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Pneumomediastinum, Tension Pneumoperitoneum Secondary to Bowel Perforation in Post COVID-19 Patient: A Case Report

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Abstract

Background: We are dealing with global coronavirus SARS-COV-2 pandemic, though majority of the population remain asymptomatic during the infection or develop only mild symptomatology, most commonly reported organs to be involved are lungs, respiratory symptoms being commonest. As our knowledge is evolving, it is now believed that SARS-CoV-2 can affect any organ system including Gastrointestinal (GI) system. GI symptoms like anorexia, nausea, vomiting, and diarrhea are encountered in patients and these end up being more critical than expected in normal scenario.

Case Presentation: We report a case of 85 years old female, in post COVID-19 period, presented in emergency room with acute onset of pain abdomen and altered mental status with associated history of reduced oral intake for past 6 to 7 days. She was intubated and started on ventilator support secondary to severe respiratory acidosis and deteriorating sensorium. Cardboard rigidity was found during abdominal examination with absent bowel sounds, though family confirmed she was passing stool normally until the day before her presentation. CT chest revealed subcutaneous emphysema, pneumomediastinum and tension pneumoperitoneum with both lungs having obvious post COVID-19 pneumonia sequela. We managed her with intravenous fluid resuscitation, invasive ventilation, broad spectrum antibiotics and other supportive management. Surgery team managed her bowel perforation with bedside abdominal drain insertion in view of high risk for operation secondary to her co-morbid status. In spite of all efforts she died.

Discussion: We believed that our patient might had a complication of bowel perforation, most probably involving upper part of the gastrointestinal tract suggested by presence of subcutaneous emphysema in neck and pneumomediastinum, apart from tension pneumoperitoneum, as a post COVID-19 sequel. Apart from direct bowel wall inflammation by SARS-CoV-2, intestinal dysbiosis as well as immunological alteration in lung via gut-lung axis, local disseminated intravascular coagulation, vasculitis secondary to hypercoaguable state in COVID-19 infection, bowel edema leading to over distension of bowel all play important pathophysiology in bowel perforation, a possible fatal complication in COVID-19 patients, that physicians should be conversant of, especially in critically ill patients or with multiple comorbidity, as these patients may or may not present with gastrointestinal symptoms.

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Introduction

It is global corona virus pandemic and one with the highest number of registered cases and deaths worldwide. Current number of cases globally is approximately 86,210,710, number of deaths 1,863,497, recovered 61,188,478. Novel coronavirus disease is caused by the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) [1] which belongs to the *Coronaviridae* families of viruses, further subdivided into Coronavirinae and Torovirinae subfamilies. Coronaviruses are large, enveloped, non-segmented, single-stranded, positive-sense RNA viruses about 26 kb to 32 kb in length [2]. In India itself total number of case is more than 10,357,569, total number of case fatalities till date is 149,886 and counting.

They have mainly been identified in zoonotic hosts, that is, birds, camels, bats, masked palm civets, mice, dogs, and cats. It is believed that SARS-CoV-2 causing COVID-19 originated from a zoonotic spill over, resulting in human infections and eventual human-to-human transmission through respiratory droplets which is found to be main mode of transmission of the virus [3]. However, fecal-oral transmission due to the shedding of the virus in the Gastrointestinal (GI)

tract may continue for up to 10 weeks after respiratory clearance and is fast becoming important. SARS-CoV-2 shows a high affinity to ACE2 receptors, making sites of high ACE2 receptor expression, such as lungs, GI tract, brain, kidneys, heart, liver and immune system, a prime target for infection. Clinical symptoms of the virus vary significantly over a wide spectrum ranging from mild fever, cough, dyspnea to respiratory failure and eventual death. Current literature focuses on the respiratory symptoms and respiratory acute complications mainly [4-6].

