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Case Report

Unraveiling the Mystery: Jejunal Stenosis Post-Stroke: A Case Report

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Abstract

Background: Jejunal stenotic strictures are relatively rare but have significant clinical entities and various complex etiologies characterized by the narrowing or obstruction of the jejunum.

Case Report: The patient presented with a two-month history of abdominal pain, persistent nausea, bilious vomiting, and dizziness, following a left posterior cerebral artery (PCA) stroke. Workup and various imaging studies revealed stenosis of the 3rd and 4th portion of the proximal jejunum, leading to the decision to proceed with exploratory laparotomy with bowel resection and anastomosis. The histological examination of the 15cm jejunum resection revealed significant transmural inflammation, encompassing both acute and chronic components. The presence of ulcers and prominent lymphocyte infiltration was noted, along with granulation tissue and hypertrophic muscularis propria.

Conclusion: The absence of focal deficits and subsequent ischemic events, accompanied by a history of inflammatory bowel disease, suggested ischemic enteritis (IE) as the primary etiology. The patient exhibited a full recovery following the surgical intervention and reported normal gastrointestinal (GI) function. This case emphasizes the necessity for further research to gain a comprehensive understanding of the pathophysiology and optimal management approaches for jejunal stenosis. It further highlights the significance of considering IE in patients with persistent GI symptoms post-stroke.

Keywords: jejunal stricture, ischemic enteritis, posterior cerebral artery stroke

Background

Intraluminal strictures are uncommon and can have various underlying causes, including congenital defects, inflammation, ischemia, neoplasms, or radiation [1]. These strictures result in a narrowing of the bowel lumen and dilation of the upstream bowel segment, a characteristic feature [2]. Histologically, most strictures exhibit findings of active and chronic inflammation accompanied by fibrosis, which contributes to the narrowing and rigidity of the affected bowel segment [2]. Jejunal stenotic strictures, specifically, are rare but significant clinical entities characterized by the narrowing or obstruction of the middle segment of the small intestine.

Diagnosing jejunal strictures can be challenging due to the diverse etiologies involved and the complex presentation of symptoms. IE is a rare but significant condition characterized by reduced arterial blood supply to the small intestine, resulting in various complications [3]. Stroke, the second leading cause of death globally, not only impacts the brain but also exerts effects on various other organs, including the GI tract. Stroke-induced disruptions in metabolic control and nutritional homeostasis can lead to GI complications which may damage the intestinal mucosa [4]. The intestinal mucosal barrier plays a critical function in the interaction between the brain and the gut, influencing protein expressions related to inflammation [4]. While ischemic colitis is more commonly reported, IE is less frequent but requires surgical intervention for relief. This case report discusses a 54-year-old female with a recent history of left PCA stroke presenting with persistent GI complaints including abdominal pain, nausea, bilious vomiting, and dizziness, leading to the diagnosis and successful management of jejunal stenosis associated with IE. The case underscores the importance of considering IE as a potential cause of GI symptoms in stroke patients and emphasizes the role of comprehensive diagnostic evaluations and surgical interventions in managing this condition effectively.

Case Presentation

A 54-year-old female with a past medical history of a recent left PCA stroke, GERD (gastroesophageal reflux disease), hypertension, and HLD (hyperlipidemia), presented to the emergency department with symptoms of abdominal pain, persistent nausea, bilious vomiting, and dizziness for the past two months. The patient reported that these symptoms had worsened following her stroke 2 months ago and were accompanied by intermittent epigastric burning and abdominal pain. Although she could tolerate solid food, she could only eat small amounts due to a persistent sensation of fullness and mentioned having bowel movements. On physical examination, she had a soft, nondistended abdomen with mild generalized tenderness. Her past surgical history includes c-section, abdominoplasty, lumbar surgery, and a recent diagnostic laparoscopy that indicated normal small bowel for similar symptoms.

