Case Report

Ogilvie's syndrome following posterior spinal arthrodesis for scoliosis

Athanasios I Tsirikos, Alok Sud[1]

Abstract

We report Ogilvie's syndrome following posterior spinal arthrodesis on a patient with thoracic and lumbar scoliosis associated with intraspinal anomalies. Postoperative paralytic ileus can commonly complicate scoliosis surgery. Ogilvie's syndrome as a cause of abdominal distension and pain has not been reported following spinal deformity correction and can mimic post-surgical ileus. 12 year old female patient with double thoracic and lumbar scoliosis associated with Arnold-Chiari 1 malformation and syringomyelia. The patient underwent posterior spinal fusion from T_4 to L_3 with segmental pedicle screw instrumentation and autogenous iliac crest grafting. She developed abdominal distension and pain postoperatively and this deteriorated despite conservative management. Repeat ultrasounds and abdominal computer tomography scans ruled out mechanical obstruction. The clinical presentation and blood parameters excluded toxic megacolon and cecal volvulus. As the symptoms persisted, a laparotomy was performed on postoperative day 16, which demonstrated ragged tears of the colon and cecum. A right hemi-colectomy followed by ileocecal anastomosis was required. The pathological examination of surgical specimens excluded inflammatory bowel disease and vascular abnormalities. The patient made a good recovery following bowel surgery and at latest followup 3.2 years later she had no abdominal complaints and an excellent scoliosis correction. Ogilvie's syndrome should be included in the differential diagnosis of postoperative ileus in patients developing prolonged unexplained abdominal distension and pain after scoliosis correction. Early diagnosis and instigation of conservative management can prevent major morbidity and mortality due to bowel ischemia and perforation.

Key words: Acute colonic pseudo-obstruction, ileus, Ogilvie's syndrome, posterior spinal fusion, scoliosis

Introduction

Scoliosis surgery can often be complicated by postoperative paralytic ileus and superior mesenteric artery (SMA) syndrome.[1-4] Both, conditions have usually a benign course and resolution of the symptoms should be expected with early instigation of conservative management. Their clinical appearance can, however, mask that of an acute colonic pseudo-obstruction (ACPO) which if left untreated can result in bowel ischemia and perforation with an estimated mortality rate of 40%.[5]

ACPO was first described by the British surgeon Sir William Heneage Ogilvie in 1948 when he reported 2 patients with chronic colonic dilatation associated with malignant infiltration of the celiac plexus.[6] In his original report, Ogilvie attributed the condition to sympathetic deprivation.[6] It is now recognized that a probable explanation of the syndrome is imbalance in the regulation of the colonic motor activity by the autonomic system, which leads to excessive sympathetic stimulation or parasympathetic suppression.

Ogilvie's syndrome presents with symptoms, signs and radiographic appearance of acute large bowel obstruction of nonmechanical aetiology. The clinical features include abdominal distension and pain (80%), as well as nausea with or without associated vomiting (60%).[5,7] Plain abdominal X-rays demonstrate varying degrees of colonic dilatation with the right colon and cecum being most markedly distended. The clinical examination confirms a dilated, tympanitic abdomen with preserved but hypoactive bowel sounds. The differential diagnosis should include mechanical obstruction, faecal impaction, cecal or sigmoid volvulus, as well as toxic megacolon.

Consultant Orthopaedic and Spine Surgeon, Honorary Clinical Senior Lecturer, University of Edinburgh, [1]Commonwealth Travelling Spinal Fellow, Scottish National Spine Deformity Center, Royal Hospital for Sick Children, Sciennes Road, Edinburgh, EH9 1LF, UK

Address for correspondence: Mr. Athanasios I. Tsirikos, Consultant Orthopaedic and Spine Surgeon, Honorary Clinical Senior Lecturer-University of Edinburgh, Scottish National Spine Deformity Center, Sciennes Road, Edinburgh, EH9 1LF, UK.
E-mail: atsirikos@hotmail.com

Access this article online	
Quick Response Code:	Website: www.ijoonline.com
	DOI: 10.4103/0019-5413.114934

We present an adolescent female patient with a thoracic and lumbar scoliosis who underwent posterior spinal arthrodesis and developed Ogilvie's syndrome. We discuss the clinical course and treatment, as well as the final outcome at latest followup. To the best of our knowledge, this is the first reported case to develop ACPO after scoliosis correction.

