

Available online at www.sciencerepository.org

Science Repository



Case Report and Review of the Literature

Wilkie's Syndrome Following Weight Loss in a Trauma Patient

Oosthuizen GV*

Department of Surgery, Ngwelezana hospital, University of KwaZulu Natal, Empangeni, South Africa

ARTICLE INFO

Article history:

Received: 22 September, 2020

Accepted: 3 October, 2020

Published: 26 October, 2020

Keywords:

Wilkie's syndrome

superior mesenteric artery syndrome

cast syndrome

mesenteric root syndrome

chronic duodenal ileus

intermittent arterio-mesenteric

occlusion

ABSTRACT

Wilkie's syndrome, or superior mesenteric artery syndrome, is a rare condition of duodenal obstruction caused by compression of the superior mesenteric artery on the third part of the duodenum. The diagnosis should be considered in a patient who has experienced significant weight loss and now presents with persistent vomiting, especially if the vomiting occurs with the patient in the supine position and is alleviated by the lateral or prone position. The diagnosis can be confirmed by imaging studies demonstrating compression of the third part of the duodenum, and the main aim of treatment should be to pass a feeding tube beyond the point of obstruction to allow enteral feeding. The condition improves spontaneously with weight gain. Further treatment options include parenteral feeding and operative bypass in select cases. Here we present a case of Wilkie's syndrome in a trauma patient with significant weight loss, together with a review of the literature on this interesting topic.

© 2020 Oosthuizen GV. Hosting by Science Repository.

Introduction

Superior mesenteric artery syndrome or what is called Wilkie's syndrome is one of the rare causes of duodenal obstruction. Its exact incidence is not known. It occurs due to a decrease in the aorto-mesenteric angle following severe weight loss, leading to external compression of the third part of the duodenum.

Case Presentation

A 19-year-old man was admitted to hospital following a gunshot to his abdomen – entry wound left upper quadrant, exit wound just to the left of the midline posteriorly at level L1, with a fractured first lumbar vertebra and paraplegia. He underwent emergency laparotomy in the middle of the night; a single injury to the anterior wall of the distal transverse colon was identified and repaired. Subsequently, the patient became progressively unwell, and was taken for relaparotomy by a trauma surgeon at 30 hours post-op. A missed injury was identified to the posterior wall of the distal transverse colon, as well as a through-and-through injury to the left ureter at the renal pelvis. An extended right hemicolectomy was performed with end ileostomy and mucous fistula,

the left ureter was repaired over a double-J stent, and a drain was left in the region of the left renal hilum.

The ureter healed uneventfully, but the patient went on to have a number of repeat laparotomies, washouts, and abdominal wall debridements due to ongoing abdominal sepsis. He was managed with an open abdomen. He finally recovered sufficiently to eat and start mobilizing and was being prepared for skin grafting to his abdominal wound when he suddenly started to vomit copious amounts of bile (6 weeks post admission). Abdominal X-ray showed no signs of obstruction or ileus (normal small bowel gas pattern). He was given a gastrografin meal which was delayed in the stomach and only progressed into the second part of the duodenum after more than 12 hours.

Seeing as he had lost a large amount of weight during the above course of events and was now fairly emaciated, coupled with the fact that he was nursed predominantly on his back for his lumbar spine fracture and paraplegia, we suspected he may have Wilkie's syndrome. He was now on free nasogastric drainage (passing in excess of 500 mL per day) and on total parenteral nutrition. Upper endoscopy showed normal mucosa of stomach and duodenum. The endoscope was passed into the third part of the duodenum and revealed what looked like external compression.

*Correspondence to: Oosthuizen GV, F.C.S. (SA), Department of Surgery, Ngwelezana Hospital, Thanduyise Road, Empangeni, 3880, South Africa; E-mail: george.oost@gmail.com

CT scan was done next, which confirmed the suspected Wilkie's syndrome (Figures 1, 2 & 3).



Figure 1: CT image - distended proximal duodenum, collapsed beyond superior mesenteric artery.

