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CASE REPORT

Wilkie's syndrome: a fortuitous finding

IN Palkowski, K Polden

General Surgery, George Regional Hospital, South Africa

Corresponding author, email: *ivan.palkowski@westerncape.gov.za*

Summary
Wilkie's syndrome, also known as superior mesenteric artery (SMA) syndrome, is a rare condition characterised by the compression of the third portion of the duodenum between the abdominal aorta and the overlying SMA. Due to its rarity, the exact number of documented case reports is limited. As of recent literature, approximately 500 cases have been reported worldwide with a reported incidence of 0.013–0.3%.
Keywords: superior mesenteric artery syndrome, Wilkie's syndrome

Case report

A 39-year-old female with no known comorbidities underwent a total abdominal hysterectomy (TAH) for a symptomatic multi-fibroid uterus. The surgery was uneventful, and the patient was discharged on the second postoperative day.

The patient presented 2 weeks later with features of adhesive bowel obstruction. She had reported bile-stained vomitus and obstipation. Incidentally, the patient also reported intermittent bilious vomiting persisting for over 10 years, accompanied by a gradual, progressive loss of weight. These symptoms began following a caesarean section for the birth of her first child. Notably, no endoscopic evaluations had been performed at the time. Clinically, the patient was dehydrated, and tachycardic but normotensive. The abdomen was distended but soft and non-tender. There were no systemic signs of sepsis. An abdominal X-ray demonstrated a mid- small bowel obstruction.

A venous blood gas revealed a significant hyponatraemic hypokalaemic metabolic alkalosis more consistent with a gastric outlet obstruction (Table I). Her urea and creatinine were of normal value.

Table I: Patient values and normal range

Laboratory index	Patient value	Normal range
pH	7.6 ↑	7.35–7.45
HCO_3 (mmol/L)	60 ↑	22–26
Lactate (mmol/L)	3.3 ↑	< 2
Base excess (mmol/L)	46.1 ↑	-2 to +2
Sodium (mmol/L)	122 ↓	136–145
Potassium (mmol/L)	2.15 ↓	3.5–5.1
Chloride (mmol/L)	73 ↓	96–106

A full blood count revealed an elevated white cell count (WCC) of 17.2 x 109/L (normal values 3.9–12.6 x 109/L). The haemoglobin and platelet counts were normal. Calcium, magnesium, and phosphate were normal.

The patient was resuscitated with intravenous crystalloid fluids over four hours, and the electrolytes were replaced. A repeat venous blood gas showed a moderate improvement but ongoing hyponatraemic hypokalaemic metabolic alkalosis.

Conservative management that comprised of decompressive nasogastric drainage, nil per mouth and intravenous rehydration fluids was attempted for 48 hours but was unsuccessful. A venous blood gas was repeated and revealed a return to normal values.

Failure of resolution of the adhesive bowel obstruction to conservative measures prompted further investigations. The patient could not tolerate gastrografin and therefore a contrasted abdominal computerised tomography (CT) scan was performed which showed duodenum interposed between the abdominal aorta and superior mesenteric artery (SMA) (Figure 1). The aortomesenteric angle (AOM) was 14.5° suggesting SMA syndrome (Figure 2). Additionally, there was adhesive small bowel obstruction with a point of obstruction in the proximal ileum (Figure 3).

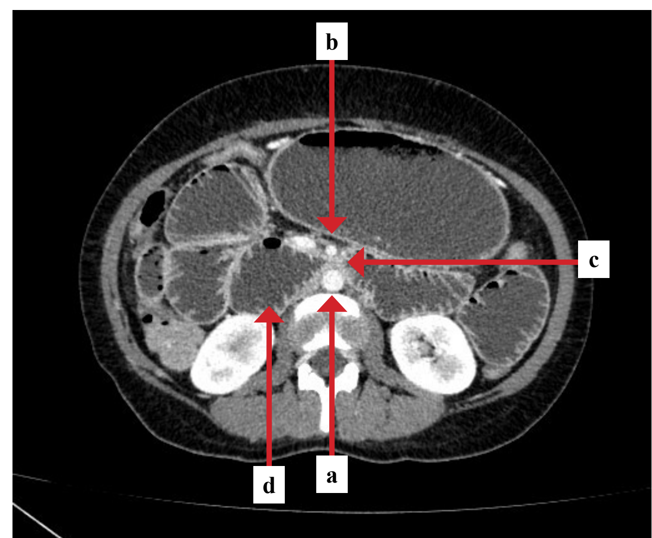

Figure 1: CT scan of the abdomen demonstrating compression of the duodenum between the aorta and SMA; a) aorta, b) SMA, c) duodenum; d) small bowel obstruction distal to this compression point can be seen

