



## Case study

# Acute colonic pseudo-obstruction in two patients admitted with severe acute respiratory syndrome–coronavirus-2 pneumonia



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## ABSTRACT

We have described two cases of severe SARS-CoV-2 pneumonia presenting with acute colonic pseudo-obstruction with normal liver enzymes and serum lactate. These older adults presented predominantly with constitutional symptoms, silent hypoxia, distended abdomen, sluggish bowel sounds, and colonic dilatation supported by abdominal imaging (plain X-ray and computerized tomography of abdomen) to a tertiary care center in South India. Both patients received standard treatment for severe SARS-CoV-2 pneumonia and acute colonic pseudo-obstruction according to available guidelines but succumbed to complications during hospital stay. Acute colonic pseudo-obstruction in patients admitted with SARS-CoV-2 infection requires high index of suspicion as it warrants early mitigation by cessation of offending agents, optimizing electrolytes, and colonic decompression to prevent morbidity and mortality.

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## Introduction

In December 2019, the World Health Organization (WHO) issued a warning regarding an outbreak of respiratory illness in China, and declared a global health emergency on January 30, 2020 as it started spreading across many nations [1]. Although the pathogen – an enveloped single-stranded large RNA virus – resembles other coronaviruses like the Middle East Respiratory Syndrome Coronavirus (MERS-CoV), Severe Acute Respiratory Syndrome Coronavirus (SARS-CoV), SARS-CoV-2 has a unique amino acid sequence of 1 ab polyprotein and surface glycoprotein (S-protein) [2,3]. The novel coronavirus pandemic caused by the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) is presently responsible for more than 3,800,000 deaths worldwide as per WHO data [4]. Current literature mainly focuses on the respiratory symptoms and is limited on extra-pulmonary manifestations, especially gastrointestinal presentations [5]. Colonic pseudo-obstruction with normal liver transaminases have never been reported in patients admitted with SARS-CoV-2. We report two cases of colonic pseudo-obstruction in older patients admitted with SARS-CoV-2.

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## Patient information

## Case-1

An 87-year-old male, a retired teacher, presented to the emergency department with generalized weakness, anorexia and easy fatigability for three days with no localizing symptoms. Comorbidities included vascular dementia, heart failure with reduced ejection fraction, bronchial asthma, essential hypertension, dyslipidemia, benign prostatic enlargement, and functional impairment due to poor mobility. Medication history included hydrochlorothiazide, pantoprazole, atorvastatin, doxofylline, amlodipine, bisoprolol, losartan, clopidogrel, alfuzosin, dutasteride, and inhaled salbutamol. There was no history of recent travel or exposure to sick contacts, exposure to chemicals, agricultural products or radiation, and the patient did not have any avocation. He was a non-smoker and never consumed alcohol.

On examination, he was disoriented and hypoxic (oxygen saturation of 91 %). Other vital signs were normal and there were no features of fluid overload or pulses paradoxus. Per-abdominal examination revealed a distended abdomen with sluggish bowel sounds. Shifting dullness and fluid thrill were absent. Rest of the systemic examination was unremarkable.

On laboratory evaluation, SARS-CoV-2 Real-Time Polymerase Chain Reaction (RT-PCR) nasal swab test was positive. Other abnormal laboratory results were serum sodium (128 mmol/L), serum potassium (3.2 mmol/L), urine red cells of 1475 cells per high power field, elevated C-reactive protein (20.4 mg/dl), D-dimer

