6-17% of periampullary carcinomas (PACs). Literature supports a slightly higher occurrence of ACs among men than women. The incidence rate of adenocarcinoma increases with age, and the highest rate is seen among people above 50 years of age. More specifically, based on the Surveillance, Epidemiology, and End Results (SEER) database, in the USA, the median age at diagnosis is 65 years, with a slight preference for males [8–11].

The majority of ACs consist of adenocarcinomas, with two main histologic subtypes described according to their epithelium of origin: intestinal- and pancreaticobiliary-associated ACs [12]. The occurrence of the intestinal subtype is often associated with the growth pattern of duodenal adenoma. It appears

suspicious for colon cancer, unlike the pancreaticobiliary subtype, which is prevalent in pancreatic tumors and cholangiocarcinoma.

If histomorphology is used exclusively, it may not be able to differentiate tumor entities, so further immunohistochemical markers can be stained. This is where the intestinal form, in most cases, has CDX2 and CK20 markers for colon cancers. In contrast, the pancreaticobiliary form tends to have markers typical for cholangiocarcinoma and pancreatic cancer, such as CK7, as seen with our patient [9]. Pancreaticobiliary abnormalities were primarily observed in the KRAS and TP53 genes [9,11,13], and the most apparent mutations in the APC gene were observed in the intestinal type.
Several histological classifications of ACs have evolved based on their microscopic appearance and growth pattern from the intestinal side or tumor extension.

Three distinct categories of carcinomas are recognized after the correlation of gross and microscopic features: (1) intra-ampullary neoplasms, wherein the growth preinvasive neoplasms are mainly intraluminal, commonly including the neoplasm that protrudes into the duodenal lumen from patulous orifices of the papilla of Vater; (2) peri-ampullary, which often manifest as vegetating, ulcerous components originating on the Identification: ulcerating part correlates with the invasive phase, meanwhile vegetating part corresponds with the preinvasive phase; and (3) mixed exophytic and mixed ulcerated lesions. The fact is that ulcerating tumors are usually found on tumors that have associated poor survival rates [14].

ACs usually present symptoms and signs frequently observed in extrahepatic cholangiocarcinoma and pancreatic adenocarcinoma: jaundice, diarrhea, steatorrhea, and gastrointestinal bleeding with melena appear to be frequent. This malignancy can be diagnosed early due to these symptoms, as they can appear earlier than other similar cancers. EUS, ERCP, and FNAC are the preferred techniques for AC diagnosis, while CT of the chest, abdomen, and pelvis stand out as essential staging tests. Endoscopic pulpectomy is the primary diagnostic tool and may be curative in some exceptional cases—for instance, early AC, in situ carcinoma, or low-grade dysplasia [10,13].

Typically, noninvasive ACs are asymptomatic and are discovered incidentally during EGD for some other indications. Some patients present with different symptoms, such as jaundice, abdominal pain, diarrhea, pancreatitis, cholangitis, steatorrhea, and melaena, which add up to the differential diagnosis with choledocholithiasis, extrahepatic cholangiocarcinoma, and pancreatic adenocarcinoma [13].

Ampullary cancer manifests mainly as obstructive jaundice, a symptom that accounts for about 85% of the cases because the tumor compresses the biliary duct of the distal portion of the organ. However, in carcinomas, jaundice would rather be persistent though not intermittent, making it very possible for the abdomen to become palpably engorged (Courvoisier's sign), although it is an uncommon event (it accounts for only 15% of the cases. A gallstone case is a constituent in a third of patients, making the diagnosis problematic [14].

Many patients also experience symptoms such as significant weight loss, fatigue, and abdominal discomfort. Acute pancreatitis, as compared to ampullary cancer, is less common; however, the two should not be ranked compared to this case. The rate of bleeding may lie between mild to severe, with as many as one-third of the patients being unable to emit any gastrointestinal blood until the time they

bleed heavily. Sometimes, large lesions can impede the outlet of the belly, which may lead to gastric outlet obstruction [14].

