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CORRESPONDENCE

Wilkie or Ogilvie?

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The clinicians who care for patients with spinal cord injury (SCI), are aware of the alarming phenomenon of abdominal distention with or without signs of paralytic ileus. In the ISCoS textbook, we can find the following: in acute SCI patients, gastric dilatation and intestinal paralysis can be observed, causing absence of bowel movements, nausea, vomiting, anorexia, respiratory difficulties, and inadequate venous return [1]. Hamilton Bailey's classic descriptions of acute and subacute intestinal obstruction [2], summarizes the full spectrum of the problem. The characteristics of acute obstruction of the small intestines are: colic pain, pain in the back (to the lumbar region), caused by tension on the mesentery occasioned by an extreme increase in intraluminar pressure as described by the Australian Dr. Victor John Kinsella (1900–1983) [3], vomiting, dehydration, constipation, (except in mesenteric vascular occlusion, Richter's hernia, and pelvic abscess), brown furred, and dry tongue. The superior mesenteric artery (SMA) syndrome (body cast syndrome) is a rare phenomenon in which the third portion of the duodenum is intermittently compressed by the overlying SMA. Sir David Percival Wilkie (1882–1938) from Edinburgh had described this syndrome. It is characterized by abdominal pain, vomiting, blood in stool and/or vomitus, and abdominal distention. Radiologic appearance is unique. "The key in identifying SMA syndrome is obtaining a small bowel follow-through study to fluoroscopically visualize the bowel behavior in real-time, particularly demonstrating improved contrast passage in the prone position compared to the supine state. This was the key test, helping clinch the diagnosis. Fluoroscopic imaging of the abdomen following barium intake demonstrates contrast pooling in the first and second parts of the duodenum with an abrupt cut-off at its third part, coinciding with the superior mesenteric artery impression. Abdominal radiograph after one-hour demonstrate persistent contrast pooling in the proximal duodenum with an abrupt cut-off at its third part (arrows), coinciding with the superior mesenteric artery impression" [4]. If not treated, bowel infarction follows [5, 6].

The factors which may cause this syndrome are:

1. rapid weight loss,
2. a flaccid abdominal wall which becomes spastic,
3. prolonged supine position, especially with the head elevated,
4. hyperlordotic deformity of the lumbar spine.

Sensory impairment, emotional lability, muscle atrophy and accompanying complications such as paralytic ileus or "stress ulcer" "[first described by the British surgeon Thomas Blizard Curling, 1811–1888]" may cause a delay in the diagnosis. The syndrome is sometimes difficult to diagnose: slow and vague onset leading to a dynamic intestinal obstruction that might lead to gangrenous bowel and perforation. SMA stenosis may cause "intestinal angina" characterized by abdominal colicky pain, appearing after a meal, diarrhea, positive occult blood, and weight loss.

The mesenteric vascular occlusion he described is a condition that simulates acute intestinal obstruction: it starts suddenly, with hypotensive/vascular shock, pain, vomiting, circumscribed abdominal rigidity, rebound tenderness, ill-defined lump is felt, hematemesis or melena. The Wilkie syndrome was reported in many other occasions. The spectrum of treatments is varied: correction of the medical condition, and if it fails, retrocolic duodenojejunostomy is unavoidable. Concerning SMA syndrome among SCI (mainly tetraplegics), rapid weight loss, refractory autonomic dysreflexia, and severe spasticity are common, as well as left renal vein dilation [7]. We have reported on a traumatic patient with motor incomplete paraplegia, who developed acute intermittent arterio-mesenteric occlusion of the duodenum after the use of Harrington's spinal

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instrumentation. This rare condition was treated conservatively. It is presumed that some degree of hyperlordosis of the lumbar spine by the rods was responsible for this phenomenon [8]. Later on, we described a complete and immediate disappearance of the syndrome in a paraplegic, only by pulling out the pillow which was inserted below the lumbar spine [9]. There are a few more reports in the literature [10–12].

Sir Heneage Ogilvie, Surgeon to Guy's Hospital (1887–1971), gained his clinical experience in both World Wars [13]. He described the syndrome in 1948, which bears his name: Acute colonic pseudo-obstruction: abdominal distention and pseudo-distention due to an autonomic disturbance [14]. Old age, various comorbidities, major surgical procedure, and presence of an acute illness are the common factors associated with the development of Ogilvie's syndrome. Recurrence has been reported in 17–38% of cases after initial success with neostigmine When patients receive neurotoxic drugs (like in chemotherapy) or narcotics, they might develop this syndrome. It is also used as a synonym for all forms of large bowel obstruction without mechanical cause (pseudo-obstruction) [15, 16]. This condition may lead to cecal perforation but sometimes spontaneous recovery was reported. It may accompany situations such as herpes zoster infection in lymphomas [17], old age, bedridden patients, obese patients, in chronic constipation, in patients with blunt trauma, burns, hip replacement, after Caesarian section, choriocarcinoma etc. The colonic dilatation, which is accompanied by intermittent flatus change and lack of bowel sounds, is relieved by colonoscopic decompression, water soluble contrast administered orally or rectally, naso-gastric aspiration, enema, neostigmine, or operation. Ogilvie's syndrome was described following spinal deformity correction and tethered cord release in an adolescent who presented with acute abdominal distension, nausea, and vomiting on postoperative first day [18]. This case is the first reported instance of neostigmine use for Ogilvie's syndrome treatment following a pediatric neurosurgical operation.

I found only one article which associates SCI with the Ogilvie syndrome [19]: "This article discusses the case of a 64-year-old bedridden, paraplegic, male nursing home resident who presented to the Emergency Department with a chronic history of abdominal distention that acutely worsened on the day of his arrival. A diagnosis of acute colonic pseudo-obstruction was made and 2 mg of intravenous neostigmine (an acetylcholinesterase inhibitor) was administered, with resolution of the patient's condition allowing for subsequent Emergency Department discharge".

Clinically speaking, SCIP may develop paralytic ileus, Wilkie's or Ogilvie's syndromes. I challenge the readers, general surgeons, and the experienced "spinalists" to try and to solve the clinical and perhaps the nosological problem: who can clearly differentiate between these situations in the spinally injured patient? Or rather is there an overlapping aspect of these situations? The treatment essentially is not the same for these situations. It is possible that these conditions differ from the almost general distention and bowel dysfunction seen after SCI and during spinal shock.

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