(610 ng/mL), serum ferritin (641.2 ng/mL), troponin-T (25.4 pg/mL) with no serial elevation in cardiac enzymes, and low arterial partial pressure of oxygen on room air (PaO<sub>2</sub> – 59 mmHg). Serum alanine transferase (S-ALT), serum aspartate transferase (S-AST) and serum lactate were within normal limits (Table 1) [1–22]. Abdominal X-ray showed distended colonic shadows (Fig. 1A), ultrasound abdomen showed cholelithiasis, and chest X-ray revealed reduced right lung volume, bilateral heterogeneous opacities and obliterated costophrenic angle. In the hours that followed, the patient developed high grade fever, tachypnea and worsening of delirium. It was associated with leukocytosis and left shift (total white cell count of 13,100 with 91 % neutrophils). He was diagnosed to have sepsis with probable gut translocation as the source of infection. Contrast-enhanced computerized tomography (CECT) of the abdomen showed dilated transverse colon, descending colon and markedly dilated redundant sigmoid colon with no functional obstruction (Fig. 1B). Patient was started on intravenous Meropenam. Three blood cultures, stool and urine cultures were sterile. There was no worsening of pulmonary infiltrates on chest X-ray or hypoxemia on arterial blood gas analysis. The patient received standard treatment including correction of electrolyte abnormalities. Sub-acute intestinal obstruction recurred despite conservative management and colonic decompression was deferred in view of poor surgical outcome due to sepsis, poor baseline functional status and cognitive impairment. Unfractionated Heparin was stopped as the patient developed frank hematuria probably due to urethral injury due to an indwelling urinary

catheter. Despite resuscitative efforts, the patient succumbed on day 21 of hospital admission (Table 2).

#### Case-2

A 75-year-old male weaver presented with a one day history of low-grade intermittent fever associated with an episode of post-prandial vomiting, anorexia, generalized weakness and easy-fatiguability. Comorbidities included type-2 diabetes mellitus since 2008, dyslipidemia, and bronchiectasis. Regular medications included metformin, glimepiride and atorvastatin. There was no history of recent travel or exposure to sick contacts, exposure to chemicals, agricultural products or radiation, and the patient did not have any avocation. He was a non-smoker and never consumed alcohol.

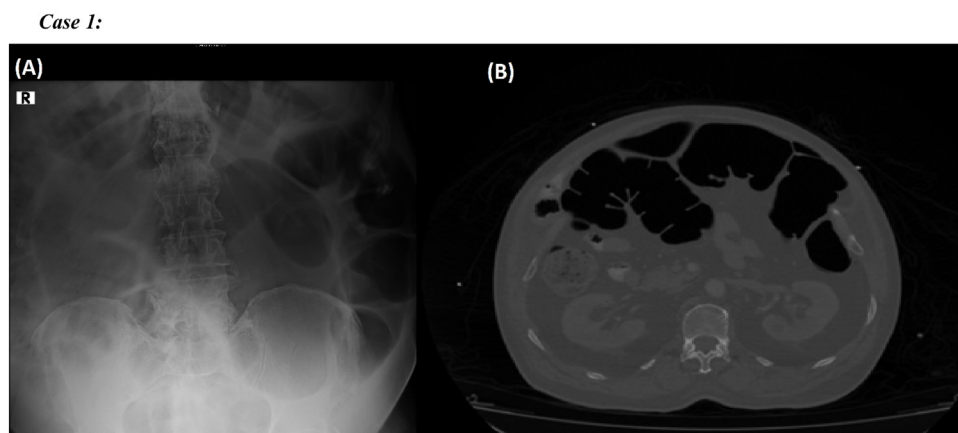
On examination, the patient was conscious. There was tachycardia (pulse rate of 102 beats/minute) and hypoxia (room air saturation of 84 %). Per-abdominal examination revealed a distended abdomen with sluggish bowel sounds. Respiratory system examination revealed bilateral rales.

On laboratory evaluation, the nasal swab RT-PCR test for SARS-CoV-2 was positive. Other notable laboratory results were elevated serum total leucocyte count ( $21.6 \times 10^9$  cells per litre with 83 % neutrophils). In the hours that followed, the patient developed high grade fever, tachypnea and worsening of delirium. It was associated with leukocytosis and left shift (total white cell count of 13,100 with 91 % neutrophils). He was diagnosed to have sepsis

