Diagnosis and treatment of Ogilvie's syndrome after lumbar spinal surgery

Report of three cases

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Three patients who developed Ogilvie's syndrome following lumbar spinal surgery are described. Ogilvie's syndrome, also known as pseudo-obstruction of the colon, is characterized by massive cecal distention without mechanical obstruction. If this condition is not recognized and not promptly treated, it may be complicated by cecal perforation, a life-threatening hazard. The etiology, diagnosis, management, and potential relationship between lumbar spinal surgery and Ogilvie's syndrome are discussed.

KEY WORDS  •  lumbar spine  •  colon  •  cecum  •  colonoscopy  •  Ogilvie's syndrome

Ogilvie's syndrome, also known as pseudo-obstruction of the colon, is a potentially life-threatening condition usually seen in elderly or debilitated patients. To the unsuspecting, this condition may appear to resemble simple adynamic ileus, as it did following lumbar spinal surgery in three patients described in this report. Early recognition and prompt, appropriate treatment of Ogilvie's syndrome may prevent significant morbidity following lumbar spinal surgery.

Case Reports

Case 1

This 62-year-old man presented with a 2-year complaint of right leg weakness and bilateral leg numbness, the latter exaggerated with walking. He had undergone a posttraumatic L-4 to sacral fusion 40 years previously and had experienced no intervening sequelae. He had a history of hypertension treated with nifedipine. There was no prior history of gastrointestinal illness or surgery. On examination, he was stockily built and had significant weakness in his right leg associated with reduced tendon reflexes in both legs. Magnetic resonance (MR) images revealed severe lumbar spinal stenosis extending from L1–2 to L4–5, narrowest at L2–3 with significant disc bulging at that level. Myelography was performed the day before surgery. An uncomplicated L1–5 decompressive laminectomy and L2–3 discectomy was carried out with the patient in the prone, semiflexed position.

Postoperatively, the patient noted relief of leg symptoms and had improvement of strength in his right leg. On the second postoperative day, he was uncomfortable due to abdominal distention and crampy abdominal pain. He did not complain of nausea and did not vomit. High-pitched bowel sounds were noted. He was receiving morphine sulfate via a patient-controlled analgesia device for treatment of wound pain. On the same day, general surgical consultation was requested and abdominal x-ray films were ordered which showed distended small and proximal large bowel (Fig. 1). As the cecum measured 14 cm, the patient underwent laparotomy that day. At surgery, a massively dilated cecum was found and an appendectomy and tube cecostomy were undertaken. The patient continued to have abdominal distention and ileus for the 1st week after laparotomy. He had a partial dehiscence of his laparotomy incision with wound infection. His hospital stay was 23 days; at discharge, the patient was symptom-free in his legs, ambulating and urinating without difficulty, and had good recovery of strength in his right leg. The tube cecostomy was removed 1 week prior to discharge and normal bowel motility had been restored, but laparotomy wound packings were still required. At his 6-month follow-up examination, the patient had minimal right

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FIG. 1. Anteroposterior abdominal x-ray film of Case 1 at Day 4 following lumbar surgery. Arrows delineate the distended cecum and proximal colon. Calcifications are incidental phleboliths.

leg weakness and no other residual symptoms related to his lumbar condition. The laparotomy incision was fully healed but he was left with a ventral hernia. He had no residual gastrointestinal sequelae.

Case 2

This 58-year-old man complained of progressively uncomfortable numbness in both legs of 3 months' duration which was intensified by standing and walking. Aside from hypertension, for which he was treated with labetalol and clidinium, the patient was in good health. There was no history of prior gastrointestinal illness or surgery. The only abnormality found on examination was absence of ankle reflexes. Radiographic studies included computerized tomography scans and MR images of the lumbosacral spine which showed spinal stenosis from L3–4 and L5–S1. Plain x-ray films showed an unstable degenerative spondylolisthesis at L4–5. Conservative treatment, including three epidural steroid injections, failed. Eight months after the first evaluation, surgery was performed. The procedure undertaken was an L-3 to sacral laminectomy and L4–5 fusion using metal plates fixed with pedicle screws and transverselateral iliac bone graft, performed with the patient in the prone/kneeling (knee/chest) position.

