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RESEARCH ARTICLE

WILKIE'S SYNDROME: CASE REPORT AND SURGICAL APPROACH IN A PATIENT WITH HIGH INTESTINAL OBSTRUCTION

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Abstract

Superior mesenteric artery syndrome (SMAS), also known as Wilkie's syndrome, is a rare and frequently underrecognized cause of upper intestinal obstruction. It occurs due to extrinsic compression of the third portion of the duodenum between the abdominal aorta and the superior mesenteric artery. This condition is anatomically defined by a decreased aortomesenteric angle and distance, typically resulting from significant loss of retroperitoneal fat following acute or chronic weight loss. We report the case of a 38-year-old male who presented with symptoms of high intestinal obstruction accompanied by unintentional and marked weight loss. A diagnosis of SMAS was confirmed via contrast-enhanced computed tomography (CT), which revealed a reduced aortomesenteric angle of 17° and a decreased distance of 7 mm.The patient was treated surgically through a lapa roscopic duodenojejunostomy in a Roux-eY configur ation, resulting in an uneventful postoperative course and satisfactor y recovery. This report underscores the importance of early clinical suspicion, timely imaging, and definitive surgical int ervention in the effective management of SMAS to p revent complications associated with prolonged obstruction and nutritional compromise.

Introduction:-

Superior mesenteric artery syndrome (SMAS) is an uncommon but clinically significant condition characterized by external vascular compression of the third portion of the duodenum. This compression occurs between the superior mesenteric artery (SMA) anteriorly and the abdominal aorta posteriorly, resulting in partial or complete duodenal obstruction. The narrowing of this space is primarily attributed to the loss of the intervening mesenteric fat pad, which normally maintains a protective cushion. This leads to a decreased aortomesenteric angle—normally between 38–65°—and a shortened distance, typically ranging from 10–33 mm. A reduction below these parameters predisposes to SMAS. The estimated incidence of this syndrome in the general population is extremely low, ranging from 0.013–0.3% [1], which contributes to frequent underdiagnosis and delays in treatment.

The clinical presentation is often vague and nonspecific, with symptoms that include early satiety, postprandial epigastric fullness, nausea, vomiting, and progressive weight loss. These features overlap with more common gastrointestinal disorders such as functional dyspepsia, peptic ulcer disease, or gastroparesis, which can lead to misdiagnosis or underappreciation of the underlying mechanical obstruction [2,3]. Consequently, imaging studies—especially contrast-enhanced computed tomography (CT)—are essential in identifying the anatomical alterations characteristic of SMAS.

Additional diagnostic modalities such as upper gastrointestinal series, Doppler ultrasound, or magnetic resonance angiography can also support the diagnosis. The syndrome may occur secondary to rapid catabolic states such as trauma, major burns, anorexia nervosa, malignancy, or chronic debilitating illnesses, which induce retroperitoneal fat depletion and predispose to this mechanical obstruction [4]. Moreover, surgical procedures such as scoliosis correction and bariatric surgery have been associated with postoperative SMAS.

Case Presentation

A 38-year-old male presented with a 7-day history of intense abdominal pain, We present the case of a 38-year-old male who arrived at our hospital with a 7-day history of colicky abdominal pain of moderate to severe intensity, persistent nausea, frequent vomiting, abdominal distension, and absence of flatus or bowel movements, suggestive of a high intestinal obstruction. His past medical history was notable for substance use, including tobacco, marijuana, and methamphetamine, along with a reported unintentional weight loss of approximately 20 kg over the preceding 3 months. The patient reported that food intake had become progressively intolerable, with early satiety and vomiting occurring after minimal oral consumption, which had led to progressive malnutrition and generalized weakness.

Initial laboratory evaluation revealed leukocytosis (20.8 x10^9/L), a slightly elevated serum creatinine of 1.34 mg/dL, a C-reactive protein (CRP) level of 1.69 mg/dL, and a procalcitonin value of 1.28 ng/mL. These findings were suggestive of an inflammatory response, though without clear evidence of sepsis or systemic compromise. Given the severity and progression of his symptoms, a contrast-enhanced abdominal CT scan was performed. The study demonstrated significant gastric and proximal duodenal dilatation, with a marked transition zone at the third portion of the duodenum. Notably, the aortomesenteric angle was measured at 17°, and the aortomesenteric distance was found to be 7 mm, both consistent with a diagnosis of SMAS.

