

# Fatal Late Onset Ogilvie's Syndrome Causing Cecal Perforation after Unilateral Total Knee Arthroplasty

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## Authors' contributions

This work was carried out in collaboration between all authors. Author AA was the primary consultant in this case. He contributed to the conception, design and analysis of the case study. He wrote the first draft of the manuscript and approved the final work to be published. Author HS was the attending surgeon in the case. Authors NS and AC managed the tabulation work, literature searches and final grammar correction of the manuscript. All authors read and approved the final manuscript.

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Case Study

## ABSTRACT

Acute colonic pseudo-obstruction (Ogilvie's syndrome) is a disorder characterized by acute dilatation of the colon in the absence of an anatomic lesion that obstructs the flow of intestinal contents. It is characterized by massive dilatation of the cecum and right colon on abdominal X-ray. The main clinical feature in patients with acute colonic pseudo-obstruction is abdominal distension. We present a case of an elderly male who developed late Ogilvie's syndrome after a month of unilateral total knee arthroplasty (TKA). He was managed conservatively but later developed cecal perforation and was operated upon. However he succumbed to his illness. The diagnosis and management of the case and Ogilvie's syndrome is discussed.

**Keywords:** Total knee arthroplasty; abdomen distention; Ogilvie's syndrome; bowel dilatation; cecal perforation.

## 1. INTRODUCTION

In 1948 Ogilvie H first described two cases of massive colonic dilatation in the absence of

mechanical obstruction [1]. Since then many case reports and case series have been published. However, the exact cause of colon dilatation in the absence of mechanical

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obstruction remains obscure. Most current research supports the theory that it is due to large bowel parasympathetic dysfunction. There perhaps occurs excessive parasympathetic suppression in most cases and this theory is supported by the successful use of parasympathomimetic agents in the treatment of Ogilvie's syndrome [2,3]. In a case series Neostigmine methyl sulfate had been shown to completely resolve Ogilvie's syndrome in 12 of 18 patients [4].

The main clinical feature is abdominal distension that usually occurs gradually over three to seven days but may develop rapidly within 24 to 48 hours. Other features are abdominal pain (80% patients), Nausea and vomiting (60% patients), Constipation (50% patients) and paradoxically diarrhea (40% patients).respectively [5,6]. Diagnosis is based on physical examination (tympanitic abdomen), observation, and imaging to diagnose dilatation of the colon. Ogilvie's syndrome in the postoperative patient is not easily diagnosed as it is confused with simple postoperative ileus due to metabolic cause such as dyselectrolytemia. This happened in the present case which is discussed. Most of the patients can be managed with conservative treatment consisting of nasogastric tube placement with gravity aided suction; fluid resuscitation, enemas, colonoscopy, and decompression colonoscopy in some .The prognosis depends on the presence of complications.

The mortality rate in acute intestinal pseudo-obstruction in the absence of complications is approximately 15 percent with early appropriate management as compared with up-to 60% in patients with a perforated or ischemic bowel [7].

## 2. CASE PRESENTATION

A 68 year-old man with a history of chronic smoking, hypertension, chronic obstructive airway disease and kyphoscoliosis was referred to the orthopedic outpatient department of another facility for right side total knee arthroplasty (TKA). He had complaints of chronic progressive bilateral knee pain for many years due to osteoarthritis of knee joints. The pain was worse on the right side. There was no evidence of neurological, gastrointestinal, immunological, or hematological dysfunction, or underlying malignancy on systemic examination. His surgical history was remarkable for lumbar laminectomy done more than 10 years back for

back pain. However, no records were available for perusal. His routine preoperative laboratory tests, including complete blood count, electrolytes, and coagulation profile were normal.

The patient underwent uncomplicated elective right total knee arthroplasty (TKA) (Fig. 1) on May 09, 2018 which was performed under epidural anesthesia. Perioperatively he received epidural analgesia (neuraxial analgesia) with ropivacaine 0.2% for 48 hours. He was discharged on the fourth postoperative day.