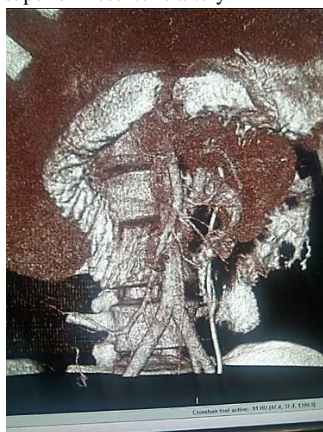


Figure 2: 3D reconstruction of the image seen in (Figure 1).



Figure 3: Saggital/lateral view on CT: superior mesenteric artery compressing duodenum posteriorly – contrast can be seen in the flattened duodenum.

A nasojejunal feeding tube was passed under endoscopic guidance. The patient was fed enterally and weaned from parenteral nutrition. He slowly gained weight and was mobilized with the help of his physiotherapists. After 4 weeks, the enteral tube feeding was discontinued, and he was allowed to start taking a graded diet orally. He progressed to full diet and the nasogastric tube was removed. He made an uneventful further recovery, received a skin graft to his abdominal wound, and was discharged in satisfactory condition after 12 weeks in hospital.

Discussion

Superior mesenteric artery syndrome (SMAS) is an uncommon condition thought to be caused by intermittent functional obstruction of the third part of the duodenum between the superior mesenteric artery anteriorly and the vertebral column and aorta posteriorly [1]. The entity was first described by von Rokitansky in 1861, discovered at autopsy [2]. Laffer presented one of the first reviews, and in 1927, Wilkie published the largest and most complete study of this disease, based on 75 cases [3, 4]. The existence of the condition was viewed with skepticism until the 1960s when new radiological techniques provided evidence to support the diagnosis [5].

SMAS goes by a variety of names, including Wilkie's syndrome, cast syndrome, mesenteric root syndrome, chronic duodenal ileus and intermittent arterio-mesenteric occlusion. It is a well-known complication of scoliosis surgery, anorexia, and trauma. It often poses a diagnostic dilemma; its diagnosis is frequently one of exclusion. The superior mesenteric artery usually forms an angle of approximately 45 degrees with the abdominal aorta, and the third part of the duodenum crosses caudal to the origin of the superior mesenteric artery, coursing between the superior mesenteric artery and aorta. Any factor that sharply narrows the aortomesenteric angle (e.g. loss of retroperitoneal fat, spinal corrective surgery for scoliosis, any surgery that distorts the anatomy) can cause entrapment and compression of the third part of the duodenum as it passes between the superior mesenteric artery and aorta, resulting in SMAS [6, 7]. Patients typically present with features suggestive of gastric outlet obstruction, which may be relieved when the patient is in the left lateral decubitus, prone, or knee-to-chest position, and aggravated when the patient is in the supine position [8].

The diagnosis of SMAS can be difficult. Confirmation usually requires upper GI series and CT scanning, revealing dilatation of the first and second portions of the duodenum with an abrupt narrowing at the third portion, and delayed gastroduodenal emptying. Additionally, the obstruction of the duodenum may be relieved by a change in position, especially left lateral decubitus position [9, 10]. Upper GI endoscopy may be necessary to exclude mechanical causes of duodenal obstruction. However, the diagnosis of superior mesenteric artery syndrome may be missed with this study. Abdominal ultrasonography may be helpful in measuring the angle of the superior mesenteric artery and the aortomesenteric distance. When combined with endoscopy, this may offer an alternative way to diagnose superior mesenteric artery syndrome in children to avoid other tests with a risk of radiation exposure [11].

Conservative initial treatment is recommended in all patients with superior mesenteric artery syndrome; this includes adequate nutrition, nasogastric decompression, and proper positioning of the patient after eating (i.e., left lateral decubitus, prone, or knee-to-chest position). Enteral feeding using a nasojejunal feeding tube passed distal to the obstruction under fluoroscopic assistance is an effective adjunct in treatment of patients with rapid severe weight loss while also eliminating the need for total parenteral nutrition. In some instances, both enteral and parenteral nutritional support may be needed to provide optimal calories. The patient's weight should be monitored daily. Once symptoms of obstruction start abating, graded oral intake may proceed. Metoclopramide treatment may be beneficial. The vast majority of cases should resolve on non-surgical therapy [12, 13].