In terms of differential diagnosis, the most crucial consideration is the distinction between primary ACs, pancreatic cancers, distal cholangiocarcinoma, and small bowel cancers. However, FNAC also faces issues distinguishing ACs from other PACs; therefore, a surgical biopsy is often necessary to make realistic conclusions [12]. Unlike other periampullary cancers, true ampullary cancers start presenting earlier with biliary obstruction symptoms [10,13].

Although patients with an oncological ampullary adenoma can be managed conservatively with endoscopic therapy, the oncological presence of a localized carcinoma requires oncologic pancreatoduodenectomy resection. Nevertheless, the significant recurrence rate has prompted multimodal therapy to be the first choice in many patients [9].

Extensive surgical resection with lymphadenectomy forms the basis of treatment for AC, and pancreaticoduodenectomy (PD) equals the current modality for operations against cancer, with 50% of cases believed to be possible at wider resection [9].

One of the main issues in the procedure is the nodes' high rate (not less than 30–40%) involvement, especially those located in the vicinity of the superior mesenteric artery and the pancreaticoduodenal area. This significantly indicates the pivotal role of proper and adequate lymphadenectomy for both lymph node dissection and the reckoning of at least 12. First-line therapy in AC is surgery followed by extended oncological resection of the head of the pancreas with lymphadenectomy taken systematically [9].

This operation involves the removal of the GB together with the transverse section of the CBD immediately cranially off the bifurcating cystic duct originating from the duodenum, the first jejunal loop after the ligament of the Treitz, and the pancreatic head. This is performed as an en bloc resection [9]. Regional lymph nodes of the hepatoduodenal ligament, superior margin of pancreaticoduodenal part, to the celiac trunk, and in the triangle between the portal vein and splenic/lienal artery and splenic/lienal vein make up the resectate [9]. It is not easy to differentiate between primary ampullary and other periampullary cancers by anatomic site by preoperative imaging. In the beginning, ampullary cancers are surgically treated, just like pancreatic cancers. They are usually eliminated using a Whipple procedure, which involves removing the duodenum and pancreas.

The management of unresectable and metastatic disease is mainly chemotherapy comprising fluoropyrimidine, gemcitabine, and a platinum compound (usually cisplatin or oxaliplatin) [13]. Even if performing radical surgery is extremely challenging for early-stage AC, the recurrence rate is still about 50%. On this basis, i.e., a class of cancer treatment techniques like chemotherapy, radiation therapy, or chemoradiation has been compared over time in certain studies.

In addition, the histological subtype is considered when imparting an adjuvant and first-line treatment. Hence, ACs that are pancreaticobiliary-like are treated as pancreatic adenocarcinoma and occasionally biliary tract cancer [12]. The historical approach indicates a poor prognosis, even at the resectable stage,

and raises questions about possible further improvement in the metabolic course by chemotherapy administration, 5-FU, or gemcitabine [9].

The current evidence points out that subtypes of resectional parts affect the biological behavior and, thus, the patient's response. Accordingly, jaundice is a harbinger of the advanced stages of the disease and the accumulation of the disease in the tissues, interfering with their function [8]. Hence, it is very instrumental in avoiding and improving the effectiveness of this condition.

Recurrence can be regionally adjacent to the excision site (involves the tumor bed, para-aortic lymphatics, or any distant site). Often enough, the lymph nodes perivisceral are involved. In these cases, the disease progression tends to remain localized in the region. The spreading of AC generally follows a halsteadian progression: it usually originates in the nodes, which undergo the first changes, and the liver becomes affected. Metastases proceed to other organs [14].

The general survival rate at 5 years is randomly indicated from one source to another, slightly from 32% to 67.7%. A favorable result or not is associated with the allocation of lymph nodes, the degradation of cellular elements, and the invasion of the pancreas into resectable patients [14]. From all those, AC offers a better prognosis in comparison to pancreatic and bile duct adenocarcinoma, because of their early presentation, the resection rate is high as compared to other periampullary carcinomas [8,13]. When it comes to surgical treatment (e.g., pancreatoduodenectomy), there is a uniform approach irrespective of whether it is ampullary adenocarcinoma or pancreatic cancer adenocarcinoma. However, the long-term outcomes among periampullary adenocarcinomas can be markedly different [13]. More than 80% of ampullary cancers, with also good 5-year survival, ranging from 30% to as high as 50%, are being resected, which is far better than the rates of resection and survival seen in bile duct cancers and pancreatic cancer. Nevertheless, though pancreatic tumors are more resectable compared to ampullary adenocarcinomas, only a few patients with AC that have been resected live longer [13]. After curative resection, nodal status is one of the strongest predictors of survival: on the other hand, in one series performed, pancreaticoduodenectomy with the negative paternosc's zone showed a survival rate of 80% of patients after 5 years, whereas with positive nodes, only 25% of patients were alive for 5 years [14].