It's baleful effects on other organ systems is still evolving and very little is known about complications occurring during the period of one to two months of post COVID-19 status, such as acute myocardial infarction, stroke, deep vein thrombosis and other thrombotic complications, gastrointestinal etc. We report a case of patient with SARS-CoV-2, treated for COVID-19 two months back and came to ER with the one of the dreaded complications of SARS-Cov-2, i.e. intestinal perforation that required emergency surgical intervention.

Case Presentation

An 85 year old female presented to our Emergency Room (ER) with complaint of acute onset of pain abdomen & altered mental status with the History of (H/O) reduced intake orally since 6 to 7 days, not passed urine since last since last 6 h, had dry cough for a week. She had passed stool one day back, which was normal in consistency and was able to pass flatus.

She had no recent H/O of fever, shortness of breath, any recent episode of vomiting or diarrhea. Although she had H/O vomiting 2 weeks back, only few episodes, persisted for a day. Her urine output was normal at home.

She was recently treated for COVID-19 pneumonia; Acute Respiratory Distress Syndrome (ARDS) for which she was hospitalized for 23 days one month back prior to ER arrival. Initially she needed non invasive ventilator support for few days and treated with antiviral (Remdesivir), Low Molecular Weight Heparin (LMWH), steroid, antibiotics and other supportive treatment. She was discharged home in stable condition.

Past history of operated brain tumor 1 year back, k/c/o hypertension, hypothyroidism. At the time of arrival patient's GCS was E3V1M4, Heart Rate (HR) 130/min, Spo_2- 86% on room air, Respiratory Rate (RR) was 44/min, Blood Pressure (BP) 100 mmHg/60 mmHg. Due to low oxygen saturation patient was put on high flow oxygen, iv fluid started. In view of low GCS, arterial blood gas was sent which showed: Respiratory acidosis with type 2 respiratory failure (pH-7.171, Po2-79.6, PCo2-138.9, HCO3-49.7, and Lactate-1.13). Patient intubated and ventilated.

After ventilation BP dropped to 80 mmHg/50 mmHg, after fluid resuscitation patient as her blood pressure was not improved she was started on inotropic support. On examination: Chest: Bilateral (B/L) equal air entry with mild crepitation. Abdomen was distended, rigid and bowel sound was absent. On per rectal examination: No impacted stool was noticed. There was no focal neurodeficit. X-ray abdomen showed large/tension pneumoperitoneum. Patient shifted to HRCT Chest in view of old COVID-19 pneumonia which showed CORADS III with gross pneumoperitoneum, mild left sided pneumothorax, pneumomediastinum, and subcutaneous emphysema noticed involving bilateral deep neck spaces and chest wall. B/L lower lobe consolidation noticed with irregular septal thickening associated

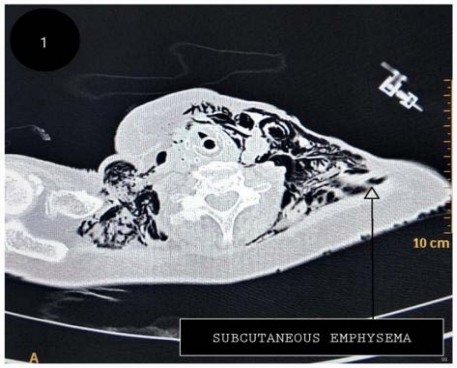

Figure 1: HRCT chest showing subcutaneous emphysema in soft tissue neck.

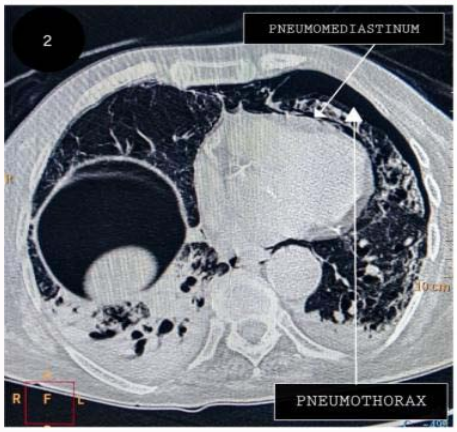

Figure 2: HRCT chest showing left sided pneumothorax, pneumomediastinum, bilateral lower lobe consolidation with irregular septal thickening associated with traction bronchiectasis in both lungs, most likely sequel of COVID-19 pneumonia with fibrosis.