Due to worsening and unresolved symptoms, the patient was transferred to the surgical floor for further investigations. A subsequent CT scan of the abdomen and pelvis revealed severe narrowing of the 3rd-4th portion of the duodenum, suggesting obstruction or internal hernia. Abdominal and pelvic X-rays conveyed partial or resolving small bowel obstruction. Esophagogastroduodenoscopy (EGD) showed proximal jejunal stenosis, and a biopsy was obtained revealing granulation tissue with transmural acute on chronic inflammation. An upper GI series with a small bowel follow-through (Figure 1) demonstrated a redundant duodenum, and stenosis of the 4th portion/proximal jejunum, with contrast passing into the remaining small and large bowel. Considering the patient's recent hospitalizations, unsuccessful diagnostic laparoscopy, inability to identify the cause of jejunal stenosis, and increased risk of recurrence,

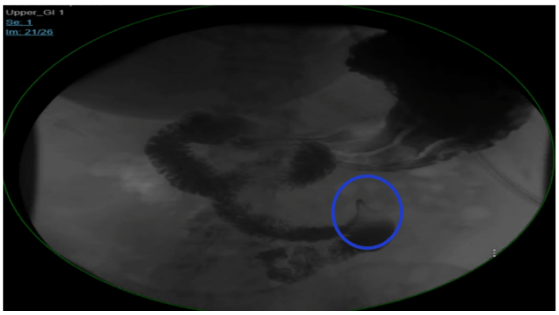

Figure 1: Upper GI Series with Small Bowel Follow-Through Indicating stenosis between the 3rd and 4th portions of the proximal jejunum.

the decision was made to proceed with exploratory laparotomy with potential bowel resection, and/or gastrointestinal bypass. During the surgery, an obstruction was discovered approximately 15 cm from the ligament of Treitz, caused by a stenotic segment of the jejunum. The proximal jejunum was distended, and the distal jejunum collapsed. Dense adhesions (Figure 2) were also found, involving the antimesenteric side of the stenotic jejunum and the transverse colon mesentery.

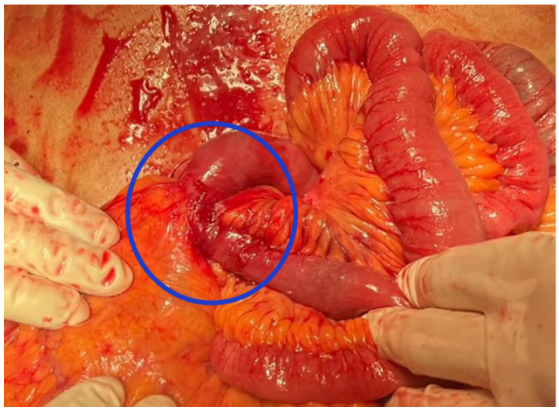

Figure 2: Exploratory Laparotomy showing dense adhesions involving stenotic jejunum.

The small bowel mesentery appeared twisted with distal small bowel herniating between the transverse colon mesentery and the mesentery of the jejunum. Nearly 15 cm of jejunum was resected (Figure 3) involving approximately 6 cm of dense circumferential fibrosis and thickening, which resulted in significant stenosis of the lumen. The proximal jejunal stump and the distal jejunal stump were examined followed by an anastomosis between the two stumps.

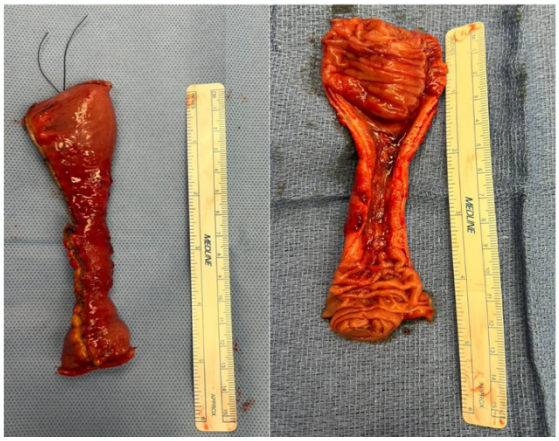

Figure 3: Jejunum Resection showing 15 cm of jejunum resected involving 6 cm of dense circumferential fibrosis and thickening.

Postoperative Outcome And Follow-Up: The patient had a smooth recovery during the postoperative period following the surgical intervention. On postoperative day 2, an X-ray with upper GI series (Figure 4), including a small bowel follow-through, revealed no obstructive signs or leakage. The patient was advanced to a regular diet from clear liquids on postoperative day 3. Importantly, there were no reports of nausea, vomiting, or abdominal pain during this time. Subsequently, the patient was discharged with an outpatient visit in two weeks. During the follow-up visit, the patient exhibited a full recovery with no acute symptoms. She reported tolerating a regular diet, passing flatus, and having normal bowel movements, indicating a return to normal gastrointestinal function.