Conclusion:

AC presents as a rare but significant malignancy of periampullary cancers. Despite advancements in diagnostic modalities, including endoscopic and radiological techniques, definitive diagnosis often necessitates histopathological examination. Surgical resection, particularly pancreatoduodenectomy, remains the cornerstone of treatment, offering the best chance of cure. However, the high recurrence rate underscores the importance of multimodal therapeutic approaches. Prognosis varies based on histological subtype, nodal involvement, and resection margins. ACs generally have a better prognosis than pancreatic and bile duct adenocarcinomas.

While challenges persist in managing AC, advancements in understanding its biology and refining therapeutic strategies offer hope for improved outcomes, underscoring the need for multidisciplinary approaches tailored to individual patients.

References:

1. Al-Jumayli M, Batool A, Middiniti A, et al.: Clinical Outcome of Ampullary Carcinoma: Single Cancer Center Experience. J Oncol. 2019, 2019:3293509. 10.1155/2019/3293509
2. Rizzo A, Dadduzio V, Lombardi L, Ricci AD, Gadaleta-Caldarola G: Ampullary Carcinoma: An Overview of a Rare Entity and Discussion of Current and Future Therapeutic Challenges. Curr Oncol. 2021, 28:3393–402. 10.3390/curroncol28050293
3. Balci S, Basturk O, Saka B, et al.: Substaging Nodal Status in Ampullary Carcinomas has Significant Prognostic Value: Proposed Revised Staging Based on an Analysis of 313 Well-Characterized Cases. Ann Surg Oncol. 2015, 22:4392–401. 10.1245/s10434-015-4499-y
4. Zimmermann C, Wolk S, Aust DE, et al.: The pathohistological subtype strongly predicts survival in patients with ampullary carcinoma. Sci Rep. 2019, 9:12676. 10.1038/s41598-019-49179-w
5. Wakasugi M, Tanemura M, Furukawa K, et al.: Signet ring cell carcinoma of the ampulla of Vater: Report of a case and a review of the literature. Int J Surg Case Rep. 2015, 12:108–11. 10.1016/j.ijscr.2015.05.031
6. Zheng-Pywell R, Reddy S: Ampullary Cancer. Surg Clin North Am. 2019, 99:357–67. 10.1016/j.suc.2018.12.001
7. Romiti A, Barucca V, Zullo A, et al.: Tumors of ampulla of Vater: A case series and review of chemotherapy options. World J Gastrointest Oncol. 2012, 4:60–7. 10.4251/wjgo.v4.i3.60
8. Askew J, Connor S: Review of the investigation and surgical management of resectable ampullary adenocarcinoma. HPB (Oxford). 2013, 15:829–38. 10.1111/hpb.12038
9. Walter D, Schnitzbauer AA, Schulze F, Trojan J: The Diagnosis and Treatment of Ampullary Carcinoma. Dtsch Arztebl Int. 2023, 120:729–35. 10.3238/arztebl.m2023.0195
10. Maselli R, de Sire R, Fugazza A, et al.: Updates on the Management of Ampullary Neoplastic Lesions. Diagnostics (Basel). 2023, 13:. 10.3390/diagnostics13193138
11. Tsagkalidis V, Langan RC, Ecker BL: Ampullary Adenocarcinoma: A Review of the Mutational Landscape and Implications for Treatment. Cancers (Basel). 2023, 15:. 10.3390/cancers15245772
12. Rizzo A, Dadduzio V, Lombardi L, Ricci AD, Gadaleta-Caldarola G: Ampullary Carcinoma: An Overview of a Rare Entity and Discussion of Current and Future Therapeutic Challenges. Curr Oncol. 2021, 28:3393–402. 10.3390/curroncol28050293
13. Ahn DH, Bekaii-Saab T: Ampullary cancer: an overview. Am Soc Clin Oncol Educ Book. 2014, 112–5. 10.14694/EdBook_AM.2014.34.112
14. Panzeri F, Crippa S, Castelli P, et al.: Management of ampullary neoplasms: A tailored approach between endoscopy and surgery. World J Gastroenterol. 2015, 21:7970–87. 10.3748/wjg.v21.i26.7970