with traction bronchiectasis in both lungs, more marked in lower lobes, most likely sequel of COVID-19 pneumonia with fibrosis. Left sided small pneumothorax and pneumomediastinum were managed conservatively with low PEEP in invasive ventilation. Follow up chest X-rays did not show any significant increase in both. Laboratory investigations revealed both rapid antigen for COVID-19 and RT-PCR for COVID-19 nasopharyngeal swab both were negative on this admission, Hemoglobin- 10.7 gm/dl, total WBC-12,000/cumm, platelet Count-3,05,000/cumm, procalcitonin-0.335, ESR30, serum sodium-143.3 meq/Lt , serum potassium-5.32 meq/Lt, creatinine 0.84 mg/dl.

Patient was diagnosed with intestinal perforation with huge/ tension pneumoperitoneum and peritonitis in septic shock and Acute Renal Failure (AKI). General surgery team was involved and considering the age and co-morbid status of the patient bedside intra-abdominal drain was inserted as patient family was unwilling for high risk aggressive surgical laparotomy. In spite of all effort patient deteriorated and died on day 4 of admission. We believed that our patient might had a complication of bowel perforation, most probably involving upper part of the gastrointestinal tract suggested by presence of subcutaneous emphysema in neck and pneumomediastinum, apart from tension pneumoperitoneum, as a post COVID-19 sequel.

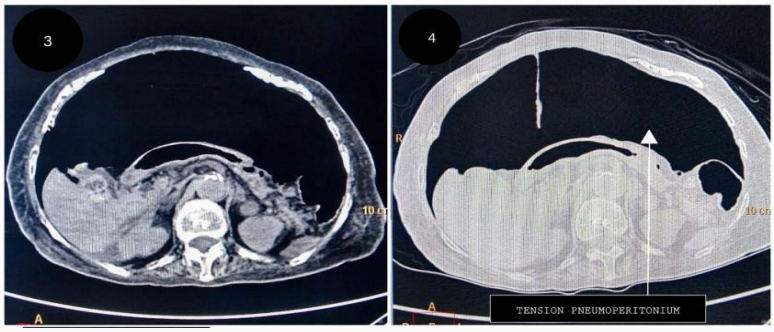

Figure 3 and 4: HRCT chest showing tension pneumoperitonium probably secondary to bowel perforation.

Discussion

Besides common symptoms like cough and cold, shortness of breath, fever etc. some COVID-19 patients seem to experience gastrointestinal symptoms such as diarrhea, nausea, vomiting, and anorexia. Digestive symptoms also found to be associated with COVID-19 disease severity. Among the overall population, diarrhea, nausea, vomiting, abdominal pain, and anorexia appeared in patients admitted into the Intensive Care Unit (ICU) more prominently than in those not transferred into the ICU [7,8].

Pathophysiology

Virus structure: SARS-CoV-2 is single-stranded RNA viruses characterized by two groups of proteins, structural proteins and nonstructural proteins. Structural proteins are Spike (S) proteins marking all coronaviruses and bind to receptors on the host cell, Nucleocapsid (N) that protects the genetic information of virus, Matrix (M), and Envelope (E). Non-structural Proteins encoded by Replicase Complex, such as proteases (nsp3 and nsp5) and RdRp. This enzyme is an ideal target as it helps the virus to replicate. Each monomer of S protein contains two subunits: S1 and S2, which mediate attachment and membrane fusion ***via*** endocytosis. Priming and cleavage of CoV (S) proteins by one of several host proteases including furin, trypsin, cathepsins, Transmembrane Protease Serine Protease-2 & 4 (TMPRSS-2 & 4) are essential for viral entry into the cell [9-11].

