Case Report

Superior mesenteric artery syndrome caused by acute weight loss in a 16-year-old polytrauma patient: A rare case report and review of the literature

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ARTICLE INFO

Keywords:
Superior mesenteric artery syndrome
Weight loss
Polytrauma

ABSTRACT

Introduction: Superior mesenteric artery syndrome is a rare entity, caused by compression of the duodenum between the superior mesenteric artery and the aorta.

Case presentation: A 16-year-old male in the inpatient service of our hospital complained of vomiting for two weeks. The patient had a long hospital stay in our center for the management of trauma-related complications, which resulted in significant weight loss despite the parenteral nutrition as he was unable for taking the enteral nutrition due to severe facial traumatic injury. In oral contrast study, the proximal duodenal segments were dilated. The contrast media could not reach the duodenojejunal junction in the supine position, though a small amount of the contrasts passed the distal duodenal part on repositioning the patient to prone. On abdominal CT images, the angle and distance between the superior mesenteric artery and the abdominal aorta were decreased to 20.8° and 7.3 mm respectively. The findings were consistent with the superior mesenteric artery compression syndrome. He underwent a mini-laparotomy for the placement of a jejunostomy feeding tube aiming to promote weight gain and mesenteric fat restoration for preventing the SMA compression effects on the duodenum.

Discussion: The decrease in retroperitoneal fat owing to weight loss may result in aortomesenteric angle reduction and duodenal compression. Prompt nutritional support and timely diagnosis may preclude the need for more invasive surgical intervention.

Conclusion: Superior mesenteric artery syndrome is a rare condition, often resulting in small bowel obstruction. The clinical symptoms of this syndrome are nonspecific, which may underestimate the diagnosis. However, clinical suspicion supported by imaging study may help the accurate diagnosis. Superior mesenteric artery syndrome should be considered in all polytrauma and longstanding immobile patients present with rapid weight loss and vomiting.

1. Introduction

Superior mesenteric artery syndrome is a rare entity, caused by compression of the duodenum between the superior mesenteric artery and the aorta, resulting in bowel obstruction [1].

The compression may be precipitated by progressive weight loss due to malignancy, dietary disorders, trauma, or surgery that reduces the aortomesenteric angle [2].

Clinically, the patients may have the manifestations of acute intestinal obstruction or more commonly may present with chronic insidious symptoms including recurrent episodes of abdominal pain, bilious vomiting, early satiety, and postprandial fullness [1,3]. These symptoms are usually increased post-prandially and relieved by lying in knee-elbow or left lateral position [1].

The diagnosis of SMA syndrome is often challenging due to its insidious onset, but clinical suspicion supported by imaging study may help the accurate diagnosis [4,5].

Conservative therapy aiming for weight gain to increase the retroperitoneal fat and the aortomesenteric angle is the mainstay of treatment. However, surgical therapy may be the next approach with duodenojejunostomy being the currently preferred treatment if the noninvasive management is not satisfactory [6].

Abbreviations: SMA, Superior mesenteric artery; CT, computed tomography.

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https://doi.org/10.1016/j.amsu.2021.102284
Received 25 February 2021; Received in revised form 4 April 2021; Accepted 4 April 2021
Available online 10 April 2021
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The authors present here an unusual case of superior mesenteric artery syndrome caused by acute weight loss in a 16-year-old polytrauma patient.

This work has been reported in line with the SCARE 2020 criteria [7].

2. Case presentation

2.1. Patient information

A 16-year-old male in the inpatient service of our hospital complained of vomiting for two weeks. He was the victim of a polytrauma after having fallen from the 4th floor of a housing block at midnight about two months ago. Though he had no known comorbidity before the trauma, he was an addicted drug user. The patient had a long hospital stay in our center for the management of trauma-related complications such as intracranial vascular aneurysms, aortic transection, lung contusion, splenic laceration, and skeletal bone fractures, which resulted in his significant weight loss despite the parenteral nutrition as he was unable for taking the enteral nutrition due to severe facial traumatic injury. In the physical exam, his blood pressure was: 132/80 mmHg, HR: 98 b/min, T: 36.6c, and RR: 22 cycle/min. There were signs of previous trauma on his face and jaws and a tracheostomy tube was observed in his neck. Other findings were unremarkable.

2.2. Laboratory and radiologic findings

The laboratory analysis yielded white blood cells: 6380/mm3, platelets: 355000/mm3, urea: 14 mg/dl, Creatinine: 0.41mg/dl Na: 141mEq/L, and K: 3.6mEq/L. He was negative for HBV, HCV, HIV, and Covid-19. In oral contrast study, the duodenal bulbous mucosa had a normal pattern and the proximal duodenal segments were dilated. The contrast media could not reach the duodenojejunal junction in the supine position. (a). On repositioning the patient to prone, consistent with superior mesenteric artery compression syndrome [Fig. 1a and b]. On abdominal CT images, no intraabdominal mass or lymphadenopathy was detected. The angle and distance between the superior mesenteric artery and the abdominal aorta were decreased to 20, 8°, and 7.3 mm respectively. The findings were compatible with the superior mesenteric artery compression syndrome [Figs. 2 and 3]. The Nasojejunal feeding tube was endoscopically placed by the gastroenterologist. However, this approach was not successful and the tube occluded after one day.

2.3. Therapeutic intervention

Following multidisciplinary consultation, the patient was taken up for surgical jejunostomy through a supra-umbilical median mini-laparotomy. A feeding tube of 10 f was placed in the distal jejunum and fixed with the skin. The bleeding was controlled, the tissue layers were closed properly, and the procedure was terminated without any complication. Close control follows up was recommended. As the patient has polytrauma therefore he will be followed in the different surgery units till complete recovery.