Surgical intervention is indicated when conservative measures are ineffective, particularly in patients with a long history of progressive weight loss, pronounced duodenal dilatation with stasis, and complicating peptic ulcer disease. A trial of conservative treatment should be instituted for at least 4-6 weeks prior to surgical intervention. Options for surgery include a duodenojejunostomy or gastrojejunostomy to bypass the obstruction or a duodenal derotation procedure (otherwise known as the Strong procedure) to alter the aortomesenteric angle and place the third and fourth portions of the duodenum to the right of the superior mesenteric artery [13, 14].

Conclusion

SMAS is a rare cause of duodenal obstruction, but the diagnosis should be borne in mind. Persistent vomiting after significant weight loss should raise the suspicion of this diagnosis. Upper GI endoscopy should be done to exclude other mechanical causes of duodenal obstruction and contrast enhanced CT scan is useful in definitive diagnosis. Nonoperative management is successful in most cases.

Conflicts of Interest

None.

REFERENCES

1. Gustafsson L, Falk A, Lukes PJ, Gamklou R (1984) Diagnosis and treatment of superior mesenteric artery syndrome. *Br J Surg* 71: 499-501. [[Crossref](#)]
2. Rokitsky CV (1861) *Lehrbuch der pathologischen Anatomie*. Vienna: Braumuller & Seidel.
3. Laffer LC (1908) Acute dilatation of the stomach and acute arteriomesenteric ileus. *Ann Surg* 47: 94-97.
4. Wilkie DP (1927) Chronic duodenal ileus. *Am J Med Sci* 173: 643-649.
5. Cimino CV (1961) Arteriomesenteric occlusion of the duodenum: an entity? *Radiology* 76: 828-829. [[Crossref](#)]
6. Cohen LB, Field SP, Sachar DB (1985) The superior mesenteric artery syndrome. The disease that isn't, or is it? *J Clin Gastroenterol* 7: 113-116. [[Crossref](#)]
7. Kaur A, Pawar NC, Singla S, Mohi JK, Sharma S (2016) Superior mesenteric artery syndrome in a patient with subacute intestinal obstruction: a case report. *J Clin Diagn Res* 10: TD03-TD05. [[Crossref](#)]
8. Smith BG, Zargar MH, Thomson JD (2009) Low body mass index: a risk factor for superior mesenteric artery syndrome in adolescents undergoing spinal fusion for scoliosis. *J Spinal Disord Tech* 22: 144-148. [[Crossref](#)]
9. Welsch T, Buchler MW, Kienle P (2007) Recalling superior mesenteric artery syndrome. *Dig Surg* 24: 149-156. [[Crossref](#)]
10. Agrawal GA, Johnson PT, Fishman EK (2007) Multidetector row CT of superior mesenteric artery syndrome. *J Clin Gastroenterol* 41: 62-65. [[Crossref](#)]
11. Sundaram P, Gupte GL, Millar AJW, McKiernan PJ (2007) Endoscopic ultrasound is a useful diagnostic test for superior mesenteric artery syndrome in children. *J Pediatr Gastroenterol Nutr* 45: 474-476. [[Crossref](#)]
12. Shiu JR, Chao HC, Luo CC, Lai MW, Kong MS et al. (2010) Clinical and nutritional outcomes in children with idiopathic superior mesenteric artery syndrome. *J Pediatr Gastroenterol Nutr* 51: 177-182. [[Crossref](#)]
13. Zaraket V, Deeb L (2015) Wilkie's syndrome or superior mesenteric artery syndrome: fact or fantasy? *Case Rep Gastroenterol* 9: 194-199. [[Crossref](#)]
14. Ha CD, Alvear DT, Leber DC (2008) Duodenal derotation as an effective treatment of superior mesenteric artery syndrome: a thirty-three year experience. *Am Surg* 74: 644-653. [[Crossref](#)]