From Eye to Gut: A Melanotic Journey

Iskander, Peter MD [1] ; Hayat, Umar MD [2]; Daniel, Mina MD [1]; Ali, Khizar Amin MD [3]; Shahzad, Muhammad Talha MD [4]; Ali, Aman MD [3]

1. The Wright Center for GME, Scranton, PA
2. Geisinger Wyoming Valley Medical Center, Wilkes-Barre, PA
3. Wilkes-Barre General Hospital, Wilkes-Barre, PA
4. University of Michigan Health-West Hospital, Wyoming, MI

Abstract:

Uveal melanomas are one of the most common primary malignancies of the eye [1]. Metastasis to solid organs has been documented, but very rarely has it been noted to occur in the stomach [2]. Symptoms can depend on the site of metastasis and can include, but are not limited to, nausea/vomiting, iron deficiency, malabsorption, obstruction, etc. We present a case of a patient with choroidal melanoma with associated development of esophageal, gastric, hepatic, and lung malignancies.

Case:

A 64-year-old male with a past medical history of intermittent palpitations, nicotine abuse, and choroidal melanoma of the left eye status post radiation (10/2022) presented due to sporadic episodes of dysphagia associated with occasional nausea and vomiting.

The patient proceeded to undergo EGD, for which findings of esophageal inflammation were observed; biopsy noted marked atypia, and high pan-cytokeratin/Ki-6,7, which was concerning for squamous cell carcinoma (SCC).

Repeat EGD showed dysplastic-looking epithelia between 34 cm - 37 cm from the incisors (Figure 1). Endoscopic mucosal resection was performed. PET-CT was unremarkable for any obvious areas of abnormal uptake or lymphadenopathy. The EMR scar site was observed upon follow-up EGD, as well as a pigmented gastric polyp (Figure 2). Chromoendoscopy of the esophagus was unremarkable, with scar tissue pathology biopsies noting benign esophageal squamous mucosa. Pathology of the polyp, however, was concerning for metastatic malignant melanoma.

The tumor was positive for SOX10, Mart-1, HMB45, and BRAF (+ cytoplasmic staining) and negative for pan-cytokeratin and CD68 immunostains, which supported the diagnosis.

Repeat CT showed interval development of liver and lung nodules concerning further metastasis. The patient is to follow up to discuss further management and goals of care.

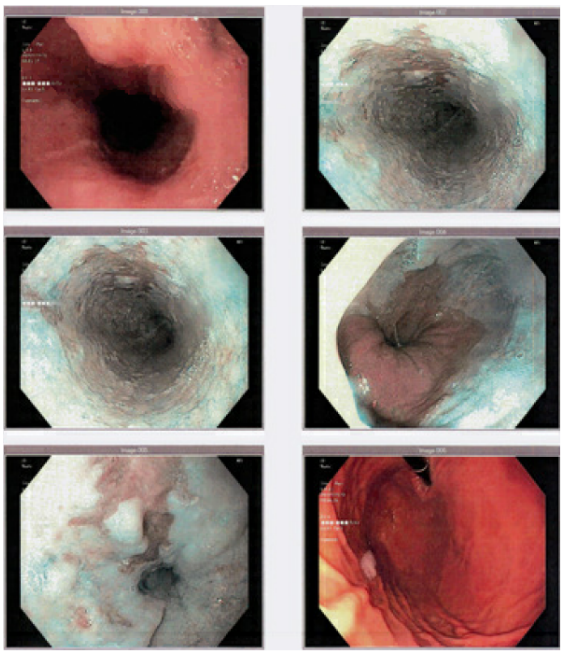


Figure 1: EGD findings showing dysplastic-appearing epithelia 34 cm - 37 cm from the incisor.