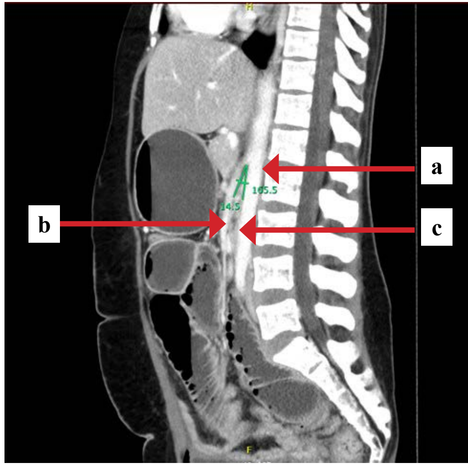

Figure 2: Sagittal view of a CT of the abdomen demonstrating an aortomesenteric angle of 14.5°; a) aorta, b) SMA, c) duodenum

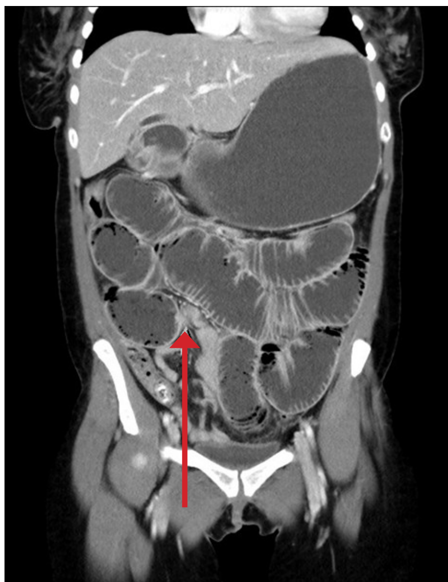

Figure 3: CT scan of the abdomen demonstrating adhesive small bowel obstruction with a point of obstruction in the proximal ileum

An exploratory laparotomy demonstrated an adhesive bowel obstruction at the level of the proximal ileum and an adhesiolysis was performed. It was also noted that the stomach and proximal duodenum were distended and there was an inability to decompress small bowel content proximally into the stomach or duodenum. A gastroenterostomy was performed using a stapled anastomosis. A loop of proximal jejunum from 45 cm from the ligament of Treitz was stapled to the greater curvature of the stomach.

On the first postoperative day, the nasogastric tube was removed, and the patient was started on a clear fluid diet.

On the third postoperative day, the patient was tolerating a full ward diet, and passing stool and she was discharged home.

She was followed up at the surgery outpatient clinic 2 weeks later and remained asymptomatic. A follow-up gastrografin meal and follow-through was done one month later which demonstrated adequate passage of contrast from the stomach into the small intestine.

She reported an improvement in her appetite with appropriate weight gain at a 3 month and 6 month follow-up visit.

Discussion

SMA syndrome, also known as Wilkie's syndrome, is a rare but serious condition that results in obstruction of the upper digestive tract. It occurs when the 3rd part of the duodenum becomes compressed in the horizontal position between the abdominal aorta and the SMA, which can cause duodenal obstruction.[1]

The incidence of SMA syndrome is estimated to be 0.013–0.3% with only 500 cases reported worldwide to date.[2,3]

The true prevalence may be underreported due to misdiagnosis or lack of awareness.[2]

Most cases of SMA syndrome occur in individuals aged 10 to 39, mean age of 23, with a higher prevalence in females, exhibiting a male-to-female ratio of 2:3.[4]

SMA syndrome may have a congenital origin, commonly caused by an anatomical shortening of the ligament of Treitz, leading to the duodenum being suspended in an abnormal position.[5]

Additional anatomical factors contributing to the condition include a high duodenal insertion at the ligament of Treitz, a low-originating SMA, and duodenal compression caused by peritoneal adhesions.[6]

However, acquired SMA syndrome is more prevalent and typically results from a substantial reduction in perivascular fat in the AOM region, often triggered by medical, psychological, or surgical conditions.[7] The fat pad cushion functions to hold the SMA off the spine and protect it from duodenal compression.[6] In this case report, the diagnosis of SMA syndrome was incidental and may have remained undetected if not for the adhesive bowel obstruction. It is presumed that the symptoms of SMA syndrome were unmasked or exacerbated by the loss of the mesenteric fat pad following the TAH and adhesive bowel obstruction soon thereafter.

