Ogilvie's syndrome after lower extremity arthroplasty

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Objective: To alert surgeons who perform arthroplasty to the possibility of acute colonic pseudo-obstruction (Ogilvie's syndrome) after elective orthopedic procedures. To identify possible risk factors and emphasize the need for prompt recognition, careful monitoring and appropriate management so as to reduce morbidity and mortality.

Design: A case series.

Setting: A university-affiliated hospital that is a major referral centre for orthopedic surgery.

Patients: Four patients who had Ogilvie's syndrome after lower extremity arthroplasty. Of this group, 2 had primary hip arthroplasty, 1 had primary knee arthroplasty and 1 had revision hip arthroplasty.

Main outcome measures: Morbidity and mortality.

Results: In all 4 patients Ogilvie's syndrome was recognized late and required surgical intervention. Two patients died as a result of postoperative complications.

Conclusions: Our case series identified increasing age, immobility and patient-controlled narcotic analgesia as potential risk factors for Ogilvie's syndrome in the postoperative orthopedic patient. Prompt recognition and early consultation with frequent clinical and radiographic monitoring are necessary to avoid colonic perforation and its significant associated death rate.

Objectif : Prévenir les chirurgiens qui procèdent à des arthroplasties de la possibilité d'une pseudo-occlusion aiguë du côlon (syndrome d'Ogilvie) après des interventions orthopédiques électives. Définir les facteurs de risque possibles et insister sur la nécessité de les reconnaître rapidement, de les suivre attentivement et de les prendre en charge comme il se doit de façon à réduire la morbidité et la mortalité.

Conception : Série de cas.

Contexte : Hôpital affilié à une université qui constitue un important centre de référence pour la chirurgie orthopédique.

Patients : Quatre patients qui ont eu le syndrome d'Ogilvie après une arthroplastie des membres inférieurs. Deux ont subi une arthroplastie primaire de la hanche, un, une arthroplastie primaire du genou et un, une arthroplastie secondaire de la hanche.

Principales mesures des résultats : Morbidité et mortalité.

Résultats : Chez les quatre patients, on a reconnu le syndrome d'Ogilvie trop tard et il a fallu pratiquer une intervention chirurgicale. Deux patients sont morts à la suite de complications postopératoires.

Conclusions : Notre série de cas a permis de définir l'âge élevé, l'immobilité et l'analgésie au moyen de stupéfiants contrôlée par le patient comme facteurs de risque éventuel du syndrome d'Ogilvie chez un patient qui a subi une intervention chirurgicale orthopédique. Il faut reconnaître rapidement le syndrome, consulter rapidement et effectuer un monitorage clinique et radiographique fréquent pour éviter la perforation du côlon et son taux élevé de morbidité connexe.

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Acute pseudo-obstruction of the colon is a relatively uncommon but potentially serious condition characterized by the clinical and radiographic appearance of obstruction in the absence of an obstructing lesion. This pattern was first identified in 1948 by Ogilvie, who described the condition in a series of patients with malignant retroperitoneal infiltration, leading him to conclude that the condition was caused by "sympathetic deprivation."[1] The syndrome has since been described with a wide variety of metabolic, traumatic and postoperative states[2] and has been referred to by a number of terms, including acute colonic pseudo-obstruction, colonic ileus and Ogilvie's syndrome. The syndrome has a reported death rate ranging from 25% to 31%, reflecting the severity of concomitant underlying illness in these patients.[3]

Previous reports emphasized the need for early diagnosis and prompt intervention.[2,4,5] Although there have been isolated reports of Ogilvie's syndrome after total joint arthroplasty[6] and extremity trauma,[7] there has been relatively little mention of this potentially fatal complication after elective orthopedic procedures.[3,8,9] There have been no reports of Ogilvie's syndrome after total knee replacement or after lower extremity surgery.

The purpose of this paper is to alert arthroplasty surgeons to the possibility of this serious postoperative complication through a small series of patients seen at our institution, to identify possible etiologic risk factors and to emphasize the need for prompt recognition, careful monitoring and appropriate management in order to reduce morbidity and mortality.

Case reports

Case 1

A healthy 70-year-old man with osteoarthritis (OA) of his left knee was admitted electively for a cemented total knee arthroplasty. Postoperatively, we used patient-controlled analgesia (PCA) with a morphine drip and adjusted-dose coumarin prophylactically for deep venous thrombosis (DVT). Two days later, the patient had 2 episodes of coffee-ground emesis and diffuse abdominal pain. The abdomen was mildly distended but not tender, and bowel sounds were present. Upper gastrointestinal endoscopy revealed a hiatus hernia with irregular mucosa on the greater curvature of the stomach, so he was treated intravenously with H_2-blocking agents.