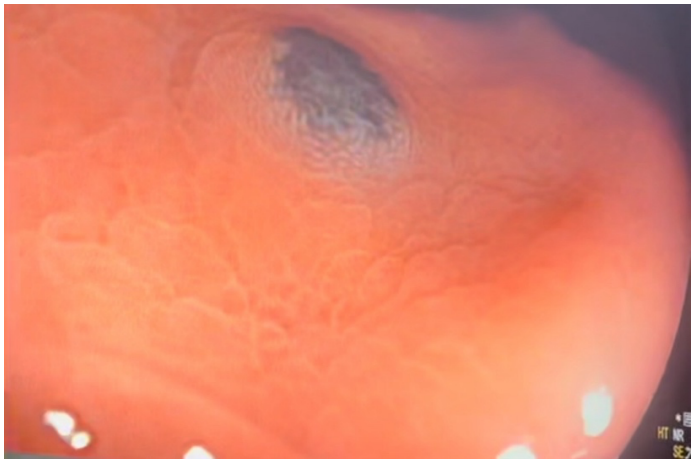


Figure 2: Pigmented gastric polyp.

Discussion:

Unlike cutaneous melanomas, known to metastasize to the GI tract, choroidal melanomas usually have a predilection for hematogenous spread to the liver [3]. Although there have been some cases detailing the possibility of esophageal carcinomas metastasizing to the eye in literature, no obvious reports have noted the opposite; this begs the question of whether the 2 cancers were somehow related or separate entities. Of the two, the latter is more likely, as melanoma and SCC have different histologies and, as far as we are aware, one type of cancer cannot turn into another. Of note, however, there have been cases reported of mixed-type tumors [4]. The patient was known to have a history of nicotine abuse, which is one of the most common risk factors for esophageal SCC [5].

As per the gastric polyp, very few cases in the literature have noted a primary choroidal melanoma metastasizing to the stomach. Pathology of the polyp did have markers that supported the evidence of choroidal melanoma metastasis. SOX10 is a transcription factor for which overexpression has been related to melanoma and prostate cancer [6]. Mart-1 is a normal transmembrane protein found in most melanocytes for which increased expression is associated with malignant melanoma [7]. Human Melanoma Black (HMB45) is a monoclonal antibody that reacts with gp100 proteins, which are expressed in immature and activated melanocytes [8]. BRAF belongs to the family of proto-oncogenes for which mutations can lead to unregulated cell signaling and growth [9].

The mean survival time has been noted to be as little as 4-6 months when there is evidence of metastasis [10]. However, there have been documented long-term disease-free periods in some who underwent early surgical resection with appropriate chemotherapy [11]. Ultimately, the management is a combination of immunotherapy and palliative interventions.

Conclusion:

A high index of suspicion should be taken with patients with previously known melanomas due to their vague metastatic symptoms. Regular mucosal inspections should be conducted with appropriate immunohistochemical biopsies for any suspicious changes [12]. Goals of care and management discussions, for chemotherapy and palliation, should be had abruptly as the signs of metastasis render a very poor prognosis.

References:

1. Ye Z, Yang Z, Zheng S, Wang W. Robot-assisted laparoscopic surgery for abdominal metastatic melanoma mimicking a gastrointestinal stromal tumor: A case report and review of the literature. Medicine (Baltimore). 2018 Jun;97(26):e11207. doi: 10.1097/MD.0000000000011207. PMID: 29952974; PMCID: PMC6039597.
2. Baba S, Akiyama Y, Endo F, Nikai H, Sugimoto R, Umemura A, Katagiri H, Hasegawa Y, Iwaya T, Nitta H, Koeda K, Sugai T, Sasaki A. Laparoscopic distal pancreatectomy for metastatic melanoma originating from the choroidal membrane: a case report. Surg Case Rep. 2021 Dec 20;7(1):268. doi: 10.1186/s40792-021-01345-x. PMID: 34928463; PMCID: PMC8688594.
3. Kaila, Vishal MD1; Lager, Donna J. MD2; Jain, Rajeev MD, FACP3. Choroidal Melanoma Metastases to the Stomach and Duodenum: 2629. American Journal of Gastroenterology 113():p S1461-S1462, October 2018.
4. Kochoumian E, Kazlouskaya V, Mangold A, Lal K, Maia-Cohen S, Elston DM. Tumor with the features of both squamous cell carcinoma and melanoma (melanocarcinoma). Indian Dermatol Online J. 2015 May-Jun;6(3):217-9. doi: 10.4103/2229-5178.156422. PMID: 26009723; PMCID: PMC4439757.
5. Tarazi M, Chidambaram S, Markar SR. Risk Factors of Esophageal Squamous Cell Carcinoma beyond Alcohol and Smoking. Cancers (Basel). 2021 Feb 28;13(5):1009. doi: 10.3390/cancers13051009. PMID: 33671026; PMCID: PMC7957519.
6. Mohamed A, Gonzalez RS, Lawson D, Wang J, Cohen C. SOX10 expression in malignant melanoma, carcinoma, and normal tissues. Appl Immunohistochem Mol Morphol. 2013 Dec;21(6):506-10. doi: 10.1097/PAI.0b013e318279bc0a. PMID: 23197006.
7. Fetsch PA, Marincola FM, Filie A, Hijazi YM, Kleiner DE, Abati A. Melanoma-associated antigen recognized by T cells (MART-1): the advent of a preferred immunocytochemical antibody for the diagnosis of metastatic malignant melanoma with fine-needle aspiration. Cancer. 1999 Feb 25;87(1):37-42. PMID: 10096358.

8. Rothberg BE, Moeder CB, Kluger H, Halaban R, Elder DE, Murphy GF, Lazar A, Prieto V, Duncan LM, Rimm DL. Nuclear to non-nuclear Pmel17/gp100 expression (HMB45 staining) as a discriminator between benign and malignant melanocytic lesions. Mod Pathol. 2008 Sep;21(9):1121-9. doi: 10.1038/modpathol.2008.100. Epub 2008 Jun 13. PMID: 18552823; PMCID: PMC2570478.
9. Aksenenko MB, Kirichenko AK, Ruksha TG. Russian study of morphological prognostic factors characterization in BRAF-mutant cutaneous melanoma. Pathol Res Pract. 2015 Jul;211(7):521-7. doi: 10.1016/j.prp.2015.03.005. Epub 2015 Apr 2. PMID: 25888143.
10. Chamoun, Romy MD1; Lander, Jared DO2; Collazzo, Jack A. MD3. S3649 Choroidal Melanoma: A Rare Cause of Gastric/Duodenal Pigmented Macules. The American Journal of Gastroenterology 117(10S):p e2288-e2289, October 2022. | DOI: 10.14309/01.ajg.0000871236.78470.89
11. Lin S, Wan D, Chen H, Chen K, Zheng S. Complete resection of isolated hepatic metastatic uveal melanoma with a notably long disease-free period: A case report and review of the literature. Oncol Lett. 2015 Jul;10(1):196-200. doi: 10.3892/ol.2015.3217. Epub 2015 May 18. PMID: 26170998; PMCID: PMC4487100.
12. Liang KV, Sanderson SO, Nowakowski GS, Arora AS. Metastatic malignant melanoma of the gastrointestinal tract. Mayo Clin Proc. 2006 Apr;81(4):511-6. doi: 10.4065/81.4.511. PMID: 16610571.

A "MacGiveric" Myotomy for a Gastric Bypass Conundrum

Iskander, Peter MD [1]; Hayat, Umar MD [2]; Asija, Udit MD [1]; Daniel, Mina MD [1]; Ali, Khizar Amin MD [3]; Shahzad, Muhammad Talha MD [4]; Ali, Aman MD [3]

1. The Wright Center for GME, Scranton, PA
2. Geisinger Wyoming Valley Medical Center, Wilkes-Barre, PA
3. Wilkes-Barre General Hospital, Wilkes-Barre, PA
4. University of Michigan Health-West Hospital, Wyoming, MI

Introduction:

Postprandial nausea and vomiting (N/V) following a gastric sleeve can be a common side effect after the procedure as the body adapts to the smaller size of the stomach. The management guidelines recommend having smaller but more frequent meals focused on protein, adequate and thorough chewing, and adequate fluid intake. Risk factors can include the female sex as well as the amount of opioid medication used for postoperative pain. We present a case of an elderly man experiencing postprandial nausea and vomiting for years after his gastric sleeve procedure, found to have distorted anatomy upon EGD evaluation, requiring further endoscopic intervention.