Postoperatively, the patient noted relief of his leg symptoms and neurological examination of his legs revealed no change. On the second postoperative day, he complained of a sense of gastric distention without pain and he was hungry. His bowel sounds were diminished and oral liquids were allowed. During this time, while awaiting the fitting of a lumbosacral brace, he remained at bed rest and received a narcotic analgesia (meperidine delivered by a patient-controlled analgesia device). On the fourth postoperative day, he complained of increased abdominal distention and of constipation. Oral intake was stopped and a cathartic was given. Later the same day, he complained of nausea and mild abdominal pain, and his temperature rose to 101.7°F. General surgical consultation was requested.

The general surgeon observed markedly expanded abdominal distention with diffuse mild tenderness and tympanic bowel sounds. A nasogastric tube was placed, and chest and abdominal x-ray films were obtained which disclosed distended small bowel and proximal colon. Free air was also noted on the right hemidiaphragm, and intravenous antibiotics were begun. Laparotomy revealed dilated small and proximal large bowel. A small cecal perforation was closed, an appendectomy was performed, and a tube cecostomy was inserted. The patient's gastric distention slowly resolved.

By the 8th day after laparotomy, the nasogastric tube was removed, the cecostomy tube was closed, signs of abdominal motility had resumed, and oral intake was begun. The patient was discharged from the hospital 15 days after admission, free of symptoms in his legs, able to walk, and tolerating a full diet. The day prior to discharge, a subcutaneous cecostomy wound abscess was drained and packed. One week after discharge, antibiotics were discontinued, the cecostomy tube was removed, and the patient was finally fitted for his lumbosacral brace. Six months later, he required readmission for repair of a ventral hernia. At his 1-year follow-up examination, the patient remained symptom-free in his legs and had no residual gastrointestinal sequelae.

Case 3

This 42-year-old man suffered a work-related injury and developed low-back and right leg pain and weakness of dorsiflexion of the right foot 8 months prior to admission for surgery. An MR image suggested a ruptured L4–5 disc on the right side. His history disclosed no illnesses, but he had undergone a prior appendectomy and he smoked one pack of cigarettes per day. When his symptoms remained unrelied by nonsurgical measures, the patient underwent a right-sided hemilaminectomy at L4–5 and removal of a ruptured disc. The operation took 1½ hours and was performed with the patient under general anesthesia in the prone/kneeling (knee/chest) position. Postoperative pain was controlled with morphine sulfate delivered by a patient-controlled analgesia device, and muscle spasms were treated with alprazolam, 0.25 mg four times daily, as needed.

The patient resumed a regular diet on the day of surgery. The following day, he complained of slight nausea. The next day, he complained of "gas pains," was observed to have a distended abdomen, and an abdominal x-ray film showed "marked proximal colonic dilatation." The general surgical consultant believed the patient had postoperative ileus. Oral intake and analgesic and muscle relaxant treatments were stopped. Intravenous hydration and metoclopramide
hydropchloride, and soap suds enemas were instituted.

The following day, the abdominal examination and x-ray findings were unchanged; a gastroenterologist performed a colonoscopy and observed a dilated cecum with an erythematous, friable mucosa with erosions and exudate. Abdominal x-ray films later that day revealed a dramatic reduction of colonic distention. The patient required a second colonoscopy for recurrent proximal colonic distention on the 6th postoperative day. On the 7th postoperative day, he was discharged free of abdominal, back, and leg symptoms and tolerated a regular diet. He remained symptom-free 2 months after discharge.

Discussion

History and Etiology of Ogilvie's Syndrome

Ogilvie first described a condition characterized by a disorder of bowel motility in 1948. Ogilvie’s syndrome is clinically characterized by massive abdominal distention with a cecal diameter greater than 9 cm in the absence of colonic obstruction (AB Jetmore, personal communication, 1991). Although both original patients described by Ogilvie had abdominal cancer, this has proved to be an uncommon association in Ogilvie’s syndrome. Abdominal x-ray films usually demonstrate marked distention of the proximal colon with distal cutoff of colonic gas suggestive of distal colonic obstruction, thus the alternative name “pseudo-obstruction of the colon.”

The cause of Ogilvie’s syndrome has been variously attributed to the loss of proximal colonic sympathetic stimulation, air-fluid or “vapor” look as a result of fluid collections in dependent portions of the bowel, sacral parasympathetic denervation, prostaglandin abnormalities, and transient ischemia of the colon.