On the fifth postoperative day the patient had 3 episodes of clear emesis and demonstrated a significantly distended, hyperresonant but still nontender abdomen with high-pitched bowel sounds. Radiographs demonstrated a distended cecum, 13 cm in diameter, with little gas in the small bowel. Nasogastric and rectal tubes were inserted and colonoscopy was attempted without relieving abdominal distension. Urgent laparotomy revealed a massively dilated colon without an enlarged small bowel or evidence of obstruction. A tube cecostomy was inserted and broad-spectrum antibiotic therapy was begun.

The patient gradually improved, and 1 month after his admission he had the cecostomy tube removed. He was transferred to a rehabilitation hospital with a functioning knee. When examined 2.5 years after the arthroplasty, his knee was stable and pain-free with a range of motion from 5° to 110°.

Case 2

A 61-year-old man was admitted for a noncemented right total hip arthroplasty. He was hypertensive, a heavy smoker and had chronic obstructive pulmonary disease. Postoperatively, low-molecular-weight heparin was administered subcutaneously for DVT prophylaxis, and PCA with morphine was started. Two days later the patient had nausea and his abdomen became distended. Radiographic findings were consistent with an adynamic ileus. The next day the patient had 2 episodes of painless coffee-ground emesis. Upper endoscopy revealed 4 duodenal ulcers that were not actively bleeding so intravenous H_2-blocking agents were instituted and his diet was advanced.

Three days later, his abdomen was significantly distended although bowel sounds were still present. He had a bowel movement and passed flatus that day, and he did not complain of nausea or abdominal pain. Radiographs demonstrated a dilated cecum, to 12 cm diameter. He also had an elevated potassium level and leukocyte count. A nasogastric tube was inserted and he was monitored daily by abdominal examination and radiography. Two days later the cecum measured 15 cm in diameter and colonoscopy revealed visible ischemic changes (Fig. 1). At laparotomy his colon was a dusky colour and dilated without obstruction. A right hemicolectomy with primary anastomosis was performed and broad-spectrum antibiotic therapy was begun (Fig. 2).

Ten days later an intra-abdominal abscess developed, resulting in systemic sepsis. A defunctioning loop ileostomy was created. One month after his second laparotomy he was discharged, able to walk without hip pain. Two months later he was admitted electively for closure of the loop ileostomy, and died on the ward after suffering a myocardial infarction.

Case 3

A 68-year-old man was admitted electively for a right total hip arthro-

plasty. He had previously undergone left nephrectomy for renal cell carcinoma, and he had hepatic cirrhosis with significant ascites. Morphine was given intermittently for postoperative analgesia. Two days later, DVT of the calf was noted on routine Doppler ultrasonography, even though low-molecular-weight heparin had been given subcutaneously. On the fourth postoperative day the patient was tolerating a full diet with no complaints and was transferred to a rehabilitation hospital.

Three days after discharge, the patient was seen in the emergency department hypotensive and moribund with a grossly distended abdomen. Paracentesis demonstrated gross spillage of fecal contents, and upright radiographs revealed free air under the diaphragm. At urgent laparotomy a necrotic, perforated cecum was found and a cecostomy was performed after abdominal lavage. The patient was managed in the intensive care unit by broad-spectrum antibiotic therapy. He initially required inotropic support and slowly demonstrated multisystem organ failure. After discussing the poor prognosis with his family, we withdrew aggressive support and did not resuscitate. The patient died of related complications 8 months after admission.

Case 4

A 54-year-old man was admitted electively for revision of the acetabular component of his left total hip arthroplasty. He had a history of cigarette smoking, hypertension and had received a cadaver renal transplant 8 years previously. Postoperatively low-molecular-weight heparin was given prophylactically for DVT and patient-controlled morphine infusion for analgesia. Two days later, a large incisional hematoma was noted and his hemoglobin level was only 49 g/L. He was otherwise well. Four units of packed red blood cells were transfused over the next 2 days. His urine output decreased and his renal function worsened. On the fifth postoperative day he had 2 episodes of coffee-ground emesis associated with mild abdominal distension and dark stools. Intravenous H_2-blocker therapy was instituted. Two days later a renal biopsy showed acute tubular necrosis, and he was treated with pulsed corticosteroids. On the ninth postoperative day, he was tolerating a full diet and was transferred to a rehabilitation hospital.

He was admitted emergently from the rehabilitation hospital 3 days later with abdominal pain, distension and feculent emesis. Upright radiographs showed free air under the diaphragm, and urgent laparotomy demonstrated a perforated cecum. The cecum was resected and an end-ileostomy created. He was transferred to the intensive care unit. Broad-spectrum antibiotic therapy was begun. Twenty days after his second admission, he had a closed posterior dislocation of his left hip, which was reduced on the ward with the aid of intravenous sedation and analgesia. He also had worsening renal function, which needed hemodialysis. He was discharged home 4 months later able to walk without pain and with a well-functioning ileostomy. Delayed closure of the ileostomy was planned.

