ORIGINAL RESEARCH PAPER

Volume-7 | Issue-6 | June-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A RARE CASE OF WILKIE SYNDROME

General Surgery

Dr. Renu Post Graduate, Department of General Surgery, Government Medical College and Hospital, Sector-32, Chandigarh.

Dr. Kumar Abhishek* Assistant Professor, Department of General Surgery, Government Medical College and Hospital, Sector-32,Chandigarh. *Corresponding Author

Dr. Rajesh Kumar Bansiwal Associate Professor, Department of General Surgery, Government Medical College and Hospital, Sector-32,Chandigarh.

Dr. Rajeev Sharma Professor, Department Of General Surgery, Government Medical College And Hospital, Sector-32, Chandigarh

ABSTRACT

Superior mesenteric artery syndrome (SMAS) is a rare condition, also known as Wilkie's syndrome, cast syndrome and arteriomesenteric duodenal compression. We report a case of 26 years old male patient admitted for head trauma and bedridden with recent weight loss who developed SMAS.

KEYWORDS

superior mesenteric, arteriomessenteric, bedridden

INTRODUCTION

Superior mesenteric artery syndrome (SMAS) is a rare condition, also known as Wilkie's syndrome, cast syndrome and arterio mesenteric duodenal compression. It is caused by compression of the third portion of the duodenum between aorta and superior mesenteric artery (SMA). A reduced space and angle between aorta and SMA causes a duodenal compression resulting in high intestinal obstruction. Most frequently it occurs in young patients who have had a significant weight loss. Surgeries for spinal deformities as well as high insertion of the ligament of Treitz are other potential causes for the occurrence of SMA syndrome. Loss of retroperitoneal fatty tissue as a result of this variety of conditions is believed to be the etiologic factor causing the acute angulation. Symptoms vary from postprandial nausea and bilious vomiting to abdominal pain as well as weight loss and can occur acutely or chronically. The severity of the symptoms largely depends on the degree of the compression as reflected by the aortomesenteric angle.[1]We report a case of 26 years old male patient admitted for head trauma and bedridden with recent weight loss who developed SMAS.

CASE REPORT

A 26-year-old male patient admitted in high dependency unit of our hospital with alleged history of road traffic accident in which he sustained head injury (pneumocephalus with subdural hematoma) with bilateral maxillary fractures. Patient remained bedridden for 30 days. On forty fifth day of admission, patient developed bilious vomiting and epigastric discomfort not tolerating oral feeds. Over the due course of hospital stay patient had a weight loss of approximately 10 kilograms. Laboratory results were unremarkable. Ryles tube was placed and there was an output of more than 2 litres per day for 2 days. Abdominal X-ray showed distension of the stomach with air fluid level. Contrast enhanced Abdominal CT-scan showed dilated stomach, first and second portion of the duodenum. The third part of duodenum appeared compressed between the aorta and SMA due to a small aorto-mesenteric distance of 6mm and aorto-mesenteric angle of 14 degree.

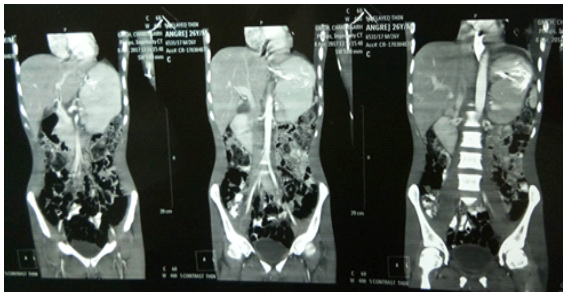

Figure 1 CECT abdomen

The diagnosis of SMA syndrome was made supported by radiological evidence. Patient was prepared for surgery and exploratory laparotomy with loop duodenojejunostomy ,in which a loop of jejunum around 15cm distal to the ligament of treitz brought in a retrocolic fashion is anastomosed with third part of duodenum was performed. The postoperative period was uneventful. The patient was discharged on the seventh postoperative day. At the follow-up visit at 1 month and 3 months the patient had complete resolution of his preoperative symptoms.

DISCUSSION

SMA syndrome was first described by Rokitansky in 1842. The pathogenic mechanism, as described by Wilkie in 1927, involves a small aorto-mesenteric space and extrinsic compression of the third portion of the duodenum between the SMA and the aorta. It is rare condition with prevalence between 0.013 % and 0.3 percent[2] . Etiological factors can be either a congenital or an acquired anatomic abnormality or, more commonly, a debilitating condition causing severe weight loss. Congenital etiologies include abnormally low insertion of the SMA or high insertion of the angle of Treitz dislocating the duodenum to a cranial position. Acquired anatomic abnormalities can occur following corrective spinal surgery such as scoliosis surgery by a relative lengthening of the spine[3], spinal trauma, and after abdominal surgery such as total proctocolectomy and ileal J-pouch anal anastomosis due to tension and caudal pull of the small bowel mesentery .[4] Severe weight loss, as in our patient, leading to a depletion of the fatty cushion around the SMA is a major cause of SMA syndrome. Catabolic states like burns[5], eating disorders such as anorexia nervosa [6], or wasting conditions such as neoplastic diseases and malabsorptive states are the most commonly reported reasons of drastic weight loss [7].A massive and rapid reduction in the thickness of adipose tissue of the aorto- mesenteric space normally helps to keep the aortomesenteric angle open and protects the duodenum of the vascular compression [8,9]. A Wambre-Nicolas *et al*. demonstrated that Aortomesenteric angle's width is related to the body mass index[10].