Case Report

A 12 years and 8 months old female presented with a double structural thoracic and lumbar scoliosis associated with Arnold-Chiari 1 malformation and an extensive cervicothoracic syringomyelia. At age 11 years and 11 months she developed acute weakness affecting both hands and feet with associated pins and needles down the arms and legs, as well as altered sensation affecting the right upper limb below the elbow while her bladder and bowel function was normal. The patient underwent an urgent cranio-cervical decompression including a posterior fossa craniotomy and C5/C6 laminectomy with syringostomy and placement of a syringopleural shunt to drain the syrinx [Figure 1]. She recovered well following surgery and there were no residual neurological signs or symptoms at the time of presentation in our clinic.

On clinical examination at age 12 years and 8 months, she had a progressive thoracic and lumbar scoliosis extending from T5 to T10 measuring 60° and T10 to L3 measuring 50° respectively. Neurological examination was normal. The patient had normal function including sport activities. She was skeletally immature and pre-menarche with Risser grade 1. An MRI of the whole spine demonstrated adequate decompression of the cranio-cervical junction and a small residual cervical syrinx which was considered to require no further treatment at the time by our neurosurgical colleagues.

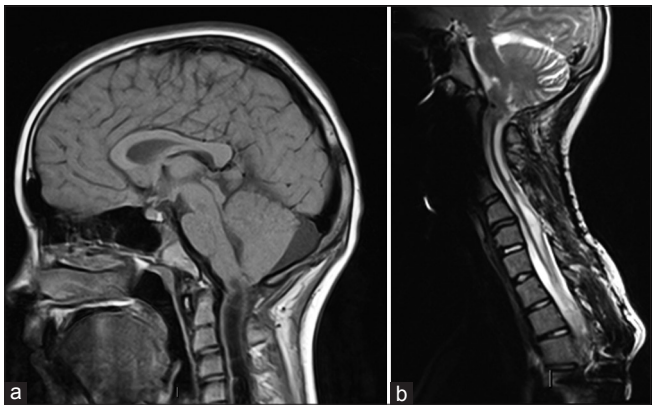

Figure 1: Preoperative MRI (a) at onset of neurological symptoms shows herniation of the cerebellar tonsils and a large syringomyelia which extended along the whole of the cervical and into the upper thoracic spine. Repeat MRI (b) after neurosurgical decompression and placement of a syringopleural shunt shows the syrinx to have markedly reduced and no pressure on the cerebellum at the foramen magnum

The patient underwent posterior spinal arthrodesis extending from T4 to L3 at age 12 years and 10 months with the use of segmental pedicle screw and rod instrumentation and autologous iliac crest bone graft [Figure 2]. Spinal cord monitoring was performed during the procedure recording motor and sensory (cortical and cervical) evoked potentials which remained stable throughout. Postoperative neurological examination was normal. The patient had 3 doses of intravenous (IV) cefuroxime; one at induction to anaesthesia, and 2 after surgery at 8-hourly intervals.

In the postoperative period, the patient developed prolonged abdominal distension without pain which was considered to be due to paralytic ileus as the result of the major spinal operation, as well as the effect of narcotic analgesic agents delivered during and after surgery. There was no nausea or vomiting and the bowel sounds were preserved but reduced. She had no electrolyte abnormalities and her blood tests were normal other than an expected small reduction in haemoglobin. The spinal wound healed uneventfully and the patient mobilized out of bed on postoperative day 2.