**Table 1**  
Laboratory profile of the patients on admission.

Laboratory profile:	Case 1	Case 2	Normal range
<b>Complete blood count</b>			
Hemoglobin (g/L)	<b>117</b>	<b>122</b>	138 – 172
Total white blood cell count (cells per litre)	$8.2 \times 10^9$	<b><math>21.6 \times 10^9</math></b>	$4.5-11 \times 10^9$
Differential WBC count (%)	N 76 L 14 M 10	<b>N 83 with shift to the left</b>	
Platelet count (cells per litre)	$312 \times 10^9$	$328 \times 10^9$	$150-350 \times 10^9$
<b>Electrolytes</b>			
Serum sodium (mmol/L)	<b>128</b>	<b>132</b>	135–145
Serum potassium (mmol/L)	<b>3.2</b>	4.7	3.5–5
Serum Calcium (mg/dl)	8.26	8.26	8.2–10.3
Serum Phosphate (mg/dl)	3.7	3.7	3.4–4.5
Serum magnesium (mg/dl)	2.12	1.72	1.6–2.6
<b>Liver function test</b>			
Total bilirubin (mg/dl)	<b>1.51</b>	0.67	0.5–1
Direct bilirubin (mg/dl)	<b>0.67</b>	0.50	0.1–0.5
Total protein (g/L)	6.7	7.8	60–80
Albumin (g/L)	3.6	3.4	35–50
Aspartate transaminase (U/L)	38	20	10–40
Alanine transaminase (U/L)	25	14	10–40
Alkaline phosphatase (U/L)	75	<b>198</b>	30–120
<b>Renal function test</b>			
Creatinine ( $\mu\text{mol/L}$ )	75	91.9	38–106
Urea (mmol/l)	1.60	3.9	2.9–8.2
<b>Other tests:</b>			
C-reactive protein (mg/L)	<b>20.4</b>	–	<6
Procalcitonin (ng/mL)	0.13	–	
LDH (U/L)	–	<b>1013</b>	
D-dimer (ng/mL)	<b>610</b>	<b>6765</b>	<500
Ferritin (ng/mL)	<b>641.2</b>	<b>361</b>	20–320
<b>Arterial blood gas at admission</b>			
(PaO <sub>2</sub> and PaCO <sub>2</sub> in mmHg at room air. Bicarbonate and lactate in mmol/L)	<b>pH 7.46</b>	pH 7.44	7.35–7.45
	PaO <sub>2</sub> 59	<b>PaO<sub>2</sub> 50</b>	75–100
	<b>PaCO<sub>2</sub> 32</b>	<b>PaCO<sub>2</sub> 29</b>	35–45
	HCO <sub>3</sub> 22.8	HCO <sub>3</sub> 22	22–26
	Lactate 1.7	Lactate 1.0	0.5–2.2

‘-’ Not done, ‘/’ – per, ‘PaO<sub>2</sub>’ – partial pressure of oxygen in arterial blood, ‘PaCO<sub>2</sub>’ – partial pressure of carbon dioxide in arterial blood, ‘pH’ – power of hydrogen, ‘HCO<sub>3</sub>’ – bicarbonate level in arterial blood.



**Fig. 1.** (A) X-ray Abdomen erect showing colonic gaseous distension. (B) Contrast-enhanced Computerized Tomography of Abdomen showing dilated transverse colon, descending colon without obvious mechanical obstruction.

with probable gut translocation as the source of infection. Contrast-enhanced computerized tomography (CECT) of the abdomen showed dilated transverse colon, descending colon and markedly dilated redundant sigmoid colon with no functional obstruction (Fig. 1B). Patient was started on intravenous Meropenam. Three blood cultures, stool and urine cultures were sterile. There was no worsening of pulmonary infiltrates on chest X-ray or hypoxemia on arterial blood gas analysis. The patient received standard treatment including correction of electrolyte abnormalities. Sub-acute intestinal obstruction recurred despite conservative management and colonic decompression was deferred in view of poor surgical outcome due to sepsis, poor baseline functional status and cognitive impairment. Unfractionated Heparin was stopped as the patient developed frank hematuria probably due to urethral injury due to an indwelling urinary catheter. Despite resuscitative efforts, the patient succumbed on day 21 of hospital admission (Table 2).