In our patient the etiology of SMAS may be related to rapid weight loss with low BMI. There are two non-specific clinical forms: The most common form (90%) is made of chronic intermittent epigastric pain, nocturnal bilious vomiting worsening in supine position and improving by the left lateral or the seated positions. The second form, such as our case, is a rare form made of a severe bowel obstruction which can be life-threatening [11]. The diagnosis is confirmed by contrast enhanced CT abdomen, which shows a gastroduodenal expansion to the third duodenal portion. The Arteriographic criteria include a significantly decreased aorto-SMA angle of 6° to 25° (normal-45°) and a shortened aortomesenteric distance of 2 to 8 mm (normal-10 to 20 mm) [12], which is the case in our observation.
Traditionally treatment has consisted of conservative measures such as

gastric decompression, parenteral nutrition and/or post-pyloric feeding when possible, followed by oral diet as tolerated [13].Posturing manoeuvres during meals and motility agents may be helpful in some patients. No time limit has yet been defined for the medical treatment. Surgery may be considered if conservative treatment fails [14]. Duodenojejunostomy is the operation of choice to relieve the obstruction, with a success rate up to 90% [14].Another less invasive surgical option, known as Strong's procedure, involves lysis of the ligament of Treitz with mobilization of the duodenum; major advantage of this procedure is avoidance of anastomosis but, this operation had a failure rate of 25% [7]. Gastrojejunostomy, a previously reported surgical treatment, has been abandoned because of increased postoperative complications like blind loop syndrome and recurrence of symptoms due to non-decompression of the duodenum.

CONCLUSION

SMAS is a rare clinical condition that is clinically a diagnosis of exclusion. Especially in patients with severe weight loss and symptoms of high intestinal obstruction SMAS should be considered as a differential. Conservative management may be sufficient in some cases but the surgical treatment of choice is duodenojejunostomy.

REFERENCES

1. Record JL, Morris BG, Adolph VR: Resolution of refractory superior mesenteric artery syndrome with laparoscopic duodenojejunostomy: pediatric case series with spectrum of clinical imaging. Ochsner J 2015;15:74–78.
2. Unal B, Aktas A, Kemal G. Superior mesenteric artery syndrome: CT and ultrasonography findings. Diagn Interv Radiol 2005; 11:90-5
3. Tsirikos AI, Anakwe RE, Baker AD: Late presentation of superior mesenteric artery syndrome following scoliosis surgery: a case report. J Med Case Rep 2008;2:9.
4. Ballantyne GH, Graham SM, Hammers L, Modlin IM: Superior mesenteric artery syndrome following ileal J-pouch anal anastomosis. An iatrogenic cause of early postoperative obstruction. Dis Colon Rectum 1987;30:472–474.
5. Reckler JM, Bruck HM, Munster AM, Curreri PW, Pruitt BA Jr: Superior mesenteric artery syndrome as a consequence of burn injury. J Trauma 1972;12:979–985.
6. Verhoef PA, Rampal A: Unique challenges for appropriate management of a 16-year-old girl with superior mesenteric artery syndrome as a result of anorexia nervosa: a case report. J Med Case Rep 2009;3:127.
7. Merrett ND, Wilson RB, Cosman P, Biankin AV: Superior mesenteric artery syndrome: diagnosis and treatment strategies. J Gastrointest Surg 2009;13:287–292.
8. Bonnet JP, Louis D, Foray P. La pince aortomesenterique.Arch Pediatr 1995;2:333 8
9. Ortiz C, Cleveland RH, Blickman JG, Jamarillo D, Kim SH. Familial superior mesenteric artery syndrome. Pediatr Radiol 1990; 20:588-9.
10. Wambre-Nicolas A, Pennec VL, Geffard B, Ardouin L, Schmutz G, Salamé E. ANAT4 La pince aorto-mesenterique : correlation entre radio-anatomie et indice de masse corporelle. Journal de Radiologie 2006; 87:1436.
11. Kalouche I, Leturgie C, Tronc F, Bokobza B, Michot F, Pons P. Le syndrome de la pince mésentérique. A propos d'une observation et revue de la littérature. Ann Chir 1991; 45:609-62.
12. Loeb T, Loubert G, Morsly R, Gabillet JM, Pasteyer J. Syndrome de l'artère mésenterique supérieure. Ann Fr Anesth Reanim 1999; 18:1000- 4.
13. Welsch T, Buchler MW, Kienle P: Recalling superior mesenteric artery syndrome. Dig Surg 2007;24:149–156.
14. Mandarry M, Zhao L, Zhang C, Wei Z: A comprehensive review of superior mesenteric artery syndrome. Eur Surg 2010;42:229–236.