Discussion

Although the precise mechanism remains unclear, a number of factors may act together in the development of acute colonic pseudo-obstruction.

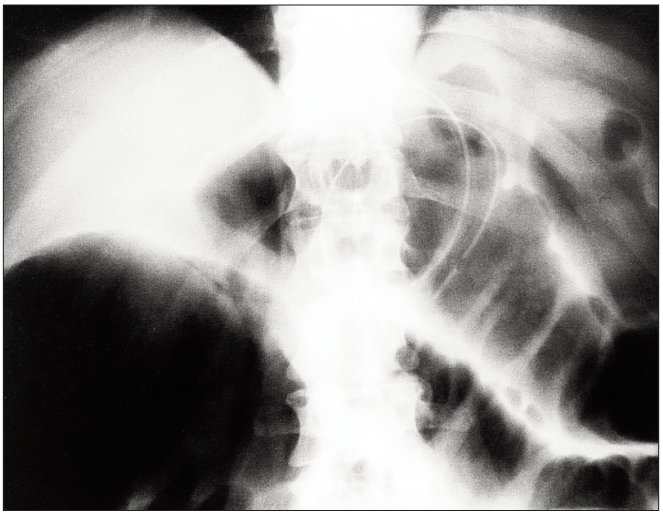

FIG. 1. Case 2. Plain abdominal radiograph of a 61-year-old man after noncemented total hip arthroplasty demonstrates a markedly dilated colon that has persisted despite the insertion of a nasogastric tube.

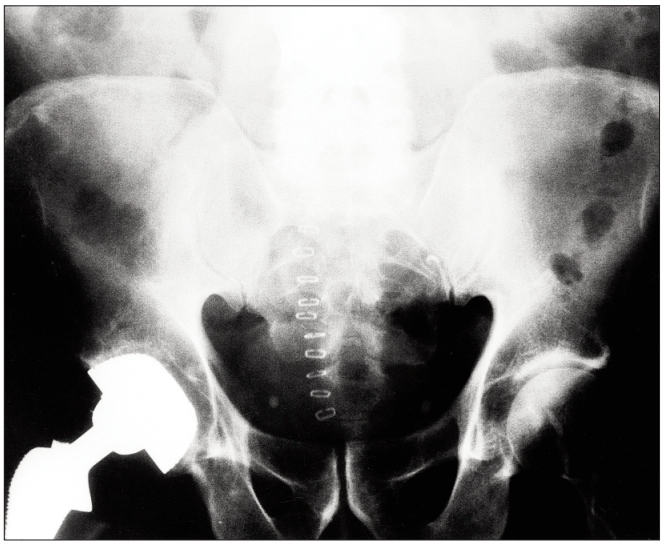

FIG. 2. Case 2. Plain abdominal radiograph after laparotomy and colectomy demonstrating adequate decompression of the large bowel.

Ogilvie's syndrome may be the result of autonomic imbalance, with a combination of excessive sympathetic stimulation or impairment of sacral parasympathetic outflow.[4] The "fluid lock" theory implicates prolonged recumbency, causing dependent bowel loops to collect fluid and air. This may lead to profound cecal dilatation in the presence of a competent ileocecal valve.[8] Ogilvie's syndrome has been reported to be associated with increased age,[8] prolonged bedrest, blunt abdominal, spinal and multiple extremity trauma,[7] continuous level narcotic use (PCA),[3] systemic sepsis, vaginal delivery or cesarean section,[5] abdominal or retroperitoneal malignant disease,[10] cardiac and pulmonary insufficiency,[9] intoxication,[7] medications (phenothiazines, calcium-channel blockers, steroids),[4,10] and metabolic abnormalities such as diabetes, uremia and hypokalemia.[4,6]

The dominant clinical feature of Ogilvie's syndrome is abdominal distension. Other symptoms, occurring with variable frequency, are nausea, vomiting, crampy abdominal pain and dyspnea secondary to diaphragmatic restriction.[10] The patient is rarely obstipated, but there is usually minimal passage of flatus and stool. The physical findings include a nontender or minimally tender abdomen with marked distension, tympanitic percussion and variable bowel sounds. In the absence of ischemia or perforation of the cecum, there are no signs of peritonitis. Digital examination usually reveals an empty rectum.[5] Laboratory analysis often reveals a mild leukocytosis, especially when the bowel is ischemic or has perforated. Electrolyte abnormalities may include hypocalcemia, hyponatremia and hypokalemia.[4] Plain abdominal x-ray films typically reveal massive colonic dilatation with comparatively little gas in the small bowel.[5] Although spontaneous resolution is common, 15% of untreated cases will progress to cecal perforation and subsequent peritonitis.