The biopsy of the resection showed marked transmural acute and chronic inflammation with ulcer and prominent lymphocyte infiltration within the segment of the small intestine, along with granulation tissue reaction and hypertrophic muscularis propria (Figure 5). The biopsy does not indicate neoplastic or granulomatous inflammation. These positive outcomes, including the absence of postoperative complications, the patient's return to normal diet and gastrointestinal function, as well as the favorable biopsy findings, suggest a successful surgical intervention and a good prognosis for the patient.

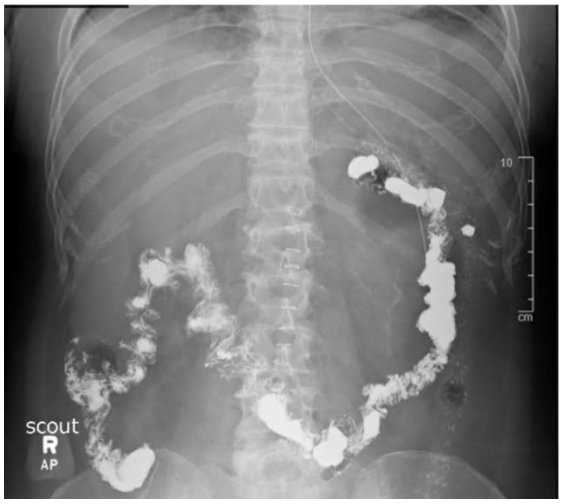

Figure 4: XRAY + Upper GI & Small Bowel Series Status post jejunal resection day 2: no evidence of intestinal obstruction or leak.

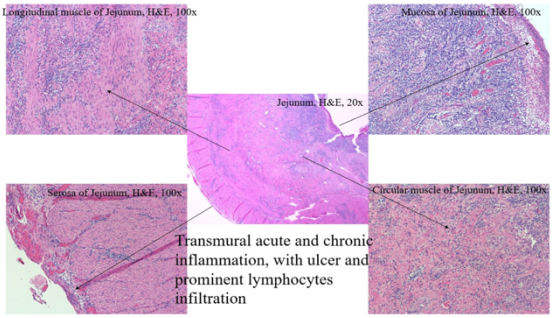

Figure 5: Histological slides of resected jejunum

Discussion

Stroke stands as the second primary cause of global mortality. Beyond its impact on the brain, it also affects diverse organs such as the lungs, heart, and gastrointestinal (GI) tract. Within the tract, stroke can disrupt metabolic control and nutritional homeostasis, leading to complications encompassing GI bleeding, fecal incontinence, dysphagia, immunosuppression leading to infections, and inflammation that can damage the intestinal mucosa [4,5]. The intestinal mucosa's barrier function plays a fundamental role in the contact between the brain and the gut, resulting in the expression of inflammation-related proteins [4,5,6]. In a recent study on rats, findings reported programmed cell death of intestinal cells, compromised intestinal mucosa barrier function, heightened intestinal permeability, and microbiota translocation after a traumatic brain injury, suggesting possible ischemia in segments of the bowel [5].

Ischemic enteritis (IE) arises from diminished arterial blood flow to the small intestine, accounting for only 0.1% of cases compared to ischemic colitis (IC) [3]. IC is caused by hypoperfusion of mesenteric vessels due to major vessel occlusion and typically occurs after an acute colonic insult for a short duration. IE, similar to the small intestine, requires surgical treatment for relief. IE is classified as a transient type in an acute phase, involving the abrupt constriction of the small intestine, and presenting as a stenotic type in its chronic phase [3]. Clinical presentations of IE include symptoms of abdominal pain in 94% of patients or vomiting in 61%, as revealed in a case study of 9,536 patients by Koshikawa et al [3,7]. An additional case series by Sada et al., revealed that 46.4% of those with stenotic IE had underlying conditions, such as hypertension, arrhythmia, ischemic heart disease, diabetes, or cerebral infarction.[8] Macroscopically, IE presents with distinct features including afferent tubular stenosis with well-defined margins and substantial thickening of the intestinal wall [9]. Histologically, IE is distinguished by a range of ulcer depths, an ulcer bed comprising granulated tissue with abundant vessels, pronounced fibrosis within the submucosal layer, marked infiltration of inflammatory cells, mainly lymphocytes, and differing degrees of hemosiderin-laden macrophages [3,8,9].