3. Discussion

Superior mesenteric artery compression syndrome was first described by Carl Von Rokitansky in 1861, though its pathophysiological association with the decreased aortomesenteric distance and reduced aortomesenteric angle explained with details by Wilkie in 1927 [3].

This syndrome is a rare entity, caused by compression of the duodenum between the superior mesenteric artery and the aorta, resulting in small bowel obstruction [1]. It is often seen in females and usually occurs in older children and adolescents with an incidence of 0.013–0.3% [6,8].

Many acquired and congenital factors are involved in the etiology of this syndrome. A low origin of the SMA or an abnormal high origin of Treitz ligament is the important congenital causes of SMA compression syndrome, while the loss of the retroperitoneal fat due to acquired reasons including polytrauma, burn, eating disorders, major surgical procedures, and cast immobilization, causing a reduction in the aortomesentric angle are the main acquired etiologic factors for superior mesenteric artery compression syndrome [1,9].

The normal aortomesentric angle and aortomesentric distance are 28–65° and 10–34mm, respectively [2]. The retroperitoneal fat and lymphatic tissues in this angle protect the duodenum from SMA compression [1]. In our case, the patient had a long hospital stay in our center for the management of trauma-related complications such as intracranial vascular aneurysms, aortic transection, lung contusion, splenic laceration, and skeletal bone fractures, which resulted in his significant weight loss despite the parenteral nutrition as he was unable for taking the enteral nutrition due to severe facial traumatic injury.

Although nausea, vomiting, recurrent postprandial pain, and bloating are the typical symptoms of this syndrome, the patient may have the manifestations of acute intestinal obstruction or chronic symptoms caused by intermittent bowel compression [10].

In a case series reported by Merrett. D.N et al., the SMA syndrome

![Oral contrast study images. (a). Dilated duodenum, the contrast media can’t pass the duodenojejunal junction in the supine position. (b). On repositioning the patient to prone a small amount of the contrast passed the distal duodenal part, consistent with superior mesenteric artery compression syndrome.](image-url)
should be well-thought-out as a cause of vomiting accompanied by substantial weight loss in young adults [11]. This was consistent with our case, as our patient presented with significant and rapid weight loss followed by vomiting.

The diagnosis of SMA syndrome is often challenging due to its insidious onset and atypical presentation that may delay the diagnosis and expose patients to more invasive treatments, but clinical suspicion supported by imaging study may help the accurate diagnosis [4,5,12].

Endoscopy, barium study, CT, and MR angiography can be used for diagnosis. The radiologic findings for SMA syndrome on barium study include duodenal obstruction with an abrupt narrowing in the third part of the duodenum [13]. In contrast-enhanced CT imaging, the reduction in the aortomesenteric angle to less than 22° and the aortomesenteric distance to less than 8-10 mm are defined as imaging criteria for SMAS diagnosis [9,14]. The same imaging findings were noted in our case.

The treatment is usually conservative that includes postural therapy and placement of a nasogastric tube to restore a normal aortomesenteric distance, promote weight gain, and relieves the obstruction [9,14-16]. However, overfeeding and underfeeding are the main limitations of this treatment that may have effects on the patient’s recovery [17].

Although conservative management is the standard treatment for uncomplicated cases of SMA syndrome, there is no clear time limit for this treatment and there have been reports of treatment lasting up to 169 days [18,19]. In the case of failure of conservative management, surgical intervention as gastrojejunostomy, duodenojejunostomy, or division of the ligament of Treitz can be performed [11]. The duodenojejunostomy is a relatively simple procedure with a success rate of 80–90%. However, current data in the literature do not support one surgical procedure over another as no randomized trials are available [13,20]. In our case, the conservative management was not successful. Thus, the patient underwent a mini-laparotomy for the placement of a jejunostomy feeding tube aiming to promote weight gain and restore the mesenteric fat pad.

This is an unusual and surgically proven case of superior mesenteric artery syndrome; however, lack of a long-time follow-up may be the only limitation for this case report.

4. Conclusion

Superior mesenteric artery syndrome is a rare condition, often resulting in small bowel obstruction. The clinical symptoms of this syndrome are nonspecific, which may underestimate the diagnosis. However, clinical suspicion supported by imaging study may help the accurate diagnosis. Superior mesenteric artery syndrome should be considered in all polytrauma and longstanding immobile patients presenting with rapid weight loss and vomiting. Prompt nutritional support and timely diagnosis may preclude the need for more invasive surgical intervention.

Ethics approval and consent to participate

The manuscript has got an ethical review exemption from the Ethical Review Committee (ERC) of our hospital, as case reports are exempted from review according to the institutional ethical review committee’s policy.

Funding

No financial support was provided for this study.
Authors’ contributions

Concept - HAE; Design – HAE; Supervision - HAE; Resources and data Collection- HAE; Literature Search - DMN; Writing Manuscript – HAE; Critical Review - DMN. All authors have read and approved the final manuscript.

Research registration

Not applicable.

Guarantor

The corresponding author (Habib Ahmad Esmat) is the guarantor for the work and he has the responsibility of access to the data and controlling the decision to publish.

Consent for publication

Written informed consent was obtained from the patient’s next of kin for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Provenance and peer review

Not commissioned, externally peer-reviewed.

Declaration of competing interest

The authors declare that they have no conflicts of interest for publication of this case report.

Acknowledgments

The authors would like to acknowledge Assoc. Prof. Dr. Gonca KOÇ for the radiologic diagnosis of this case and the EGE University Hospital PACS’s authorities for providing the patient information.

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