

## **Case study**

# **Fatal late onset Ogilvie's Syndrome causing Cecal perforation after Unilateral Total Knee Arthroplasty**

### **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~~ ~~However~~, he succumbed to his illness. The diagnosis and management of the case and Ogilvie's syndrome is discussed.

Comment [NLM[1]: Does this mean he died?

### **Consent**

The authors have obtained the patient's informed written consent for print and electronic publication of the case report.

Comment [NLM[2]: If he died how has/was informed consent obtained from the patient? Was it before his death?

### **Key words**

Total Knee Arthroplasty, Abdomen distention, Ogilvie's syndrome, Bowel dilatation, and cecal perforation.

### **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 which causes colon to dilate in the absence of mechanical 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].

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 [5]. 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 [6]. It can occur acutely or as a chronic condition. 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 [7]. The duration of dilation is probably more important than the absolute diameter of the colon [8,9]. In the case presented the cecal diameter was 7.5-8 cm but it persisted for almost two weeks and lead to cecal perforation.

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 [6, 10]. Diagnosis is based on physical examination (tympanitic abdomen), observation, and imaging to diagnose dilation 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.

Comment [NLM3]: This needs a reference

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. [11].

### 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 review. 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.

Comment [NLM[4]: Examination/Assessment?

The patient underwent uncomplicated elective right total knee arthroplasty (TKA) (Figure 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.

On day 31 postoperatively, June 08, 2018, the patient was admitted through the emergency department in medical intensive care unit (~~MICU~~ 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<sup>0</sup>F, heart rate 90 beats/minute, BP:130/90 mm Hg, respiratory rate 25/minute, and SpO2 on air was 95%. His examination showed that he ~~had abdominal~~ had gaseous 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 mentioned in Table 1 and 2. His total leucocyte count were raised, he had dyselectrolytemia (hyponatremia, hypokalemia and hypomagnesemia), ~~hypoalbuminemia~~, hypoalbuminemia and mild azotemia with a serum creatinine 1.8mg/dl. Contrast enhanced computerized

Comment [NLM[5]: Spell check

Comment [NLM[6]: Space check

Comment [NLM[7]: Displayed?

tomogram (~~CECT) abdomen~~CECT) abdomen (Figure 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. Figure 3A shows his chest X Ray chest. 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 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~~pressure 112/74 mm Hg, temperature 98.6<sup>0</sup>F and SpO<sub>2</sub> 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 (Figure 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 (Figure 4 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 (Figure 5). 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.

## 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.

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% [13,14]. It is reported even ~~higher~~ higher (5.6%) after revision ~~total~~ total hip arthroplasty [15].

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

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 [12]. ~~However, patients~~ 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 [5].

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. Though managed conservatively, despite persistent bowel dilatation with Cecal diameter of 7.5-8cm for two weeks, possibility of Ogilvie's syndrome was not ~~considered~~ considered, and no decompressive colonoscopy was attempted. In a large series of 400 ~~patients~~

**Comment [NLM[8]:** It is a discussion, so you must engage in what we have for our case in relation to what is existing in available literature then you give your opinion for each point raised. Why you think it occurred that way or how it be avoided, made better etc.

**Comment [NLM[9]:** References?

**Comment [NLM[10]:** This sentence is too long please break it into two or three sentences

**Comment [NLM[11]:** Any references to support this discussion?

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 [16]. 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 [17]. The duration of dilation appears to be more important than the absolute diameter of the colon [18,19]. 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.

Comment [NLM[12]: What was it for this case?

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 are 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.

### **Conclusion**

Comment [NLM[13]: What is your conclusion over this particular case in 2 or 4 sentences then you can give your recommendations in line with the outcomes of the case at hand.

1. Cardio-thoracic surgeons-, orthopedic surgeons and neurosurgeons should be vigilant of this complication in the patient whose abdomen becomes distended in post operative period.

2.If recognized timely and treated appropriately, POI will resolve in most patients. Frequent monitoring with clinical and radiologic abdominal examinations is crucial. Early recognition is required to avoid unnecessary morbidity and mortality.

3.If surgical intervention is required the mortality rate is high. We thus emphasize the need for early identification and appropriate management to improve patient safety.

### **References:**

1.Ogilvie H.Large intestine colic due to sympathetic deprivation:a new clinical syndrome.BMJ.1948 Oct 9;2(4579):671-3.

2.Hutchinson Rgriffiths C.Acute colonic pseudo-obstruction:a pharmacologic approach. Annals of The Royal College of Surgeons of England.1992;74(5):364-367.

3.Ponec RJ, Saunders MD, Kimmey MB. Neostigmine for the treatment of acute colonic pseudo-obstruction. N. Engl. J. Med.1999. 341 (3): 137–41.

4. Turegano-Fuentes F, Muñoz-Jiménez F, Del Valle-Hernández E, Pérez-Díaz D, Calvo-Serrano M, De Tomás J, De Fuenmayor ML, Quintans-Rodríguez A. Early resolution of Ogilvie's syndrome with intravenous neostigmine: a simple, effective treatment. Dis Colon Rectum. 1997.40(11):1353-7.