**Fig. 1. Post operative X ray of right knee dated June 21.06.2018**

On day 31 postoperatively, June 08, 2018, the patient was admitted through the emergency department in medical intensive care unit (MICU) of the same facility with complaints of fever, abdominal discomfort and distention, bilateral flank pain and decreased urine output of 5-6 day duration. At the time of admission his GCS was 15/15, temperature 100°F, heart rate 90 beats/minute, BP: 130/90 mm Hg, respiratory rate 25/minute, and SO<sub>2</sub> on air 95%. His examination showed that he had abdominal gaseous distention with tympanic note on

percussion, bilateral decreased air entry over bases with few crackles, and no knee findings. Rest of the systemic examination was unremarkable. His laboratory investigations are displayed in Tables 1 and 2. His total leucocyte count were raised, he had dyselectrolytemia (hyponatremia, hypokalemia and hypomagnesemia), hypoalbuminemia and mild azotemia with a serum creatinine 1.8 mg/dl. Contrast enhanced computerized tomogram (CECT) abdomen (Fig. 2) was done on June 10, 2018 which showed fluid filled dilated bowel loops in whole peritoneal cavity, circumscribed edematous bowel loops with wall thickening at distal ileal loops, ileocecal junction and cecum with adjacent fat stranding and mild bilateral pleural effusion with basal atelectasis. Fig. 3A shows his chest X Ray. In view of leukocytosis, fever and pneumonitis he was given injection piperacillin + tazobactam, levofloxacin and other supportive medications. His dyselectrolytemia was managed and he was put on continuous Ryles (nasogastric) tube aspiration (CRTA). His blood and urine culture grew *Escherichia coli* which was sensitive to carbapenems. His antibiotics were changed to imipenem+cilastin and clindamycin. However, his abdominal symptoms persisted with gradually increasing abdominal distention with a tympanitic note on abdominal percussion and right basal consolidation. He was then referred to another facility with the diagnosis of consolidation, sepsis, dyselectrolytemia with persistent paralytic ileus on June 18, 2018.

He was admitted at Fortis Escorts Hospital, Jaipur on the same day through emergency department in MICU. On admission his vital parameters were as follows: Heart rate 122/minute, respiratory rate 22/minute, Blood pressure 112/74 mm Hg, temperature 98.6°F and SpO2 97% on air. He looked toxic and was on CRTA and indwelling Foleys catheter. His abdomen was significantly distended, hyper-resonant but non-tender with absent bowel sounds. He also had right side infrascapular coarse crackles suggesting consolidation. An upright chest radiograph revealed free air under the diaphragm and a right basal consolidation (Fig. 3B). An urgent CT abdomen with oral and rectal contrast revealed gross pneumoperitoneum, a cecal perforation, dilated cecum with internal diameter of 7.5 cm, mild free fluid abdomen, and right basal consolidation (Figs. 4A and 4B).

At urgent laparotomy on 19/06/2018, a perforated cecum and fecal peritonitis was found. Repair of the perforation and an ileostomy was performed by the surgical team after abdominal lavage. Postoperatively patient required vasopressor, mechanical ventilation, and was managed in surgical intensive care unit (SICU). His blood, peritoneal fluid, and urine grew polymicrobial flora which was managed with antibiotics as per culture sensitivity reports. However, he developed progressive worsening with multi organ dysfunction and succumbed on July 17, 2018.



**Fig. 2. C ECT abdomen dated 10.06.2018. A shows grossly dilated bowel loops. B show thickened caecal wall (arrow) with adjacent fat stranding and dilated bowel loops. C show bilateral pleural effusion with underlying atelectasis**

**Table 1. Hematology and biochemistry**

Date	Hb (13-17 gm/dl)	TLC (4- 10x10 <sup>3</sup> /mm <sup>3</sup> )	DLC (%)	PT/INR (upto 1.2 ratio)	S.Creat. (0.8-1.3 mg/dl)	BUN (8-23 mg/dl)	AST (0-35 IU/L)	ALT (0-41 IU/L)	S.Albumin (3.4-4.8 gm/dl)	S.Na <sup>+</sup> /S.K <sup>+</sup> (136-145 meq/L)
07.05.2018	10.9	10.5	P:52 L:30	1.07	0.60	11.43				143/3.6
10.05.2018	10.4	10.0	P:68 L:30							
08.06.2018	8.4	16.5	P:90 L:14		1.8		19	26	2.2	124/2.7
14.06.2018	8.3	18.7	P:90 L:08		0.8					136/3.1
18.06.2018	9.4	15.8	P:90 L:06		2.08	60	16	08	1.7	166/3.99
20.06.2018										
27.06.2018	8.0	11.5	P:59 L:25		1.51					155/4.59
02.07.2018	7.9	8.2			1.25					142/4.02
07.07.2018	8.2	12.0		1.77	1.69					156/6.37

