



Acute Kidney Injury and Acute Pancreatitis in Patients with Chronic Hypertension and COVID-19: A Case Report

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Abstract

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BACKGROUND: Hypertensive patients with Coronavirus Disease 2019 (COVID-19) are facing high morbidity and mortality. These morbidities include acute kidney injury (AKI) and acute pancreatitis, which have an incidence of about 17% each. Severe acute respiratory syndrome-corona virus 2 (SARS-CoV2) virus penetrates cells through the angiotensin-converting enzyme 2 (ACE2) receptor which is widely found in the respiratory tract, gastrointestinal tract, kidneys, pancreas, and other organs. The virus multiplies in the airway and then enters circulation bound to ACE2 receptors in the tissue.

CASE REPORT: This case report presents a chronic hypertensive patient with COVID-19 accompanied by complications of AKI and acute pancreatitis.

CONCLUSION: AKI is a risk factor for death in COVID-19 patients, where kidney involvement in COVID-19 is thought to be due to direct infection with SARS-CoV2 or through other complicating conditions, where acute pancreatitis occurs due to COVID-19.

Background

Coronavirus disease 2019 (COVID-19) is caused by infection of severe acute respiratory syndrome-coronavirus 2 (SARS-CoV2), which has prompted a pandemic with high morbidity and mortality [1]. The SARS-CoV2 virus, the transmission of which is airborne, enters the alveoli cells through the angiotensin-converting enzyme 2 (ACE2) receptor which is widely found in the respiratory tract [2]. ACE2 receptors are also found in the eyes, gastrointestinal tract, kidneys, liver, heart, pancreas, vascular, and the central nervous system [1], [2].

Hypertension is the most common comorbidity found in COVID-19 in addition to others such as diabetes, obesity, and ischemic heart disease. Various studies have reported that COVID-19 in hypertensive patients has a poor prognosis [3].

Complications in organs due to infection by the SARS-CoV2 virus include AKI and acute pancreatitis [2], [4]. The reported incidence of AKI in patients with COVID-19 varies widely, ranging between 3 and 17% [5], [6] Wang *et al.* [7] reported that the incidence of pancreatic injury among 52 patients (17%). The following report presents a case of a chronic

hypertensive patient with COVID-19 accompanied by complications of AKI and acute pancreatitis.

Case Report

A male, 27 years old, came to the emergency room with a chief complaint of abdominal pain he had been experiencing for the past 3 days, accompanied with malaise and fever. The volume of urine had decreased and its color was dark yellow. There was a history of contact with COVID-19 patients and hypertension, but the patient was not treated. There was no history of gallstones or any other bile duct disease or history of alcohol consumption.

On physical examination, the blood pressure was 230/110 mmHg, pulse 110 beats/min, respiratory rate 26 times/min, axillary temperature 38°C, with visual analog scale 6/10, and body mass index 19.1 kg/m². There was tenderness in the epigastric region; other physical examinations were within normal limits.

The laboratory results showed leukocytes 12,300/ul, urea 185 mg/dl and creatinine 15 mg/

dl, potassium 7 mmol/l, and albumin 3.0 mg/dl. The thoracic computed tomography (CT): Left pulmonary fibrosis (Figure 1) and abdominal CT: Features of acute pancreatitis (Figure 2). From examination, the amylase and lipase enzymes were 125 U/l and 115 U/l. The patient was diagnosed with hypertensive emergencies, AKI, hyperkalemia, and acute pancreatitis. He was given intravenous anti-hypertensive therapy (Nicardipine 60 mcg/jam), hyperkalemia therapy (Calcium polystyrene sulfonate 5 g/12 h), and anti-biotics (Ceftazidime 1 g/12 h). Hemodialysis (HD) was also performed.



Figure 1: Non-contrast Thoracic CT, yellow arrow showing pulmonary fibrosis

The patient was screened using the COVID-19 Early Warning Score (EWS) with a score of 10, and a Real Time-Polymerase Chain Reaction (RT-PCR) from nasopharyngeal swabs was carried out with positive results. Based on these results, the diagnosis of COVID-19 was also established; so, it is thought that COVID-19 is the cause of acute pancreatitis and AKI.



Figure 2: CT Abdomen non-contrast, red arrow showing the appearance of acute pancreatitis

On the 3rd day while under observation, the patient's experienced worsening with an increase in the respiratory rate of 28 times/min and a decrease in oxygen saturation below 80% with $\text{PaO}_2/\text{FiO}_2 < 200$, thereby requiring mechanical ventilation assistance. Treatment interventions carried out within a few days resulted in improvement in clinical and laboratory terms.