Diagnosing SMA syndrome is often difficult and delayed due to the diverse range of symptoms it presents.[6]

In acute cases, the primary symptoms are those of duodenal obstruction, i.e. bile-stained vomitus and upper abdominal pain. They can present with significant electrolyte disturbances that mimic a gastric outlet obstruction.[8,9]

Chronic presentations, however, are characterised by persistent, vague abdominal discomfort or recurring episodes of abdominal pain accompanied by vomiting and loss of weight. Individuals may also experience symptoms like oesophageal reflux, early satiety due to delayed gastric emptying, and gastric distension, all of which arise from prolonged transit time in the digestive tract.[8]

Clinical suspicion plays a key role, and the diagnosis is typically based on clinical signs supported by imaging studies and laboratory findings. Initial assessment with a plain abdominal X-ray, followed by barium radiography, can reveal dilation of the first and second parts of the duodenum, with or without gastric distension.[6]

Additionally, it may show anti-peristaltic contrast flow above the obstruction and a delay in gastro-duodenojejunal transit time of 4 to 6 hours. Symptoms may improve when the patient is placed in different positions, such as prone, knee-chest, or left lateral.[6]

Contrast-enhanced CT or magnetic resonance angiography can provide detailed images, highlighting the vascular compression of the duodenum and allowing measurement of the AOM angle and distance.[6]

Under normal physiological conditions, and due to the upright position, the aorta-SMA angle typically ranges from 45–60°. The presence of perivascular fat tissue helps maintain an average AOM distance between 10–20 mm.[6]

However, in SMA syndrome, the angle decreases sharply to between 22 and 28 degrees, and the AOM distance narrows to 2–8 mm. These changes, when observed in the appropriate clinical context, strongly suggest SMA syndrome, leading to extraluminal compression of the duodenum.[7]

Endoscopic evaluation may also reveal external compression of the third part of the duodenum, raising suspicion of SMA syndrome.[8]

Initial management focuses on conservative approaches, including nasogastric decompression, intravenous fluid resuscitation, electrolyte correction, and intensive nutritional support. When feasible, nutrition may be provided via parenteral routes or post-pyloric feeding via naso-jejunal feeds, transitioning to an oral diet as tolerated.[6,10]

Patients with a shorter symptom duration, moderate presentation, and partial duodenal obstruction may respond well to medical therapy.[6]

Positioning strategies, such as the left lateral decubitus, knee-chest, or prone positions, can help reduce mesenteric pressure on the duodenum, offering temporary symptom relief. However, no definitive timeframe has been established for the duration of conservative management.[6,10] Previous studies have demonstrated that conservative management of SMAS can be effective, though patient outcomes are often inconsistent. In one study focused on patients who developed SMAS after colorectal surgery, 62.9% responded well to non-operative management, with most improving within 2 to 4 weeks (78.5%).[9]

Laparoscopic duodenojejunostomy has become the preferred surgical approach, replacing open bypass surgery since 1998, with high success rates (80–100%), minimal complications and quicker recovery time.[6,11] While some authors advocate strongly for laparoscopic duodenojejunostomy as the standard of care,[6,11] our case warranted a more conservative surgical approach. The diagnosis of SMA syndrome was incidental, and the patient's nutritional status had been significantly compromised following recent abdominal surgery and adhesive bowel obstruction. These factors together with surgeon experience influenced the decision to pursue a simpler, individualised surgical strategy.

Conflict of interest

The authors declare no conflict of interest.

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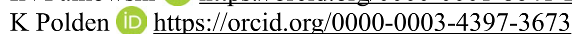
Ethical approval

Ethical approval was obtained from the University of Cape Town Faculty of Health Sciences Human Research Ethics Committee (Ref: 229/2025).

ORCID

IN Palkowski https://orcid.org/0000-0001-8641-2265


K Polden https://orcid.org/0000-0003-4397-3673

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