*Hb: Hemoglobin; TLC: Total leucocyte count; DLC; Differential leucocyte count; S. Creat.: Serum creatinine; BUN: Blood urea nitrogen; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; S.Na<sup>+</sup>: Serum Sodium; S.K<sup>+</sup>: Serum Potassium*

**Table 2. Other investigations**

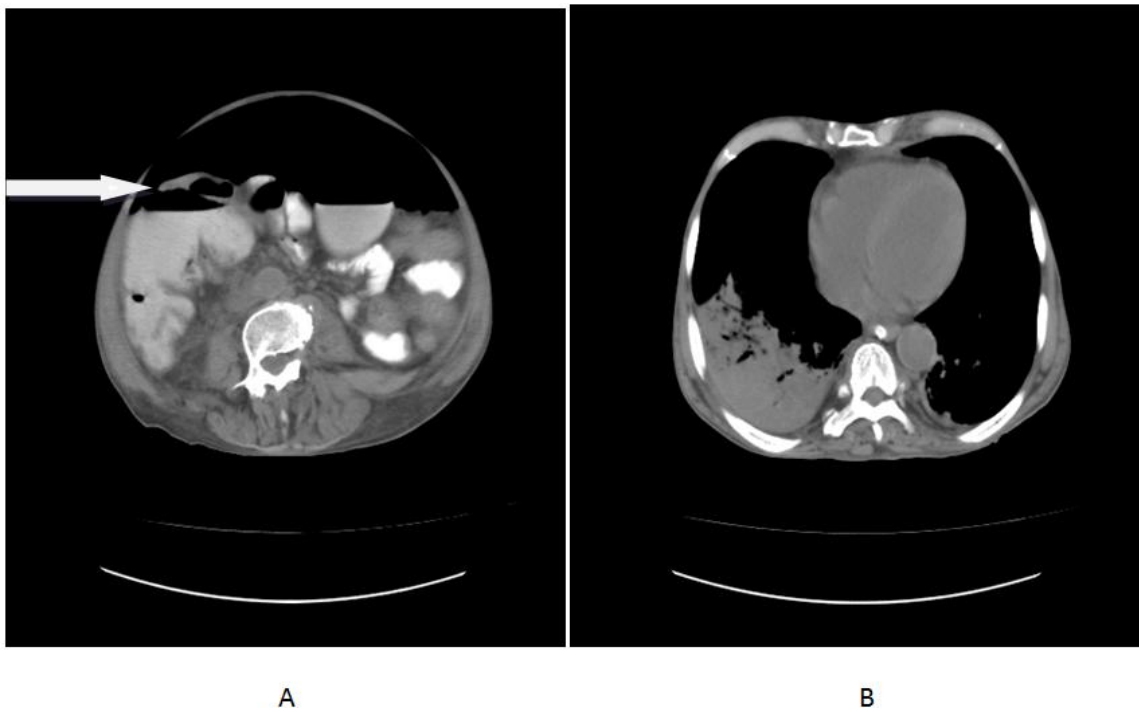
Date	Investigations	Results
08.05.2018	HIV 1 and 2 antibody HBsAg HCV ABS ECG	Not detected Negative Not detected WNL
08.06.2018	Urine Culture Urine Routine	E.Coli 8-10wbc/hpf
08.06.2018	Ultrasound whole abdomen	Mild Prostatomegaly
11.06.2018	Blood Culture	E.Coli