Despite the uncertainty about the pathophysiology of Ogilvie’s syndrome, it is known that many clinical conditions are associated with the development of this entity. The more commonly cited factors include old age, uremia, chronic alcoholism, the postpartum period, narcotic addiction, narcotic usage, diabetes mellitus, congestive heart failure, sepsis, trauma, irregular bowel habits, laxative abuse, and bed rest. Other drugs commonly associated with Ogilvie’s syndrome are phenothiazines, hemagglutination H2 blockers, and calcium channel blockers (AB Jetmore, personal communication, 1991). Regardless of the etiology, pseudo-obstruction of the colon is usually a hospital-acquired condition.

Ogilvie’s syndrome has been reported as a complication of spinal surgery, spinal trauma, spinal anesthesia, spinal metastasis, myelography, and epidural narcotic administration. Other neurological conditions, including postoperative craniotomies, Parkinson’s disease, organic brain syndrome, multiple sclerosis, stroke, meningitis, meningioma, and Guillain-Barré syndrome, have been associated with Ogilvie’s syndrome.

The most serious complication of Ogilvie’s syndrome is perforation of the cecum. Early recognition and treatment of pseudo-obstruction of the colon may prevent cecal perforation, which carries a reported mortality rate of 25% to 60%.

Adynamic Ileus Following Lumbar Surgery

Adynamic ileus is a well-recognized complication of lower back surgery and is characterized by abdominal distention. Nausea and vomiting rarely accompany adynamic ileus and respiratory distress and a sense of constipation may or may not be associated. Abdominal tenderness is rare. Bowel sounds are usually absent. This condition is common enough that some advocate that postlaminctomy patients receive no oral intake for 24 hours or until there is restitution of normal bowel function. Adynamic ileus following lumbar spinal surgery has been attributed to retroperitoneal hemorrhage. This more common form of ileus readily responds to simple measures, including restriction of oral intake, administration of bowel stimulants, enemas, and/or laxatives, and, in some instances, nasogastric suction.

Differential Diagnosis of Ogilvie’s Syndrome

Although there may be some similarities between the two entities, Ogilvie’s syndrome is differentiated from simple adynamic intestinal ileus by the striking amount of abdominal distention primarily due to right colonic dilatation, the presence of bowel sounds, the occurrence of nausea or vomiting, and a lack of response to those measures mentioned above for the treatment of adynamic ileus. The small bowel is frequently dilated in both conditions. Both conditions are part of a spectrum of intestinal motility disorders that can be associated with a wide variety of clinical situations.

The differential diagnosis of Ogilvie’s syndrome includes acute gastric dilatation, fecal impaction, volvulus, organic colon obstruction, and ischemic bowel. Acute gastric dilatation has a different radiographic appearance and responds rapidly to nasogastric suction. Actual colonic obstruction may be caused by cancer, fecal impaction, or volvulus and can usually be diagnosed radiographically (contrast studies) or by colonoscopy. Colonic dilatation due to intestinal ischemia is usually diagnosed by colonoscopy.

Diagnosis of Ogilvie’s Syndrome

As already indicated, symptoms of Ogilvie’s syndrome are variable and may include nausea (63%), vomiting (57%), constipation (51%), diarrhea (41%), and/or pain. Examination of virtually all patients with this condition demonstrates marked abdominal distention. Plain x-ray films of the abdomen are characterized by proximal colonic dilatation. This radiographic feature may be difficult to distinguish from cecal volvulus although contrast studies or colonoscopy will resolve the issue. Gas may be seen in the small bowel depending on ileoceleal valve patency. Normal cecal diameter is 3.5 to 8.5 cm; when cecal diameter is greater than 9 cm, early intervention is warranted.
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Treatment of Ogilvie’s Syndrome