Worsening reoccurred on the 18th day of treatment there were shortness of breath and desaturation despite assistance by mechanical ventilation, ventilatory dependent respiratory failure, and even though the laboratory showed the serum creatinine reaching 18.6 mg/dl (eGFR 3 ml/min/1.73m²), urea 280 mg/dl, potassium 7.7 mmol/l, amylase and lipase 781 U/l and 1105.8 U/l, D-dimer 10.8 mg/dl, and CRP 76.7 mg/l. This worsening of the patient's condition was thought to be due to the presence of a cytokine storm, supported by the examination of IL-6 levels reaching 508 pg/ml (N: <7 pg/ml) and a marker of worsening sepsis where the procalcitonin value reached >200 mg/dl. The patient was given anti-IL6 (Tocilizumab) 8 mg/kg body weight single-dose and Dexamethasone 6mg/24 hours. On the 23rd day of treatment, there was an improvement in the patient's clinical condition.

Hypertension is reported to be a risk factor for COVID-19 and its progression is more severe with higher morbidity and mortality [8]. The SARS-CoV2 virus enters alveoli cells through ACE2 receptors [2]. In hypertensive patients, the activation of the Angiotensin II – Angiotensin II type 1 receptor (AngII-AT1) receptor pathway is more dominant than the Ang (1–7) -Mas receptor [8]. The binding of ACE2 with the SARS-CoV2 virus causes downregulation of ACE2, so that the formation of Ang (1–7) lessens [9]. The effect of AngII is more dominant and further increases inflammation and fibrosis which, in turn, is more damaging to lung tissue [8], [9].

Acute Kidney Injury in this patient could be caused by direct infection of the SARS-CoV2 virus in the kidney cells or hemodynamic disturbances triggered by severe acute pancreatitis [5], [6]. This patient had severe hypertension which was not treated. In patients with chronic hypertension, especially when uncontrolled, there will be intra-renal hemodynamic disturbances which will end in glomerulosclerosis [10]. This condition will make the kidneys susceptible to systemic changes that will cause acute impairment of kidney function [6], [10].

In this patient, acute pancreatitis was thought to be associated with SARS-CoV2 virus infection. This is because the patient did not have a history of alcohol consumption or bile duct obstruction as evidenced by abdominal Computerized Tomography (CT) results that did not show bile duct obstruction where both these conditions were the main risk factors for acute pancreatitis. Systemic inflammation as reflected by the increase in pro-inflammatory mediators such as Tumor Necrosis Factor-alpha (TNF-a) and interleukin 6 (IL-6) in severe COVID-19 patients will worsen kidney and pancreatic injury [2], [7], [11].

Acute Kidney Injury is indicated for initiation of renal replacement therapy (RRT) when there are conditions such as uremia, electrolyte balance disorders such as hyperkalemia, metabolic acidosis, hypervolemia, and others [5]. The patient had uremia and hyperkalemia that did not improve with conservative therapy. A report shows that about 1.5–9% of COVID-19 patients who

experience AKI need RRT [5], [6]. Continuous RRT (CRRT) is recommended by the American Society of Nephrology (ASN) in COVID-19 patients with severe AKI [10]. In this case, we use the IHD modality because the machine availability is still limited.

The patient's condition after undergoing HD during the first 2 weeks of treatment has improved from a clinical and laboratory perspective. However, the patient's condition began to worsen again in the 3rd week of treatment; severe ventilator dependent respiratory failure, coagulopathy, increased pancreatic enzymes, and deteriorated kidney function occurred. This condition is thought to be due to the occurrence of cytokine storms, as indicated in the laboratory by increasing levels of CRP, procalcitonin, and IL-6 [11]. Cytokine storms are directly proportional to organ damage and mortality rates [2].

Conclusion

In our case report was reported that a man, 27 years old, with chronic hypertension and COVID-19 had complications of acute kidney injury (AKI) and severe acute pancreatitis. AKI is a risk factor for death in COVID-19 patients, where kidney involvement in COVID-19 is thought to be due to direct infection with SARS-CoV2 or through other complicating conditions, where acute pancreatitis occurs due to COVID-19. Renal replacement therapy in the form of HD was performed.

Statement of Ethics

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Data Availability

The authors confirm that the data supporting the findings of this case report are available within the article.

Authors' Contribution

Achmad Fikry as corresponding author, collected data, analyzed data, and writing; Haerani Rasyid collected data, analyzed data, and writing.

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