5.Tenofsky PL, Beamer L, Smith RS (2000). "Ogilvie syndrome as a postoperative complication". Arch Surg. **135** (6): 682–6; discussion 686–7.

6.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.

7.Saunders MD. Acute colonic pseudo-obstruction. Best Pract Res Clin Gastroenterol 2007; 21:671.

8.Johnson CD, Rice RP, Kelvin FM, et al. The radiologic evaluation of gross cecal distension: emphasis on cecal ileus. AJR Am J Roentgenol 1985; 145:1211.

9. Sloyer AF, Panella VS, Demas BE, et al. Ogilvie's syndrome. Successful management without colonoscopy. *Dig Dis Sci* 1988; 33:1391.
10. Jetmore AB, Timmcke AE, Gathright JB Jr, et al. Ogilvie's syndrome: colonoscopic decompression and analysis of predisposing factors. *Dis Colon Rectum* 1992; 35:1135.
11. Tenofsky Patty L, Beamer R Larry, Smith R Stephen. Ogilvie syndrome as a post operative complication. *Arch Surg*. 2000;135:682-687.
12. Bederman SS, Betsy M, Winiarsky R, et al. Postoperative ileus in the lower extremity arthroplasty patient. *J Arthroplasty* 2001;16:1066.
13. Pavone V, Johnson T, Saulog PS, et al. Perioperative morbidity in bilateral one-stage total knee replacements. *Clin Orthop Relat Res* 2004;421:155.
14. Berend KR, Lombardi Jr AV, Mallory TH, et al. Ileus following total hip or knee arthroplasty is associated with increased risk of deep venous thrombosis and pulmonary embolism. *J Arthroplasty* 2004;19(7 Suppl 2):82.
15. Amr W. ElMaraghy, Emil H. Schemitsch, Marcus J. Burnstein, James P. Waddell. Ogilvie's syndrome after lower extremity arthroplasty. *Canadian journal of surgery*. 1999; 42(2):133-137
16. 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..
17. Saunders MD. Acute colonic pseudo-obstruction. *Best Pract Res Clin Gastroenterol* 2007; 21:671.
18. Johnson CD, Rice RP, Kelvin FM, et al. The radiologic evaluation of gross cecal distension: emphasis on cecal ileus. *AJR Am J Roentgenol* 1985; 145:1211.
19. Sloyer AF, Panella VS, Demas BE, et al. Ogilvie's syndrome. Successful management without colonoscopy. *Dig Dis Sci* 1988; 33:1391.



**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. (0.8-1.3mg/ dl)	BUN (8-23mg/ dl)	AST (0-35IU/ L)	ALT (0-41IU/ L)	S.Albumin (3.4-4.8gm/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	Not detected Negative Not detected
	ECG	WNL
08.06.2018	Urine Culture	E.Coli
	Urine Routine	8-10wbc/hpf
08.06.2018	Ultrasound whole abdomen	Mild Prostatomegaly
11.06.2018	Blood Culture	E.Coli
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  S.Procalcitonin	Positive For 1. Pseudomonas aeruginosa 2. Klebsiella pneumoniae (carbapenemase producing strain) 3. Enterococcus faecium (vancomycin resistant enterococcus strain)  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



Figure 1: Post operative X ray of right knee dated June 21.06.2018



A

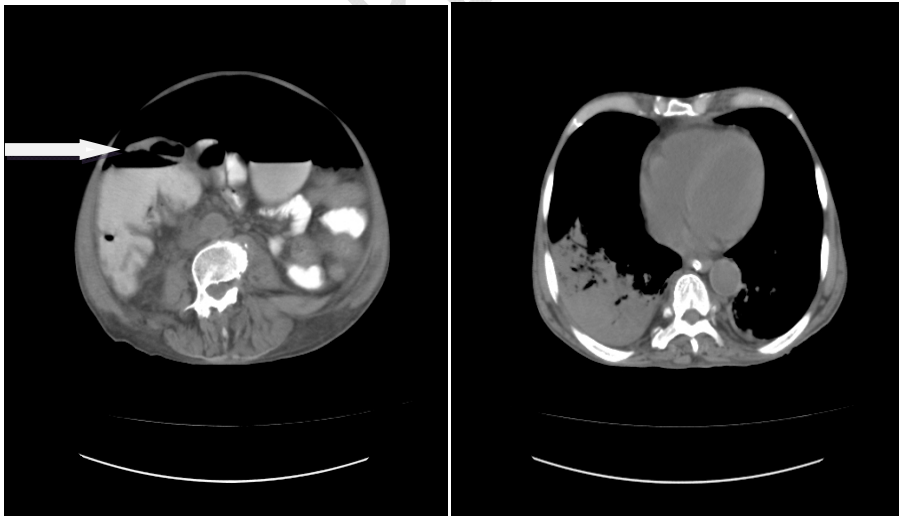
B

C

Figure 2.CECT 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.



Figure 3. A XRay chest dated 16.06.2018showing 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.



A

B

Figure 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.



Figure 5.Post operative ileostomy with drain in situ.