The cecum is the thinnest walled reservoir in the colon and has the greatest diameter. According to Laplace’s law, the pressure required to stretch the walls of a hollow viscus decreases in inverse proportion to the diameter. Thus, the cecum is the portion of the colon most vulnerable to rupture. Because of the significant mortality rate associated with cecal rupture, early intervention is prudent. For this same reason, some have chosen to call Ogilvie’s syndrome “malignant ileus.” Nasogastric suction, rectal tubes, restriction of oral intake, and cessation of narcotic medications may be useful measures before the cecum becomes overdistended. More aggressive management is suggested when the cecum is distended, but the threshold for concern varies among different authors, ranging from radiographic measurements of cecal diameters of 12 to 16.5 cm. Colonic decompression in Ogilvie’s syndrome was first described in 1977 by Kukora and Dent. Colonoscopy is now accepted as the treatment of choice for Ogilvie’s syndrome. To be effective, the colonoscope must reach the hepatic flexure; if ischemia is encountered, colonoscopy should be discontinued. Laparotomy and tube cecostomy are reserved for patients in whom intestinal rupture has already occurred. Tube cecostomy is superior to right colon resection because the procedure enjoys a lower mortality rate and does not require a second operation to re-establish intestinal continuity.

Outcome of Treatment of Ogilvie’s Syndrome

The prognosis for recovery from this condition is excellent if appropriate treatment is instituted before the cecum perforates. Colonic decompression is successful in 80% to 85% of cases, although multiple decompressions are required in about 25% of patients. The mortality rate for patients with Ogilvie’s syndrome treated with colonoscopy and conservative management is 13%, whereas in patients treated with cecostomy it approaches 30%. In cases where treatment is prompt and appropriate, patients with Ogilvie’s syndrome are frequently more at risk from their underlying predisposing condition. Five factors are reported to affect the risk of mortality: treatment (colonoscopy vs. cecostomy), patient age, cecal diameter, delay in decompression, and ischemia or perforation of the colon.

Etiology of Ogilvie’s Syndrome Following Lumbar Surgery

Given the rarity of Ogilvie’s syndrome following lumbar surgery, the etiology in these three cases is speculative at best. Retroperitoneal bleeding following lumbar surgery is thought to be a cause of postlamincotomy adynamic ileus. Perhaps larger, lengthier, and more complex spinal procedures are likely to cause more extensive retroperitoneal hematoma. In neither of our two patients who underwent laparotomy did the general surgeons observe retroperitoneal blood. If one views Ogilvie’s syndrome as the malignant end of a spectrum of conditions involving diminished intestinal motility, one might expect to see the pseudo-obstructive syndrome with increasing frequency as lumbar surgical procedures become more complex. However, in contrast to Cases 1 and 2, surgery for Case 3 was simple and brief. A prolonged operation with the patient in the prone position with consequent abdominal pressure may also be an underlying factor. In Cases 2 and 3, prone/kneeling (knee/chest) position was used, thus eliminating all direct anterior abdominal wall pressure. Each of these patients received narcotic analgesic medication and remained on bed rest in the postoperative period until they developed the pseudo-obstructive syndrome. Bed rest and narcotic medication are both thought to predispose to Ogilvie’s syndrome. Most likely, some combination of these and other factors (prior abdominal surgery, other medications used before and after surgery, and preoperative myelography) underlay the development of this condition in these three patients.

Management of Patients With Ogilvie’s Syndrome Following Lumbar Surgery

As with intestinal immotility following abdominal trauma and surgery, Ogilvie’s syndrome following lumbar surgery may be prevented if careful attention is paid to the early management of intestinal immotility by withholding oral intake and the use of gastric decompression. These considerations should be particularly emphasized in cases in which lumbar surgery is lengthy or complex. It is also possible that some aspect of the positioning of patients for lumbar surgery, including prolonged abdominal compression, may predispose some patients to develop Ogilvie’s syndrome. Additional factors, such as bed rest and narcotic analgesic medications used in these patients, have been thought to predispose patients and should alert the surgeon to the potential for the development of pseudo-obstruction of the colon. Should clinical manifestations of this condition appear, prompt recognition and colonoscopic decompression may prevent the potentially more serious consequences such as those described in Cases 1 and 2.

Acknowledgments

The authors thank Ronald F. Young, M.D., Division of Neurosurgery, University of California, Irvine, California, for editorial advice and help with manuscript preparation, and Mrs. Jane Knowles, librarian, St. Vincent Hospital, Santa Fe, New Mexico, for editorial assistance.

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Manuscript received January 30, 1991.
Accepted in final form November 18, 1991.
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