At the end of the first week following surgery the abdominal distension began to increase and she started having generalized abdominal pain with associated tenderness in the right iliac fossa. She had no vomiting and no bowel movements. Repeat blood tests showed marginal leukocytosis coinciding with the onset of abdominal pain while the remaining parameters including electrolytes, kidney and liver function tests were within normal limits. She was initially managed conservatively maintaining fluid and electrolyte balance and placing a nasogastric tube for aspiration of gastric contents. Repeat ultrasounds and a CT scan of the abdomen did not demonstrate evidence of mechanical obstruction, congenital anomaly (such as a fibrous band or a malrotated bowel), or cecal/sigmoid volvulus [Figure 3]. During the whole postoperative period our patient was under regular review by our general surgical colleagues.

Due to aggravation of the patient's abdominal distension and absence of clear signs of peritonitis pain a laparoscopy was decided on postoperative day 16 followed by an exploratory laparotomy. At that stage no definitive diagnosis was made. The laparotomy showed the cecum and ascending colon to be grossly inflamed with multiple ragged perforations, no dilatation and gross contamination of the abdomen. Multiple loops of small bowel were adherent to the anterior abdominal wall in the right iliac fossa. The remainder of the transverse, descending and sigmoid colon was found to be entirely normal. A limited right hemi-colectomy and ileocecal anastomosis was performed and the patient returned to the intensive care with nasogastric aspirations. She remained

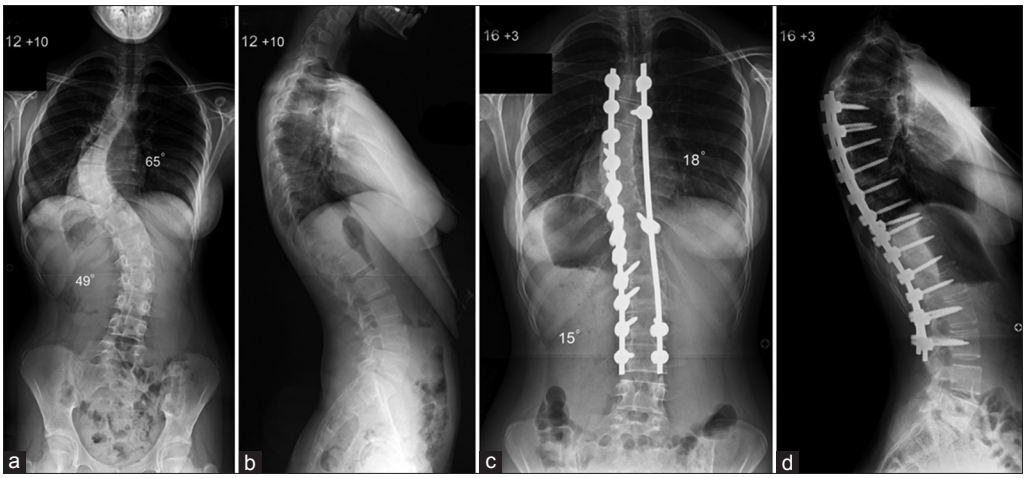

Figure 2: Preoperative posteroanterior (a) and lateral (b) radiographs of the spine show a left thoracic and a right lumbar scoliosis. Repeat radiographs at latest followup (c and d) show a balanced spine in the coronal and sagittal planes with good maintainance of scoliosis correction

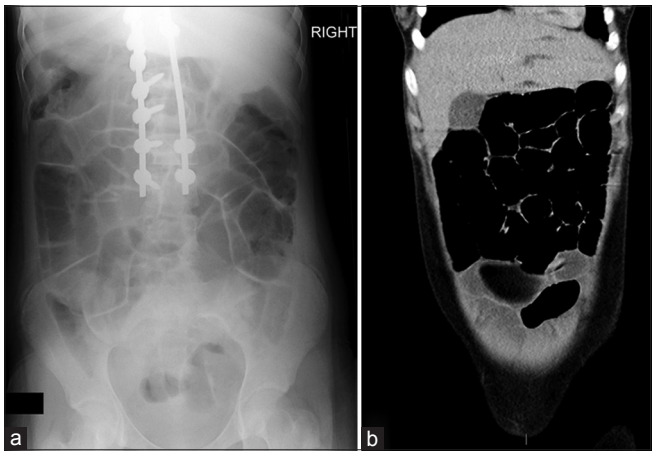

Figure 3: (a) Abdominal x-ray at 2 weeks after scoliosis surgery showing dilated bowel loops across the whole of the abdomen with a similar appearance on the CT scan (b) performed before decision was made for surgical exploration through a laparotomy