<b>Date</b>	<b>Investigations</b>	<b>Results</b>
15.06.2018	Pleural Fluid	TLC:1400/cmm, Lymp:20%, Neutrophil:80%, Rbc:10-12 Mesothelial Cells +, Sugar :181, Protein:2.2, Albumin:1.0
16.06.2018	ABG	7.56/28/54/25.1/104
18.06.2018	PBF	Predominantly microcytic hypochromic anemia with mild evidence of hemolysis , neutrophilic leucocytosis and thrombocytopenia.
18.06.2018	TSH	7.940(0.27-4.2)
18.06.2018	Free T4	0.64 ng/dl(0.93-1.7ng/dl)
20.06.2018	Peritoneal Drain	Positive For 1. Pseudomonas aeruginosa 2. Klebsiella pneumoniae (carbapenemase producing strain) 3. Enterococcus faecium (vancomycin resistant enterococcus strain)
	S.Procalcitonin	18.640ng/ml(0-0.046ng/ml)
21.06.2018	Wound Swab	Positive For 1. Pseudomonas aeruginosa 2. Klebsiella pneumoniae (carbapenemase producing strain) 3. Enterococcus faecium (vancomycin resistant enterococcus strain)
23.06.2018	Abdominal Drain Fluid	Positive For 1. Pseudomonas aeruginosa 2. Klebsiella pneumoniae (carbapenemase producing strain)
27.06.2018	Creatine kinase	66 U/L(39-308U/L)
27.06.2018	N-Terminal Pro B-Type natriuretic peptide	3716 pg/ml(0-125Pg/ml)
27.06.2018	Hs Troponin T	77 Pg/ml(0-14Pg/ml)
30.06.2018	Stool For occult blood	Occult blood detected

*TLC: Total leucocyte count; RBC: Red blood cells; WBC: White blood cells; PBF: Peripheral blood film; HIV: Human immunodeficiency virus; HBsAG: Hepatitis B surface antigen; TSH: Thyroid stimulating hormone; HCV ABS: Hepatitis C Antibody; E. coli: Escherichia coli; Hs troponin T: High sensitivity troponin T  
Lymp: Lymphocyte; USG: Ultrasonography; WNL: Within normal limit*



**Fig. 3. A. XRay chest dated 16.06.2018 showing kyphoscoliosis. B. XRay Chest dated 18.06.2018 show free air under diaphragm (arrow) along-with right basal consolidation and kyphoscoliosis. C. Postoperative day 1 XRay Chest dated 20.06.2018 showing right basal consolidation**



**Fig. 4. CECT abdomen dated 19.06.2018. Abdomen and lower chest with oral and rectal contrast. A show gross pneumoperitoneum with breach in caecal wall anteriorly (arrow). B. show right basal consolidation**

### 3. DISCUSSION

We present an uncommon postoperative complication following an elective TKA in a 68-year-old man who developed Ogilvie's syndrome 25 days post TKA, which was managed

conservatively and later developed cecal perforation and succumbed to postoperative complications.

Ogilvie syndrome is a rare complication of surgery and is reported to occur after

obstetrical/gynecologic, abdominal/pelvic, and orthopedics procedure. However, recent data confirms that patients undergoing orthopedic and spinal procedures are at higher risk but the surgical procedure most commonly leading to Ogilvie's syndrome is coronary artery bypass surgery [7]. Drugs that disturb colonic motility (e.g., anticholinergics, opioid analgesics, phenothiazines, calcium channel blockers, alpha-2-adrenergic agonists, epidural analgesics) contribute to the development of this condition.

Though reported in children acute colonic pseudo-obstruction appears to be more common in men and in patients over the age of 60 years [5]. It can occur acutely or as a chronic condition [5]. In patients with acute colonic pseudo-obstruction, increasing colonic diameter increases the risk of colonic ischemia and perforation. The risk of colonic perforation increases when cecal diameter exceeds 10 to 12 cm and when the distention has been present for greater than six days [8]. The duration of dilation is probably more important than the absolute diameter of the colon [9,10]. In the case presented the cecal diameter was 7.5 cm but it persisted for almost two weeks and lead to cecal perforation.

