Author’s response to reviews

Title: Prolonged Gastroparesis after corrective surgery for Wilkie's Syndrome: A Case Report.

Authors:

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Author’s response to reviews: see over
**Reviewer:** The authors do not analyze a potential cause of duodenal obstruction. Usually there are a debilitating disease, surgery, trauma, severe weight loss e.g. as precipitating factors. The reported patient had Raynaud's syndrome and arthralgia but we do not know for how long and if these conditions did affect or trigger the SMA syndrome.

**Author:**

Her past medical history was remarkable for symptoms suggestive of Raynaud’s syndrome, multi-joint arthralgia and an episode of anorexia 4 years ago. She started experiencing symptoms of Raynaud’s syndrome nearly 5 years ago when she changed her occupation and started working in food catering industry. There was no deterioration of symptoms of Raynaud’s syndrome associated with weight loss. There was no history of recent trauma, surgery, prolonged immobilisation and neurological illness. Her weight loss was gradual. Other than gradual weight loss there was no real precipitating factor for this disease.

**Reviewer:**

Important and precise information are missing: how long did the patient have symptoms of duodenal obstruction, how much weight did she lose?

**Author:**

This lady was investigated for intestinal Dysmotility with 8 months history of symptoms of gradual weight loss, post-prandial epigastric bloating, sense of repletion and vomiting. Since her teenage she maintained a steady weight of 48 kg but now lost 6 Kg weight over a period of 8 months.

**Reviewer:**

Usually patients with SMAS are treated conservatively with nasogastric Decompression and nutritional support (enteral or parenteral) to restore Retroperitoneal fat (which is correctly stated in the discussion section). However, The reported patient did only undergo a trial of prokinetics and PPIs. Can the authors comment, why there was no nutritional support? How long did the authors try a conservative approach before surgery?

**Author:**

A seven days trial of conservative management with nasogastric tube, enteral feeding supplements and prokinetics failed. Patient’s symptoms rather worsened, she experienced more frequent vomiting and lost further 1 Kg weight. In the view of continued weight loss and worsening of symptoms, the conservative management was abandoned and patient chose to have Strong’s Procedure + open surgery.
Reviewer:
- The authors performed a duodenojejunostomy but did not mention Strong's Procedure (duodenal mobilization) which has revealed comparable results in the past. Did the authors consider this approach?

Author:
An attempted laparoscopic duodeno-jejunostomy and mobilisation of duodenum (Strong’s procedure) was abandoned as the massively distended stomach did not allow satisfactory assessment of the third part of duodenum.

Reviewer:
Again, there is no precise information about the time after that symptoms recurred. "Few months after surgery" is too vague. It is well-known that symptoms of SMAS may persist for a prolonged time after surgical correction because of duodenal atony, especially if there was massive dilatation of the proximal duodenum and stomach before surgery (reviewed in Welsch et al. Dig Surg 2007). How long was the follow-up after surgery and what was the further management? The case report ends somewhat open without a clear take-home message. I would have learned something from the report, if the authors would have reported that the symptoms relieved and gastric distention improved e.g. 4 months after surgery. Did the authors search for other causes of duodenal obstruction, e.g. megaduodenum caused by intestinal myopathy (biopsy) if the patient had still a distended stomach after surgery?

Author:
Endoscopy confirmed enlarged redundant stomach with patent duodeno-jejunostomy and poor gastric emptying. Her symptoms improved with supplement feeding for three days and correction of her hypokalaemia. Her endoscopic biopsies of duodenum and stomach failed to show any alternative diagnosis ie intestinal myopathy. She was also reviewed in our hospital and a tertiary hospital 14 months after her initial operation. She still had few episodes of vomiting especially after eating large meal and her weight remained unchanged. Again endoscopic and radiological investigation confirmed the diagnosis of gastric paresis related to Wilkie’s Syndrome. The follow up was for 14 months after surgery. Symptoms initially relieved for 5 months and then returned. She had on and off symptoms of bloating and vomiting especially after large meals. She had repeated CT and endoscopic examination. Biopsies confirmed the absence of any myopathic causes. She was referred to tertiary hospital for second opinion about diagnosis and gastric pacemaker implant assessment. Again the diagnosis was considered to be gastric paresis related to Wilkie’s syndrome. There is no real evidence for role of gastric pacemaker in such cases so it was not tried in her case.