The differential diagnosis of Ogilvie's syndrome includes true mechanical obstruction, ischemic colitis, acute gastric dilatation and adynamic ileus.[6] Adynamic or paralytic ileus is characterized by generalized bowel hypomotility, mild to moderate abdominal distension and abdominal radiographs that show dilatation of both small and large bowel.[5] The diagnosis of acute gastric dilatation can be quickly confirmed and treated by passsing a nasogastric tube.[6] It is imperative that suspected Ogilvie's syndrome be distinguished from cecal volvulus and other true obstructive conditions of the large bowel.[2,10] Patients with uncomplicated Ogilvie's syndrome have minimal or no abdominal tenderness, appear well despite massive abdominal distension and fail to demonstrate the high-pitched tinkling bowel sounds often associated with mechanical obstruction.[6]

Once abdominal distension has been noted, and the diagnosis of Ogilvie's syndrome is being considered, initial management should include withdrawal of oral intake and narcotic analgesics, administration of intravenous balanced electrolyte solution, placement of a nasogastric tube and the correction of any metabolic abnormalities, including electrolyte disturbances. Other less universally agreed upon treatments reported in the literature are the adjunctive use of laxatives and promotility agents,[3,6] or epidural anesthesia to block sympathetic innervation.[5] Timely general surgical consultation for frequent monitoring with clinical and radiologic abdominal examinations is crucial.

Current definitive management of Ogilvie's syndrome is aimed at direct mechanical decompression of colonic

Table I

Ogilvie's Syndrome in 4 Patients After Arthroplasty

Case no.	Sex/ age, yr	Diagnosis	Treatment	Delay in diagnosis, d	Cecal diameter, cm	Result
1	M/70	Avascular necrosis of left lateral femoral condyle	Cemented total knee arthroplasty	2	13	Tube cecostomy
2	M/61	Osteoarthritis of the right hip	Uncemented total hip arthroplasty	4	15	Colectomy, ileostomy. Died 4 mo postop of myocardial infarction
3	M/68	Osteoarthritis of the right hip	Cemented total hip arthroplasty	5	N/A	Perforated cecum, cecostomy, sepsis. Died 8 mo postop of multisystem organ failure
4	M/54	Osteoarthritis of both hips	Revision of an uncemented acetabular component	7	N/A	Perforated cecum, colectomy, ileostomy

gaseous distension. For those with a benign abdomen and massive cecal dilatation, colonoscopic or percutaneous tube cecostomy decompression are reasonable alternatives. Colonoscopic decompression for Ogilvie's syndrome has become the most widely applied first-line treatment since its introduction by Kukora and Dent[11] in 1977. Some authors suggest that the risk of perforation is related more to the rate and duration of cecal dilatation than to the absolute cecal diameter.[4] Cecal dilatation accompanied by signs of peritoneal irritation, clinical deterioration or laboratory evidence of ischemic bowel constitute indications for laparotomy.[3] Cecal perforation with free intraperitoneal air represents an absolute indication for laparotomy and is accompanied by a reported death rate of between 43% and 50%.[6,9,10]

Other authors have identified orthopedic patients as being at risk for colonic pseudo-obstruction only if there has been trauma or surgery involving the retroperitoneum or pelvis.[4] Our report demonstrates that even after elective lower extremity arthroplasty, patients are at risk for Ogilvie's syndrome. One risk factor in lower extremity arthroplasty patients may be their increasing age. The average age of the patients in our case series was 63 years (Table I), which is consistent with previous reports that the majority of those afflicted are older than 40 years.[2,3,5] Chambers and associates[3] identified the use of narcotic patient-controlled analgesia infusions, which were used in 3 of our 4 cases, as a risk factor for Ogilvie's syndrome. They theorized that the peak-and-valley phenomenon of intermittently administered intramuscular narcotics, although less efficacious for pain relief, may facilitate gut peristalsis. Although age appears to be an independent risk factor, elderly patients are also more sensitive to the antiperistaltic effect of morphine on the gastrointestinal tract.[3] It is interesting that although none of the patients in our series were ordered strict bed rest, all 4 were reluctant to ambulate because of postoperative pain or apprehension except for short periods of supervised physiotherapy. Prolonged recumbency is probably the most prevalent risk factor for this condition in orthopedic patients, further emphasizing the need for early mobilization after lower extremity arthroplasty.[6,8]

Unfortunately, delay in the diagnosis of Ogilvie's syndrome is common, as patients may be tolerating a diet with no abdominal distress.[4] Delay in diagnosis was a significant contributor to the adverse outcome and death rate in our cases. Recognition of abdominal distension was delayed in rehabilitation hospitals, where the staff may be less accustomed to recognizing this serious postoperative complication. Early recognition by thorough physical examination and abdominal radiography is required to avoid unnecessary morbidity and mortality.

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