Case report

“Less is more”: Non operative management of short term superior mesenteric artery syndrome

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HIGHLIGHTS

- We present a case of rare superior mesenteric artery syndrome.
- No apparent predisposing factor was present.
- Non operative management with decompression and enteral feeding proved to be successful.
- Non operative management with nutritional supplementation remains the first line of therapy.

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ABSTRACT

Introduction: Superior mesenteric artery (SMA) syndrome is a relatively rare aetiology of proximal intestinal obstruction. This is caused by narrowing of vascular angle of SMA and aorta compressing the third part of the duodenum (D3). Predisposing factors may include precipitous weight loss, corrective spinal surgery or repair of an aortic aneurysm.

Presentation of case: A 53 year old male presented to our department with worsening post-prandial vomiting and epigastric pain for last three months. One examination, epigastric region was distended with succussion splash on abdominal auscultation. History did not include any predisposing factor. CT scan showed narrow angle of 12.77° between SMA and aorta owing to the compression of D3. Since onset of vomiting and resultant poor oral intake, he had lost 11 kg of his usual body weight. After gastric decompression, nasojejunal enteral feeding was started. Diet was progressed to oral feedings gradually and following return to his baseline weight, he continued to be free of symptoms in follow-up visits.

Discussion: Although there are recognised predisposing factors but sometimes aetiology remains idiopathic. SMA syndrome should initially be managed non-operatively with gastric decompression, correction of water and electrolyte imbalance, and hemodynamic instability. Regaining weight helps increasing vascular space between SMA and D3 thus relieving obstruction. Persistence of symptoms beyond 3–4 weeks warrants surgical intervention.

Conclusion: Non operative management with nutritional supplementation remains the first line of therapy.

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1. Introduction

Superior mesenteric artery (SMA) syndrome is a relatively rare aetiology of proximal intestinal obstruction. This is caused by narrowing of vascular angle of SMA and aorta compressing the third part of the duodenum (D3). Predisposing factors may include precipitous weight loss, corrective spinal surgery or repair of an aortic aneurysm.

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2. Presentation of case

A 53 year old male presented to our department with worsening post-prandial vomiting and epigastric pain for last three months. Vomitus contained of bilious contents. Other associated symptoms included abdominal bloating and constipation for three days. His usual weight was 70 kg prior to the onset of vomiting. Previous history did not include any intentional weight loss, spinal surgery or malignancy. Clinical examination demonstrated the distended epigastric region with mild tenderness and succussion splash on abdominal auscultation. Haematological investigations were
unremarkable except for leucocytosis. CT scan showed markedly distended stomach and proximal duodenum. There was a transition of calibre at the third part of duodenum between Superior Mesenteric Artery (SMA) and aorta accounting for proximal obstruction. Pancreas was compressed extrinsically by distended stomach. Left renal vein was also compressed between aorta and SMA, also known as Nutcracker sign (Fig. 1).

Nasogastric tube (NGT) was inserted and intravenous rehydration commenced. Nasogastric tube drained 4.7 L over first 24 h. Since onset of emesis, he had lost 11 kg of his usual body weight. Concomitant total parenteral nutrition (TPN) with essential vitamins, albumin and dietitian’s input were initiated. Upper gastrointestinal endoscopy was conducted to rule out any sinister lesion. Examination was unremarkable. A nasojejunal (NJ) tube was inserted for promoting enteral feeding. (Fig. 2). Its position was confirmed on abdominal X-ray.

On 7th admission day, his TPN was gradually reduced with introduction of clear fluids through NJ tube. He gradually improved with regaining weight. After discussing with upper GIT surgical team, he was transferred to specialist centre for further management. In view of his improving symptoms and nutritional status, a repeat CT scan was conducted which showed decompression of third part of duodenum (D3) and reduction of 50% of compression of left renal vein. (Fig. 3).

On 17th day, NJ tube was removed and trial of oral diet was given which was well tolerated. He was subsequently discharged and continued to gain weight with no recurrence of obstruction on further follow up visits at 3rd and 5th month.