Initial management included nasogastric decompression, intravenous fluids, electrolyte correction, and parenteral nutritional support. The patient was maintained in a semi-left lateral decubitus and prone position during feeding, which has been suggested to alleviate duodenal compression transiently.

Conservative measures were maintained for 5 days. However, the patient's clinical symptoms failed to improve satisfactorily. Given the persistence of symptoms and anatomical obstruction, the decision was made to proceed with definitive surgical management via a laparoscopic approach, after a multidisciplinary discussion including surgery, nutrition, and radiology.

The patient underwent a laparoscopic duodenojejunostomy with a Roux-en-Y configuration under general anesthesia. Intraoperatively, the third portion of the duodenum was mobilized using the Kocher maneuver, providing optimal exposure and tension-free access. A side-to-side anastomosis was created between the duodenum and jejunum using a 45 mm linear stapler. The enterotomies were closed with a continuous barbed suture (V-Loc 3-0), and the anastomosis was reinforced with interrupted serosal Lambert stitches using absorbable monofilament sutures to reduce the risk of leak. Estimated intraoperative blood loss was minimal, approximately 10 mL. The patient was monitored in a high-dependency unit during the initial 24 hours postoperatively. A contrast study at 24

hours confirmed adequate anastomotic flow and absence of leakage. Oral intake was resumed at 72 hours after verifying clinical stability and tolerance to clear liquids. The patient was discharged home on postoperative day 5 in good clinical condition, with nutritional follow-up and smoking cessation counseling.

Discussion:-

SMAS is a rare condition, frequently underdiagnosed due to the vague nature of Superior mesenteric artery syndrome is a rare yet important differential diagnosis in patients presenting with upper gastrointestinal obstruction and a recent history of significant weight loss. The etiology is fundamentally mechanical, resulting from a reduced aortomesenteric angle and distance, which compromises the duodenal lumen and impedes the passage of gastric contents. In our patient, this pathophysiological mechanism was clearly illustrated through imaging and correlated with the clinical scenario [5,6]. The gradual onset of symptoms, often attributed initially to functional disorders, tends to delay appropriate imaging and diagnosis, especially in patients with complex psychosocial or nutritional backgrounds.

Diagnostic modalities such as CT angiography or magnetic resonance imaging (MRI) play a pivotal role in confirming the anatomical criteria of SMAS. As established in the literature, an aortomesenteric angle <22° and a distance <8 mm are considered diagnostic thresholds [7]. These objective measurements, when combined with clinical context, provide strong evidence to support the diagnosis.

Conservative treatment, consisting of nutritional optimization, weight restoration, postural modifications, and gastric decompression, remains the initial strategy, especially in mild or subacute cases. However, in patients with severe or persistent symptoms, or when conservative measures fail—as was the case in our patient—surgical intervention is warranted. The decision for surgical management should balance the risks of malnutrition, aspiration, electrolyte imbalance, and potential progression to perforation or ischemia.
Among the surgical options available, laparoscopic duodenojejunostomy is currently regarded as the gold standard due to its high efficacy, lower complication rates, quicker postoperative recovery, and minimal invasiveness. This procedure effectively bypasses the obstructed duodenal segment, allowing restoration of gastrointestinal continuity and function [8,9]. Other surgical alternatives such as Strong's procedure or gastrojejunostomy have been described but are associated with higher failure or recurrence rates. Numerous studies have validated the outcomes of laparoscopic management, reporting significant symptom relief, early return of oral intake, and improved nutritional status [10]. Our case aligns with these findings, reinforcing the value of minimally invasive surgery in the treatment of SMAS and emphasizing the importance of timely surgical referral in non-responding patients.

Conclusion:-

Superior mesenteric artery syndrome should always be considered in patients presenting with high gastrointestinal obstruction in the context of recent, rapid weight loss. Despite its rarity, SMAS can have a significant impact on quality of life if left unrecognized or untreated. Prompt diagnosis through imaging and careful clinical evaluation are essential for initiating appropriate management. While conservative therapy may suffice in some cases, surgical intervention remains necessary for those with persistent symptoms or anatomical compromise. Laparoscopic duodenojejunostomy, as demonstrated in this report, is a safe and effective treatment option that offers excellent functional outcomes and rapid recovery in appropriately selected patients. Further awareness among clinicians can improve early detection and reduce morbidity associated with this overlooked syndrome.

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