Superior mesenteric artery syndrome in a young girl following spinal surgery for scoliosis

Introduction

The superior mesenteric artery (SMA) syndrome is a rare condition thought to result from extrinsic compression of the third portion of the duodenum, where it is crossed by the SMA as it descends from the abdominal aorta. The diagnosis is made clinically, but can be confirmed radiologically with an upper gastrointestinal series (Shapiro et al, 2001).

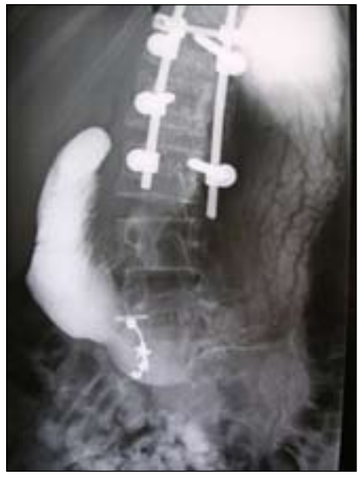
SMA syndrome, also known as cast syndrome, Wilkie's syndrome, arteriomesenteric duodenal compression syndrome and chronic duodenal ileus, is a unique form of high intestinal obstruction as a result of entrapment of the third portion of the duodenum by a decrease in the angle between the SMA and the abdominal aorta (Wilson-Storey and Mackinlay, 1986). Although rare it is seen in orthopaedic patients who undergo spinal surgery or who are in hip spica or body casts.

This article presents a case report of a 15-year-old girl who developed SMA syndrome following spinal surgery for scoliosis. Increased awareness of this condition may result in early diagnosis and timely management, thus reducing its potential morbidity and complications.

Discussion

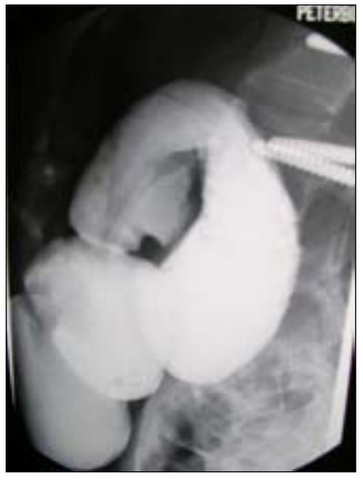
SMA syndrome was first described by von Rokitansky in 1861 (Shetty and Schmidt-sommerfield, 1999) and was more extensively analysed by Wilkie (1921). The SMA is the second branch of the abdominal aorta and supplies the entire small bowel. The duodenum lies in the angle between the SMA anteriorly and the abdominal aorta and spine posteriorly. Any factor that reduces this angle causes compression of the third part of duodenum resulting in SMA syndrome.

Figure 1. Barium meal study demonstrating hugely distended stomach with spinal fixation rods in the background.



There are various theories to explain the association with the scoliosis corrective surgery and the SMA syndrome. First, similar to the patient described in the case report, children usually present for surgical correction of scoliosis during their growth spurt, when rapid musculoskeletal growth may alter the relation between the spine, the SMA and the duodenum. Second, surgical correction produces upward tension on the SMA root and the mesentery, decreasing the SMA angle and compressing the duodenum. Third, the relatively more extended spine in scoliosis results in increased vertical tension on the soft tissues anterior to the spine, including the SMA.

Figure 2. Barium meal study oblique view showing obstruction in the third part of duodenum with grossly distended proximal duodenum and stomach with tip of nasogastric tube.



The conditions that can predispose patients to SMA syndrome are casting and increased lordosis that can be caused by scoliosis surgery, prolonged bed rest, rapid weight loss from anorexia nervosa and malabsorption syndromes. The classic symptoms of the syndrome are postprandial abdominal fullness and vomiting, abdominal distension and epigastric pain (Shetty and Schmidt-Sommerfield, 1999). Early diagnosis is sometimes made difficult by non-specific and intermittent symptoms.

The clinical diagnosis is confirmed with a sharp cut-off of contrast material in the second or third part of the duodenum where it crosses over the spine at the L3–L4 disc space. Fluoroscopy and hypotonic duodenography have been used to depict the site of obstruction. A contrast enhanced spiral computed tomography can provide diagnostic information including the aorto-SMA distance, duodenal distension and amount of intra-abdominal and retroperitoneal fat. The arterio-mesenteric angle can be determined by conventional or magnetic resonance angiography (Gockel et al, 2004).

Therapy is aimed at breaking the cycle of duodenal oedema, duodenal compression by the SMA and resultant obstruction. Patients are treated with intravenous fluid and nasogastric suction. If the patient is wearing a cast, it should be removed to check for reversal of symptoms. The removal of spinal hardware is rarely warranted. A jejunostomy tube can be inserted past the obstruction to allow continuous nutritional support, and hyperalimentation can be used if symptoms do not resolve after several days. For refractory cases various surgical procedures have been described, including duodeno-jejunostomy, gastro-jejunostomy, and division of the ligament of Treitz. **BJHM**

Gockel I, Graupe F, Hain J et al (2004) Superior mesenteric artery (Wilkie) syndrome: different faces. *J Gastroenterol Hepatol* **19**(6): 720–2

Shapiro G, William DG, Nunzia S et al (2001) Medical complications in scoliosis surgery. *Curr Opin Paediatr* **13:** 36–41

Shetty AK, Schmidt-Sommerfield E (1999) Radiological case of the month - superior mesenteric artery syndrome. *Arch Paediatr Adolesc Med* **153:** 303–4

Wilkie DPD (1921) Chronic duodenal ileus. *Br J Surg* **9:** 204–14

Wilson-Storey D, Mackinlay GA (1986) The superior mesenteric artery syndrome. *J R Coll Surg Edinb* **31:** 175–8

Case Report

A 15-year-old girl was admitted with nausea, severe bilious vomiting, loss of appetite and loss of weight of 2 weeks' duration. She underwent spinal surgery for thoracic scoliosis with multiple congenital abnormalities of the spine 3 weeks before admission. Examination revealed a thin, dehydrated patient with distended upper abdomen, positive succussion splash and normal bowel sounds. Blood investigations including urea and electrolytes were normal except for mild leucocytosis. Abdominal X-ray was unremarkable. A nasogastric tube was inserted which drained 2–3 litres of bilious fluid each day. She was treated conservatively with intravenous fluids and anti-emetics.

The history of spinal surgery and copious amount of bilious nasogastric aspirate suggested upper gastrointestinal obstruction for which a barium meal with follow through was done on day 3 (*Figures 1* and *2*). It revealed a distended stomach and dilatation of the first and second parts of the duodenum with an abrupt linear cut-off at the third part, suggesting extrinsic compression. On the basis of her history and radiological findings, the diagnosis of superior mesenteric artery syndrome was made. Conservative treatment was continued with total parenteral nutrition for about 3 weeks, by which time the nasogastric output gradually reduced to less than 100 ml and the patient was tolerating oral fluids

The patient was discharged after 4 weeks on soft diet. She was reviewed in clinic after a month and was found to be completely asymptomatic and comfortable.

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CASE REPORT