intubated for 7 days and was treated with meropenem and vancomycin for 2 weeks as the peritoneal cultures showed a mixed growth of Enterococcus and *E. coli*. This was changed to ciprofloxacin for an additional period of 3 weeks. She made gradual recovery with good bowel function, returned to feedings over a period of 2 weeks and was discharged on postoperative day 32.

The surgical specimen sent for histopathology included 3 cm of terminal ileum, 5.5 cm appendix and 12 cm of colon. A single large defect was identified at the cecum and multiple defects in the ascending colon above the ileocecal valve. The microscopic examination of the small bowel and appendix were normal. Sections from the colon and cecum showed ulcerations and perforation with organized fibrinous peritonitis. There were no features of Crohn's disease, ulcerative colitis or vascular abnormality. The surgical findings combined with the clinical picture and the pathology report confirmed the diagnosis of Ogilvie's syndrome.

At latest followup, 3.2 years after scoliosis surgery our patient was skeletally mature (Risser 5), had no abdominal complaints and was on a normal diet. Spinal radiographs showed maintenance of an excellent correction of her scoliosis and no signs of infection, instrumentation failure or nonunion. She had normal activities including sports and no complaints of her back.

Discussion

The most common abdominal complications occurring in patients who undergo scoliosis surgery include postoperative paralytic ileus and SMA syndrome.[1-4] These have a good prognosis and usually respond well to conservative management. Post-surgical ileus is expected to settle within 48-72 h after the procedure and as the patients become mobile out of bed with fewer requirements for opioid analgesia. SMA syndrome requires insertion of a nasogastric tube for gastric decompression, as well as a nasojejunal tube for bowel feedings until the temporary compression to the third part of the duodenum by the SMA resolves and the patient gradually returns to oral feeds within a few days.

Ogilvie's syndrome has not been previously recognized as a cause for abdominal distension and pain following spinal deformity surgery. Our patient had a prolonged prodromal period of gradually increasing painless abdominal distension, un-associated with vomiting, which was not accompanied by any mechanical obstruction to the small or large bowel, typically seen in ACPO. The most common predisposing conditions of this syndrome include nonoperative trauma, infections (pneumonia/sepsis) and cardiac diseases (heart

failure/myocardial infraction), usually in elderly patients who require long treatment in the high dependency unit.[5] Operative procedures often associated with Ogilvie's syndrome include caesarean section, abdominal and pelvic surgery, urologic/thoracic/neurosurgical and coronary by pass procedures.[8,9] It has also been reported following hip and knee surgery, as well as after lumbar or cervical spinal operations, such as discectomy.[10-14] Neurological conditions and the use of medication such as opioids, calcium channel blockers and anticholinergics can also precipitate ACPO.[7]

Even though the exact aetiology of this syndrome is not fully clarified, it is considered to occur due to autonomic disturbance of the colonic motor function.[15] The predisposing factors produce an autonomic imbalance leading to excessive parasympathetic inhibition which in turn results in colonic atony and features of pseudo-obstruction. Our patient had at least 3 risk factors for Ogilvie's syndrome, namely a surgical insult, a neurological factor (association of scoliosis with Arnold-Chiari 1 malformation and syringomyelia), as well as the use of morphine and other opioids during the postoperative period.