Case:

A 72-year-old male with a past medical history of Gastroesophageal reflux disease (GERD), cholecystectomy, and gastric sleeve (~2015) presented with chronic persistent postprandial nausea and vomiting.

CTA abdomen pelvis showed postsurgical changes of his gastric sleeve, moderate-sized hiatal hernia, normal-appearing duodenum / small bowel, and diverticulosis of the colon.
The patient underwent esophagogastroduodenoscopy (EGD), which showed evidence of a Hill class IV hiatal hernia. In the proximal aspect of the gastric sleeve, just below the GE junction, an abnormal ridge/diaphragm was noted between the gastric fundus and sleeve body, causing food retention and secretions along the greater curve in the fundus (Figure 1). Gastric sleeve, and this ridge was dilated with a 20 mm balloon, with no improvement in his symptoms. Gastric emptying scan was normal; however, his symptoms of immediate nausea and vomiting after eating were quite convincing to point towards retention of food in the retained fundus, caused by this abnormal septum. After detailed discussions with the patient, gastric sleeve septotomy or " S-POEM" was offered.

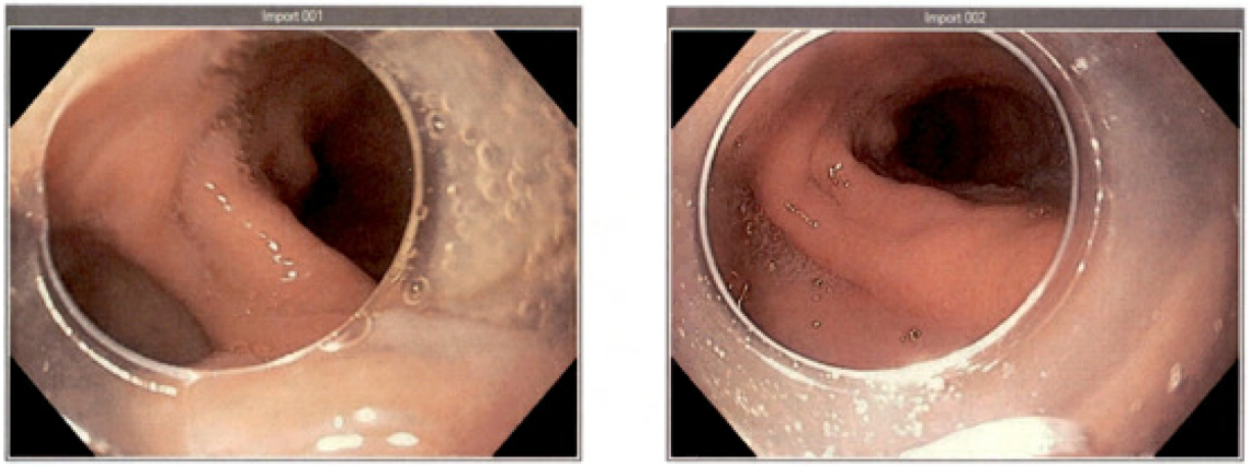


Figure 1: Abnormal prominent mucosal ridge noted just below the GE junction, suspected to cause the patient's post-prandial nausea and vomiting.

In this procedure, methylene blue was injected submucosally to delineate the details of the submucosa at the septum, followed by mucosotomy to expose the layers. Soft coagulation and endocut were combined to divide the muscle fibers horizontally (Figure 2). Once the ridge was opened up adequately, the mucosal defect was located and closed with a double-channel scope utilizing Apollo Prolene 2.0 sutures (Figure 3). The patient was followed for recovery after two weeks and could eat solids and liquids without any symptoms of nausea and vomiting.