**Case-2**

A 75-year-old male weaver presented with a one day history of low-grade intermittent fever associated with an episode of post-prandial vomiting, anorexia, generalized weakness and easy-fatiguability. Comorbidities included type-2 diabetes mellitus since 2008, dyslipidemia, and bronchiectasis. Regular medications included metformin, glimepiride and atorvastatin. There was no history of recent travel or exposure to sick contacts, exposure to chemicals, agricultural products or radiation, and the patient did not have any avocation. He was a non-smoker and never consumed alcohol.

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air saturation of 84 %). Per-abdominal examination revealed a distended abdomen with sluggish bowel sounds. Respiratory system examination revealed bilateral rales.

On laboratory evaluation, the nasal swab RT-PCR test for SARS-CoV-2 was positive. Other notable laboratory results were elevated serum total leucocyte count ( $21.6 \times 10^9$  cells per litre with 83 % neutrophils), serum sodium (132 mmol/L), serum alkaline phosphatase (198 U/L), glycated hemoglobin (HbA1c 10.3 mmol/mol), serum lactate dehydrogenase (1013 U/L), D-dimer (6765 ng/mL), serum ferritin (361 pg/mL), and arterial partial pressure of oxygen on room air (50 mmHg). Serum lactate, S-ALT and S-AST were within normal limits. Abdominal X-ray showed distended colonic shadows (Fig. 2), ultrasound abdomen showed prominent bowel loops in the hypogastrium with reduced peristalsis, and chest X-ray showed bilateral heterogeneous opacities in all lung zones. Financial constraints inhibited him from undergoing CECT abdomen and receiving Remdesivir. He was given the standard treatment for severe SARS-CoV-2 pneumonia and acute colonic pseudo-obstruction according to available guidelines. On day 4 of hospital stay, the patient developed non-oliguric acute kidney injury secondary to dehydration which resolved with adequate fluid resuscitation. On day 11 of hospital stay, he developed fever and was started on broad-spectrum antibiotics to which he responded poorly. He was started on intravenous fluconazole as the blood culture grew *Candida albicans*. However, the patient succumbed despite adequate resuscitative efforts on day 15 of hospital admission.

**Differential diagnosis**

Differential diagnoses considered in these two cases were: mechanical obstruction due to a tumor, toxic megacolon, and intestinal pseudo-obstruction. Colonic pseudo-obstruction has been associated with hypokalemia, hypocalcaemia, hypomagnesaemia, and usage of calcium channel blockers [6]. Case 1 was on Amlodipine and had hypokalemia. *Clostridium difficile* infection was considered less likely as both patients were not administered broad-spectrum antibiotics prior to the onset of intestinal obstruction.

**Discussion**

Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) may present as asymptomatic infection (15.6%), pre-symptomatic infection in asymptomatic patients who develop symptoms during follow up (48.9%), or symptomatic infection with fever and

**Table 2**  
Treatment chart of the patients during hospital stay.

	Case 1	Case 2
Oxygen	+	+
NIV/Invasive ventilation	-	-
Remdesivir	+	-
IV Dexamethasone	+	+
Anticoagulation	+	+
Antibiotics (broad spectrum)	+	+
Management received for acute colonic pseudo-obstruction	NPO IVF Nasogastric dependent drainage and flatus tube	

'-' Did not undergo, '+' Received, NIV - noninvasive ventilation, IV - Intravenous, IVF - intravenous fluid, NPO - nil-per-oral.

## Case 2:



Fig. 2. X-ray Abdomen erect showing colonic gaseous distension.

pulmonary symptoms including cough and breathlessness. However, extra-pulmonary involvement in coronavirus disease 2019 (COVID-19) has been increasingly reported in older adults [5]. The incidence of gastrointestinal symptoms in COVID-19 ranges from 5 percent to 50 percent, and the SARS-CoV-2 virus has been detected in fecal specimens [7,8].