3. Discussion

SMA syndrome, as described in 1861 by Von Rokitansky [1], is a rare vascular cause of proximal intestinal obstruction. This interesting phenomenon is caused by narrow angle between SMA and aorta compressing third part of duodenum. Predisposing factors which decrease acuity of this angle include spinal procedures, rapid linear growth, and corrective surgery for scoliosis, lumbar lordosis and abnormally high position of ligament of Treitz [2]. Acute weight loss is most commonly implicated in aetiology. It results in altering aortomesenteric angle by attenuating the fat pad between these vascular structures compressing D3 [3]. The superior mesenteric artery arises from the anterior aspect of the aorta at the level of the L1 vertebral body. It is encased in fatty and lymphatic tissue and extends in a caudal direction at an acute angle into the mesentery. In the normal individual, this adipose tissue pad displaces the SMA anteriorly away from the aorta so avoiding duodenal compression.

The aortomesenteric distance is normally 10–28 mm which may reduce to 2–8 mm in SMA syndrome [4]. The main anatomical feature of this syndrome is the narrowing of the angle between the SMA and aorta resulting in functional obstruction.

The cause of narrowing can be two-fold. It may be secondary to the alteration of the relationship of the vascular structures forming

Fig. 1. CT images showing D3 obstruction and nutcracker sign (arrow and arrowhead respectively).

Fig. 2. Endoscopic placement of NJ tube.

Fig. 3. Decompressed stomach with tip of NJ tube in Jejunum.
this angle as with the relative lengthening of the spine following surgery. The other more prevalent aetiology is postulated to be related to the loss of intra-abdominal adipose tissue or its attenuation secondary to the sudden weight loss leading to narrowing of the angle. Regardless of the aetiology, once the condition is established, self-perpetuating nature of vomiting leads to further weight loss and thus further vomiting [5]. Ongoing vomiting due to obstruction and resultant malnutrition further accentuates this problem. As in this case, history was unable to elucidate any primary aetiological factor for the onset of SMA syndrome. His initial body weight was 70 kg prior to the onset of the symptoms. It was unknown what might have set this off but once continuous vomiting ensued, his resultant loss of weight and attenuation of mesenteric fat pad was contributory to the further narrowing of the angle and worsening symptoms. Nutritional replacement not only helped him gaining weight but also improving vascular space with increasing mesenteric fat and adipose tissue around SMA.

Nature of non-specific symptoms on clinical examination often delays its diagnosis. Here role of CT is imperative as SMA syndrome is essentially a CT based diagnosis [6]. Normal range (38–56°) of vascular angle has been debated a lot in literature. Narrow angle of 25° or less is more likely to cause obstruction, as was 12.77° in our patient. Other investigations include endoscopy, CT angiography or Endoscopic ultrasound.

Initial resuscitation includes NGT placement and institution of fluid and electrolyte replacement. In the absence of established predisposing factors like spinal or aortic surgery, increase in vascular space with widening of aorto-mesenteric angle helps in alleviating the obstructive symptoms. Trial of conservative management improves the nutritional deficit with gaining weight [7]. It may prove to be definitive management as weight gain is associated with increase fat content between two vessels and release pressure on D3. On repeat CT scan for our patient, there was a noticeable increase in aorto-mesenteric angle (47.2°) leading to increase in vascular space.

Alternative nutrition like TPN or enteral feeding through jejunostomy can enhance replenishing anabolic stores [8]. However these are still invasive and not complications-free. In refractory cases, where no improvement beyond 3–4 weeks, procedures like duodenojejunostomy or Treitz ligament division (Strong’s operation) may be necessary [9]. In our patient, gastric decompression and NJ feeding finally improved the outcome. Encouraging oral feed after NJ removal also accelerated the return to basal weight and subsequently excluding the need for surgical intervention.

Ethical approval

No ethical approval was required or requested for this case report.

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Author contribution

Zainab Naseem — primary author of the case report, literature review and writing of manuscript.
Gamini.Premaratne — literature review and editing.
Rasika Hendahewa — Concept of manuscript, literature review and editing.

Conflicts of interest

There was no financial or personal relationships which could influence bias in this case report.

Guarantor

Gamini.Premaratne. Zainab Naseem.

Consent

Consent was obtained from patient. All details of the patient, in the images attached with the case report, have been deleted and patient remains anonymous.

Registration of research studies

UI#researchregistry459.

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