Crohn's disease (CD) can also contribute to the development of intestinal strictures. CD is a chronic inflammatory disorder of the GI tract with diverse clinical presentations. Isolated jejunal Crohn's disease (IJCD), constituting merely 1% of cases, is a rather uncommon manifestation of CD characterized by common symptoms such as abdominal pain, nausea, and vomiting [10]. In cases of IJCD, CT imaging typically shows jejunal thickening and proximal dilation, while biopsy reveals intestinal mucosa with ulceration and chronic inflammation [10]. For Crohn's disease, the

presence of stratified enhancement on.

imaging can be attributed to a combination of factors including granulation tissue, submucosal edema, inflammatory infiltration, intramural fat deposition, or fibrosis [2].

In our discussed case, the patient presented with an isolated instance of jejunal stenosis (JS). Although the patient experienced a stroke, it was a separate pathological event compared to the JS noted. This conclusion was based on the absence of further focal deficits that would have indicated the stroke as a major contributing factor to JS. Additionally, clinical assessments did not suggest that subsequent ischemic events, such as mesenteric ischemia, played a role in the development of the patient's JS. Furthermore, the patient did not have a history of chronic inflammatory bowel disease, which significantly reduced the likelihood of IJCD. The absence of this underlying condition indicates that other etiologies relating to IE should be considered, considering the histologic characteristics mentioned above.

Based on the presented case, several recommendations for further diagnostic considerations can be made. Firstly, radiological imaging such as an upper gastrointestinal series should be conducted to visualize the anatomy and identify any structural abnormalities within the jejunal region. Additionally, an endoscopic evaluation is warranted to directly visualize the mucosa and obtain tissue samples for histopathological examination, which can provide valuable insights into the nature of the lesions. Serologic testing should be administered to rule out conditions such as Crohn's disease, which may present with jejunal involvement. Furthermore, an infectious workup is essential to identify potential pathogens causing enteritis or other infectious etiologies of jejunal stenosis. Histopathological examination of biopsy samples obtained during endoscopy or surgical procedures can help characterize the nature of the jejunal lesions and guide further management decisions. Lastly, a collaborative multidisciplinary approach involving specialists from gastroenterology, radiology, pathology, and infectious disease should be employed to ensure a comprehensive evaluation, interpretation of results, and formulation of an appropriate management plan. By integrating these diagnostic strategies, healthcare providers can effectively identify the underlying etiology of jejunal stenosis and guide optimal patient care.

Conclusion

In conclusion, the case of a 54-year-old female with a recent left PCA stroke and a past medical history of GERD, hypertension, and HLD highlights the diagnostic challenge and successful management of jejunal stenosis (JS). Despite the initial difficulty in identifying the source of the patient's symptoms, the subsequent exploratory laparotomy revealed a stenotic segment of the jejunum, dense adhesions, and small bowel herniation. Nearly 15 cm of jejunum was resected, resulting in a significant improvement in the patient's symptoms and a favorable postoperative outcome.

The patient's clinical course, combined with the histological findings of transmural acute and chronic inflammation, ulceration, granulation tissue reaction, and hypertrophic muscularis propria, suggests that the jejunal stenosis was likely due to ischemic enteritis (IE). The successful surgical intervention, as evidenced by the absence of postoperative complications, the patient's return to a normal diet, and the restoration of gastrointestinal function, indicates a positive prognosis for the patient.

These outcomes punctuate the relevance of considering IE as a potential origin of jejunal stenosis in those with a history of stroke and persistent gastrointestinal symptoms. Additional research is warranted to further grasp the pathophysiology and optimal management approaches for JS related to stroke and its impact on the gastrointestinal tract. Increased awareness and early recognition of gastrointestinal complications

following stroke can lead to timely interventions, improved patient outcomes, and enhanced quality of life for affected individuals.

Furthermore, a comprehensive diagnostic approach involving radiological imaging, endoscopic evaluation, serologic testing, and histopathological examination, in conjunction with a multidisciplinary approach, is essential for accurate diagnosis and management of jejunal stenosis in similar cases.

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