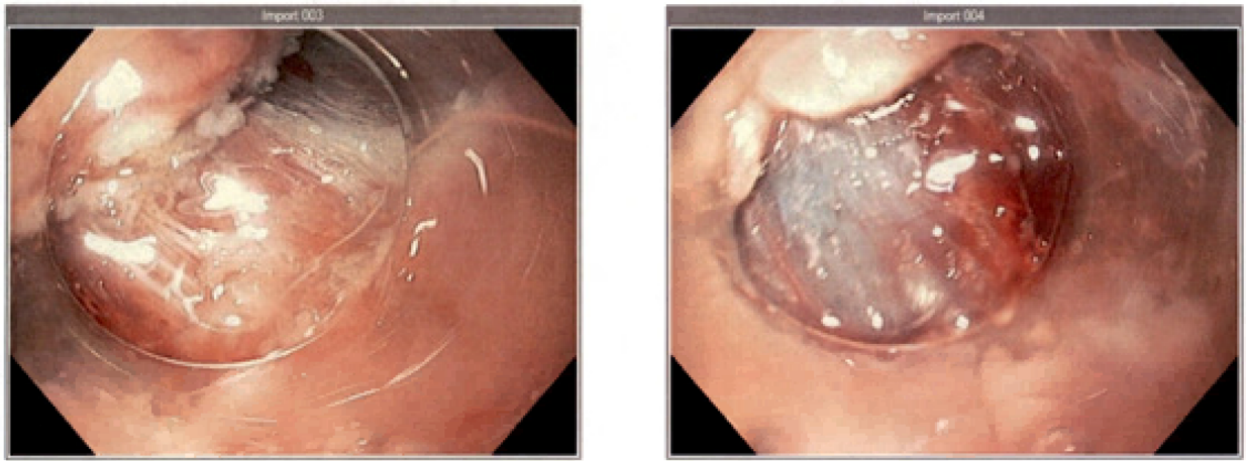


Figure 2: Horizontal mucosotomy was performed using a combination of coagulation and endocut to separate mucosal layers.

Figure 3: Defect noted upon opening of the mucosal ridge.

Discussion:

Laparoscopic sleeve gastrectomies are procedures performed to treat obesity, typically for patients with a body mass index (BMI) over 40 or 35 with other comorbidities. In addition to weight loss, the procedure has been noted to improve other health disparities, such as hypertension, obstructive sleep apnea (OSA), diabetes mellitus type 2, etc.

Postprandial N/V is a common side effect after the procedure due to the sudden decrease in gastric size and seems to be exacerbated by foods high in fiber, sugar, and fat. Rarely, however, these gastrointestinal symptoms can be secondary to postoperative anatomical changes. As with our case, an irregular mucosal ridge was noted to have developed post-surgery. This ridge was found right below the Gastroesophageal (GE) junction in the gastric cardia and was interfering with the passage of food contents into the stomach. This was verified by direct visual observation of food contents within the folds. Ingenuity had to be implemented as there are no specific guidelines for intervention for such anatomical defects. A dual-channel scope was utilized for further evaluation due to more endo-therapeutic and suction capabilities. An endoscopic incision was made to divide the submucosa from the muscularis layers until the defect was visualized. After repair, endoscopic suturing techniques were utilized to close. Per review of the literature, similar approaches to this complication have not been documented.

Conclusions:

Nausea and vomiting can be common complications after sleeve gastrectomies, but are usually the result of patients needing to acclimate to a smaller stomach volume. This case reports a unique etiology of an abnormal mucosal ridge catching food/liquid as it passed through the stomach. A novel approach was taken involving an EGD with septoplasty (Sleeve-POEM) to divide the muscular ridge between the gastric fundus and gastric sleeve to fix the deformity. This caused rapid improvement in symptoms. Persistent abdominal symptoms after procedures should be investigated for further possibilities of structural defects as the etiology.

References:

1. Song Y, Zhu J, Dong Z, Wang C, Xiao J, Yang W. Incidence and risk factors of postoperative nausea and vomiting following laparoscopic sleeve gastrectomy and its relationship with Helicobacter pylori: A propensity score matching analysis. Front Endocrinol (Lausanne). 2023 Feb 22;14:1102017. doi: 10.3389/fendo.2023.1102017. PMID: 36909334; PMCID: PMC9992875.

Think Grow Learn