Angiotensin converting enzyme-2 receptors (ACE-2): Virus entry to the host cell requires recognition of ACE2 receptor by S1 subunit of viral S protein followed by anchoring on viral membrane. S2 sub unit anchors the protein to the viral membrane and facilitates viral fusion. After completion of endocytosis, virus takes over the cellular machinery to produce viral proteins and viral RNA, which then assembles intra cellularly followed by secretion, release of cytokines then lead to various symptoms [12,13].

High ACE2 receptor expression is observed in ciliated, goblet and surfactant-producing type 2 alveolar cells, thereby providing a portal of entry of the virus into the body. High ACE2 expression is also observed on the intestinal epithelium (gastric, duodenal, and rectal epithelia) and cardiac and vascular endothelium, offering an explanation to the complication of COVID-19 in these organ systems [14,15]. It has now been established that SARS-CoV-2 can still be shed through the GI tract even after the resolution of pulmonary symptoms, may continue up to 10 weeks after initial symptoms, making fecal-oral route an important mechanism of transmission of the virus [16].

Intestinal and lung microbiome in COVID 19: The diverse microbiota colonizes the human GI tract plays pivotal role in nutritional metabolism, development and maturation of immune system, and antibacterial effects. This can be altered during SARS-COV-2 infection leading to dysbiosis. Compared to healthy controls, COVID-19 patients had significantly reduced bacterial diversity, a significantly higher relative abundance of opportunistic pathogens (*Streptococcus*, *Rothia*, *Veillonella* and *Actinomyces*), and a lower relative abundance of beneficial symbionts [17].

Emerging experimental and epidemiological evidences indicated the crucial immunological relationship between the intestinal microbiota and the lungs which was termed 'gut-lung axis'. The SARS-CoV-2 virus itself may cause disorders of the intestinal flora, which could result in digestive symptoms, could further influence the respiratory tract through the common mucosal immune system with the largest being the intestinal immune system [18,19].

Hypercoaguable state in COVID-19: Studies have stated that critically-ill patients with COVID-19 are more likely to have a hypercoagulable state. The coronavirus has an extensive tissue distribution, causing the release of a high number of pro-inflammatory cytokines that damage the microvascular system, promoting abnormal activation of the coagulation system, which manifests itself as generalized small-vessel vasculitis and extensive microthrombi causing intravascular coagulation locally that may lead to intestinal mucosal necrosis, edema [20,21]. Gartland et al. [21] described catastrophic bowel necrosis in the setting of patent mesenteric vessels in 47 year COVID-19 positive patient.

Over distension of Bowel: DeNardi et al. reported a case of patient with colonic perforation presumed to be secondary to over-distension of bowel. Another hypothesis was an altered colonic motility due to an imbalance of the autonomic innervation of the colon. Since the neuroinvasive propensity has been demonstrated as a common feature of coronaviruses, a neuronal injury should be regarded as a possible pathogenic mechanism [22].

Conclusion

So far very little is known regarding surgical emergencies related to post COVID-19 infection. Intestinal perforation being reported only in few case reports only, by Aron Kangas-Dick et al., Vishakha Kalikar et al., De Nardi et al. Sergio Carlos NAHAS et. al. Apart from direct bowel wall inflammation by SARS-CoV-2, intestinal dysbiosis as well as immunological alteration in lung *via* gut-lung axis, local disseminated intravascular coagulation, vasculitis secondary to hypercoaguable state in COVID-19 infection, bowel edema leading to

over distension of bowl all play important pathophysiology in bowel perforation, a possible fatal complication in COVID-19 patients, that physicians should be conversant of, especially in critically ill patients or with multiple comorbidities, as these patients may or may not present with gastrointestinal symptoms. Additionally in India, use of high dose of corticosteroids in treatment of COVID-19 pneumonia is another important factor of consideration in bowel perforation. In our case report though the patient unfortunately died from tension pneumoperitoneum, we believe early recognition could still play an important role in patient's outcome. Patient's age, gender, comorbidities, critically ill status are still to be evinced of having any correlation with bowel perforation as post COVID-19 complication.

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