The incidence of post operative ileus (POI) after total joint arthroplasty (TJA) is small, yet not uncommon, and it has been reported to range from 0.3% to 4.0% [11,12]. It is reported even higher (5.6%) after revision in total hip arthroplasty [13].

Although the precise mechanism remains unclear, a number of factors may act together in the development of acute colonic pseudo-obstruction [14].

The Ogilvie's syndrome is reported to be associated with increased age, prolonged bed rest, blunt abdominal, spinal and multiple extremity trauma, continuous level narcotic use (PCA), systemic sepsis, vaginal delivery or cesarean section, abdominal or retroperitoneal malignant disease, cardiac and pulmonary insufficiency, intoxication, medications (phenothiazines, calcium-channel Blockers, steroids) and metabolic abnormalities such as diabetes, uremia and hypokalemia [14]. However, patients undergoing orthopedic and spinal procedures are at higher risk, but the surgical procedure most commonly leading to

Ogilvie syndrome was reported to be coronary artery bypass grafting [7].

This patient had increased age, prolonged immobilization after TKA, dyselectrolytemia, and orthopedic surgery as the risk factors. Though advised to be ambulant he was just reluctant to ambulate because of postoperative pain or apprehension except for short periods of supervised physiotherapy.

This patient was later readmitted after 25 days as POI with electrolyte abnormalities. POI is distinguished by an accumulation of gas and secretions resulting from a lack of bowel movements. If not recognized early or improperly managed, it may result in more severe complications such as bowel perforation, peritonitis, sepsis, multiorgan failure, and even death [15]. Though managed conservatively, despite persistent bowel dilatation with Cecal diameter of 7.5 cm for two weeks, possibility of Ogilvie's syndrome was not considered and no decompressive colonoscopy was attempted. In a large series of 400 patients, all patients with a Cecal diameter of >12 cm perforated as compared to 3 of 17 patients with a diameter of <9 cm. Most perforations were diagnosed between day 3 and day 5 [5]. The risk of colonic perforation increases when cecal diameter exceeds 10 to 12 cm and when the distention has been present for greater than six days [8]. The duration of dilation appears to be more important than the absolute diameter of the colon [9,10]. In this patient ,though the Cecal diameter was < 9 cm ,it persisted for two weeks further emphasizing that duration of Cecal dilatation is more important than Cecal diameter.

The exact cause of POI remains unknown .The sympathetic over activity and/or parasympathetic dysfunction is believed to be the main abnormality. Once abdominal distension has been noted in a patient with underlying risk factors, the diagnosis of Ogilvie's syndrome should be considered at earliest. Initial management include nothing per oral (NPO) withdrawal of narcotic analgesics, administration of intravenous balanced electrolyte solution, placement of a nasogastric tube and management of metabolic abnormalities, including electrolyte disturbances. All these were done in the present case but diagnosis of Ogilvie's syndrome wasn't considered. By the time he was referred he had bowel distention of almost two weeks and had developed cecal

perforation, peritonitis, and sepsis and multi organ dysfunction syndrome.

The definitive management of Ogilvie's syndrome involves direct mechanical decompression of colonic gaseous distension. For those with a benign abdomen, colonoscopy or percutaneous tube colostomy decompression is reasonable alternatives. Colonoscopy decompression for Ogilvie's syndrome has become the most widely applied first-line treatment. All these were not done as the diagnosis of Ogilvie's syndrome wasn't considered.

Unfortunately, delay in the diagnosis of Ogilvie's syndrome is common, as patients still accept and tolerates oral feeds with no abdominal distress. Delay in diagnosis is a significant factor contributing to the adverse outcome and even death as in the present case.

#### 4. CONCLUSION

Cardio-thoracic surgeons, orthopedic surgeons and neurosurgeons should be vigilant of this complication in the patient whose abdomen becomes distended in post operative period. It should be recognized timely and treated appropriately, POI will resolve in most patients. Frequent monitoring with clinical and radiologic abdominal examinations is crucial. We thus emphasize the need for early identification and appropriate management of Ogilvie's syndrome to improve patient safety.

#### CONSENT

The authors have obtained written consent from the family for publication of the case report.

#### ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the authors.

#### COMPETING INTERESTS

Authors have declared that no competing interests exist.

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