The diagnosis of Ogilvie's syndrome is one of exclusion. The pathological features of ACPO include gross dilatation of the colon and cecum without mechanical block which may lead to ischemia and perforation.[7] The differential diagnosis includes faecal impaction, colonic or rectal tumours, cecal or sigmoid volvulus and toxic megacolon.[5] CT scan is an excellent investigation to diagnose colonic or sigmoid tumors and differentiate between cecal and sigmoid volvulus.[16] Toxic megacolon should also be considered as part of the differential diagnosis but it is associated with rapid thinning of the colonic wall along with severe toxaemic features, which make it a potentially lethal condition. This is often associated with Crohn's disease, ulcerative and pseudo-membranous colitis but can occur as a complication of any infective or noninfective condition affecting the colon.[17,18]

Having excluded a mechanical obstruction and other conditions as part of the differential diagnosis, it is possible to treat a patient with Ogilvie's syndrome conservatively. The management includes keeping the patient nil by mouth, insertion of a nasogastric tube for suctioning to limit swallowed air contributing further to colonic distension, IV fluid administration, correction of underlying electrolyte imbalance, insertion of a rectal tube, as well as minimizing risk factors including discontinuation of medication which can adversely affect bowel motility.[7,19] A trial of IV neostigmine (a reversible acetylcholinesterase inhibitor which enhances bowel motor activity) can be given to decompress the colon if mechanical obstruction, ischemia and perforation have been excluded.[20] If the symptoms persist an endoscopy (colonoscopy) or surgical decompression is warranted, especially if the patient develops fever, abdominal pain/guarding and a high white cell count in addition to abdominal distension and constipation. A cecal diameter of 12 cm or more correlates with higher risk of perforation when the distension has been present for more than 6 days.[21] Surgical intervention is reserved for patients with signs of bowel ischemia or perforation and those who fail endoscopic and pharmacologic management. In the case of a colonic perforation, sub-total resection (hemi-colectomy) with ileostomy or an ileocecal anastomosis is the procedure of choice.[22]

In retrospect, even though our patient was under close monitoring during her whole postoperative course by our pediatric surgical colleagues the diagnosis of Ogilvie's syndrome was overlooked. The CT scan and repeat abdominal ultrasounds failed to establish the diagnosis. The marked delay in diagnosis resulted in bowel perforation and developing peritonitis which required an urgent laparotomy and major bowel reconstructive surgery which could have been life-threatening. The histopathological examination of the surgical specimen obtained during the hemi-colectomy ruled out volvulus or pathologies associated with toxic megacolon and indirectly confirmed the suspected diagnosis of Ogilvie's syndrome. The absence of previous reports in the literature of ACPO developing after scoliosis surgery was the main reason why the possibility of this diagnosis was not raised, despite the presence of strong clinical and radiological features suggestive of the condition in the background of co-existing risk factors.

Postoperative abdominal complications primarily related to paralytic ileus or superior mesenteric artery syndrome are not uncommon following scoliosis surgery and these usually settle with appropriate conservative management. We recommend that surgeons involved in the management of children and adolescents with spinal deformities should maintain a high index of suspicion of the occurrence of Ogilvie's syndrome as a possible cause of prolonged abdominal distension and pain mimicking paralytic ileus following major reconstructive surgery. The key to successful treatment of ACPO lies with a) an early diagnosis, b) assessment to exclude mechanical obstruction or other causes of pseudo-obstruction, c) evaluation of signs and symptoms indicating bowel ischemia or perforation, which carries a mortality rate of 40% (compared to 15% in patients with a viable bowel)[5] and requires urgent surgical management, and d) instigation of appropriate treatment at an early stage when this is more likely to be successful.