Till date, only six cases of colonic pseudo-obstruction have been reported in literature. Kaafarani et al. described 141 critically ill patients with COVID-19 of which 74 percent developed at least one gastrointestinal complication – 1.9 percent (two patients) had colonic pseudo-obstruction [9]. Sattar et al. described a case series of three patients with colitis of whom one patient had colonic pseudo-obstruction at presentation who was treated successfully with a combination of hydroxychloroquine, azithromycin and zinc sulfate [10]. De Nardi et al. described a patient from Europe admitted with COVID-19-related acute respiratory distress syndrome (ARDS) who subsequently developed colonic pseudo-obstruction with perforation and was successfully managed with surgical decompression [11]. Ibrahim et al. described two patients from Qatar admitted with severe COVID-19 ARDS who developed colonic pseudo-obstruction and were treated by surgical decompression [12]. Although these cases of severe COVID-19 had non-mechanical causes of colonic distention diagnosed clinically or radiologically with elevated liver transaminases, the cases we report had normal liver enzymes, and had abdominal distension with constitutional symptoms at presentation which makes it unique. Serum lactate levels in both patients were within normal range. Serum lactate, a non-specific marker, has been found to be elevated in patients admitted with severe COVID-19 pneumonia with acute colonic pseudo-obstruction. Nevertheless, two cases have reported normal serum lactate levels at the time of admission [13].

There is no standard clinical definition or diagnostic criteria for acute colonic pseudo-obstruction, often described as Ogilvie

syndrome [14,15]. The following clinical features are used to make the diagnosis: abdominal distension which develops within a week, abdominal pain (80 %), vomiting (60 %), altered bowel symptoms [constipation (50 %), diarrhea (40 %)], and a radiological finding of a distended colon [14]. It is commonly seen in the sixth decade of life, predominantly in males. Although plain X-ray of the abdomen is a useful test for acute colonic pseudo-obstruction, computerized tomography of the abdomen is the gold standard [14,16]. Acute colonic pseudo-obstruction warrants urgent treatment as it can lead to colonic perforation and sepsis. Advanced age, colonic diameter more than 7 cm or cecal diameter more than 12 cm, and colonic distention over 6 days are risk factors for colonic perforation and death [15]. Treatment includes conservative management by cessation of oral feeds, insertion of nasogastric tube and flatus tube. When conservative methods fail, options include pharmacological decompression using neostigmine in patients with no evidence of bowel ischemia or perforation, colonoscopic decompression, and surgical decompression [17].

The pathophysiology of acute intestinal pseudo-obstruction in SARS-CoV-2 infection is unknown. On the other hand, neurotropic viral infections causing intestinal pseudo-obstruction are usually chronic and involve the small bowel [18–20]. It has been postulated that the coronavirus transmembrane viral spike protein binds to angiotensin-converting enzyme (ACE) receptor expressed in type-2 pneumocytes leading to respiratory failure. This is also co-expressed in the cytoplasm of epithelial cells of upper esophagus, stomach, duodenum, ileum, colon and rectum resulting in colonic distension [21]. Other hypotheses proposed are small vessel thrombosis and altered autonomic regulation of colonic motility [15,22].

Acute colonic pseudo-obstruction may be precipitated by pharmacologic adverse effects or metabolic and electrolyte derangements. Treatment includes addressing the underlying cause, nutritional support, prokinetics, and colonic decompression. Surgical decompression can be considered in patients who are non-responsive to the above measures [9].

## Conclusion

Patients with SARS-CoV-2 infection can present with acute colonic pseudo-obstruction. It is associated with high morbidity and mortality, and therefore requires a high index of suspicion as it warrants aggressive supportive management and prompt colonic decompression.

## Author contributions

Dr. Stephen Varghese Samuel had full access to all the data in the study, and takes responsibility for the integrity of the data and the accuracy of the data analysis.

**Dr. Stephen Varghese Samuel:** Data collection, conceptualization, methodology, Writing - original draft preparation.

**Dr. Surekha Viggswarpu:** Conceptualization, Methodology, Writing – Reviewing and Editing, Supervision

**Dr. Benny Paul Wilson:** Conceptualization, Writing – Reviewing and Editing, Methodology, Resources.

**Dr. Kango Gopal Gopinath:** Conceptualization, methodology, Data collection, Resources, Writing – Reviewing and Editing, Supervision

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## Ethical approval

Approval from the institutional review board was not required. Informed consent was obtained from the patients' family members.

## Consent

Written informed consent was obtained from the patients' family members for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

## Declaration of Competing Interest

The authors report no declarations of interest.

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