References

1. Crowther MA, Webb PJ, Eyre-brook IA. Superior mesenteric

artery syndrome following surgery for scoliosis. Spine (Phila Pa 1976) 2002;27:E528-33.
2. Tsirikos AI, Jeans LA. Superior mesenteric artery syndrome in children and adolescents with spine deformities undergoing corrective surgery. J Spinal Disord Tech 2005;18:263-71.
3. Shapiro G, Green DW, Fatica NS, Boachei-Adjei O. Medical complications in scoliosis surgery. Curr Opin Pediatr 2001;13:36-41.
4. Cho KJ, Suk SI, Park SR, Kim JH, Kim SS, Choi WK, *et al*. Complications in posterior fusion and instrumentation for degenerative lumbar scoliosis. Spine (Phila Pa 1976) 2007;32:2232-7.
5. Vanek VW, Al-Salti M. Acute pseudo-obstruction of the colon (Ogilvie's syndrome). An analysis of 400 cases. Dis Colon Rectum 1986;29:203-10.
6. Ogilvie WH. Large intestine colic due to sympathetic deprivation: A new clinical syndrome. BMJ 1948;2:671-3.
7. Saunders MD, Kimmey MB. Ogilvie's syndrome. In: McDonald, JWD, Burroughs, AK, Feagan, BG, eds. Evidence-based Gastroenterology and Hepatology. 2nd ed. Malden, USA. Blackwell Publishing 2004; pp 303-9.
8. Saha AK, Newman E, Giles M, Horgan K. Ogilvie's syndrome with cecal perforation after caesarean section: A case report. J Med Case Rep 2009;3:6177.
9. Guler A, Sahin MA, Atilgan K, Kurkluoglu M, Demirkilic U. A rare complication after coronary artery bypass surgery: Ogilvie's syndrome. Cardiovasc J Afr 2011;22:335-7.
10. Feldman RA, Karl RC. Diagnosis and treatment of Ogilvie's syndrome after lumbar spinal surgery. Report of three cases. J Neurosurg 1992;76:1012-6.
11. Caner H, Bavbek M, Albayrak A, Altinors TC. Ogilvie's syndrome as a rare complication of lumbar disc surgery. Can J Neurol Sci 2000;27:77-8.
12. Cakir E, Baykal S, Usul H, Kuzeyli K, Cinel A. Ogilvie's syndrome after cervical discectomy. Clin Neurol Neurosurg 2001;103:232-3.
13. Nelson JD, Urban JA, Salsbury TL, Lowry JK, Garvin KL. Acute colonic pseudo-obstruction (Ogilvie's syndrome) after arthroplasty in the lower extremity. J Bone Joint Surg Am 2006;88:604-10.
14. Reverdy D, Gebhart M, Kothonidis K, Gallez J, DeBecker D, Liberale G. Pseudo-colonic obstruction after lumbar spine surgery: A case report. Acta Orthop Belg 2006;72:769-71.
15. Dorudi S, Berry AR, Kettlewell MG. Acute colonic pseudo-obstruction. Br J Surg 1992;79:99-103.
16. Macari M, Speiler B, Babb J, Pachter HL. Can the location of CT whirl sign assist in differentiating sigmoid from cecal volvulus? Clin Radiol 2011;66:112-7.
17. Strong SA. Management of acute colitis and toxic mega colon. Clin Colon Rectal Surg 2010;23:274-84.
18. Sheikh RA, Yasmeen S, Pauly MP, Trudeau WL. Pseudomembranous colitis without diarrhoea presenting clinically as acute intestinal obstruction. J Gastroenterol 2001;36:629-32.
19. Sloyer AF, Panella VS, Demas BE, Shike M, Lightdale CJ, Winawer SJ, *et al*. Ogilvie's syndrome. Successful management without colonoscopy. Dig Dis Sci 1988;33:1391-6.
20. Ponec RJ, Saunders MD, Kimmey MB. Neostigmine for treatment of acute colonic pseudo obstruction. N Engl J Med 1999;341:137-41.
21. Johnson CD, Rice RP. The radiographic evaluation of gross cecal distension. AJR Am J Roentgenol 1985;145:1211-7.
22. Baker DA, Morin ME, Tan A, Sue HK. Colonic ileus: Indication for prompt decompression. JAMA 1979;241:2633-4.

How to cite this article: Tsirikos AI, Sud A. Ogilvie's syndrome following posterior spinal arthrodesis for scoliosis. Indian J Orthop 2013;47:408-12.

Source of Support: Nil, **Conflict of Interest:** None.