CASE REPORT

Intisari Sains Medis 2023, Volume 14, Number 1: 270-274
P-ISSN: 2503-3638, E-ISSN: 2089-9084

Published by Intisari Sains Medis

Acute kidney injury in a covid-19 patient with Non-ST elevation myocardial infarction: case report

Indry Agatha Rihi Pake[1], Putu Eka Dianti Putri[2*], Agung Pradnyana Suwirya[3,4], Desak Nyoman Desy Lestari[5]

[1]Faculty of Medicine, Universitas Tarumanagara, Indonesia;
[2]Faculty of Medicine, Universitas Hang Tuah, Indonesia;
[3]Departement of Cardiology and Vascular Medicine, Udayana Army Hospital, Denpasar, Indonesia;
[4]Departement of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Udayana-Prof. IGNG Ngoerah Hospital, Denpasar, Bali, Indonesia;
[5]Departement of Internal Medicine, Udayana Army Hospital Denpasar, Indonesia;

*Corresponding author:
Putu Eka Dianti Putri;
Faculty of Medicine, Universitas Hang Tuah, Indonesia;
ekadiantip@gmail.com

Received: 2023-01-29
Accepted: 2023-02-28
Published: 2023-03-31

ABSTRACT

Introduction: Coronavirus disease 2019 (COVID-19) has a variety of manifestations, from asymptomatic to acute illness that can affect several organs, although being primarily associated with the respiratory system. The virus binds to angiotensin-converting enzyme-2 (ACE-2) receptors in humans and the expression of ACE-2 in heart and kidneys explains the association of COVID-19 with the renal and cardiovascular systems.

Case description: Male, 62-year-old, was admitted to the emergency room (ER) for presented shortness of breath, angina pain, and gastric pain. He was a smoker and denied having comorbidities. At arrival, an examination of vital signs and physical showed tachypnea, tachycardia, hypertension, and bilateral crackles. The electrocardiography (ECG) showed left ventricular hypertrophy and Non-ST-Elevation in anterolateral. The chest x-ray showed pneumonia and cardiomegaly. Laboratory findings showed that decreased renal function and Polymerase Chain Reaction (PCR) test was positive, and increased in serum Troponin level supported the diagnosis of Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-Cov-2) pneumonia with Non-ST elevation myocardial infarct (NSTEMI) and Acute Kidney Injury (AKI). Loading of Aspilet, Clopidogrel, Atorvastatin, and Thrombolysis with Fondaparinux was done. The following day, transthoracic echocardiography was performed anterolateral regional wall motion abnormality with 43% of Left Ventricle Ejection Fraction (LVEF). Two weeks after hospital discharge, the echocardiography evaluation showed no improvement and was diagnosed with Chronic Heart Failure with New York Heart Association (NYHA) Functional Classification II et causa Coronary Artery Disease.

Conclusion: Treatment of covid-19 patients with cardiovascular and renal complications is quite challenging since there is no specific guideline for COVID-19 patients with renal and cardiovascular complications.

Keywords: SARS-Cov-2 Pneumonia, Acute Coronary Syndrome, Non-ST-Elevation Myocard Infarct, Kidney Failure.
Cite This Article: Pake, I.A.R., Putri, P.E.D., Suwirya, A.P., Lestari, D.N.D. 2023. Acute kidney injury in a covid-19 patient with Non-ST elevation myocardial infarction: case report. *Intisari Sains Medis* 14(1): 270-274. DOI: 10.15562/ism.v14i1.1620

INTRODUCTION

Currently, COVID-19 has spread around the world. The first report of COVID-19 was identified during the respiratory illness outbreak in Wuhan, China in December 2019, and has been responsible for substantial morbidity and mortality. The virus quickly evolves and spreads to other countries as a worldwide threat. Rapidly, WHO declared COVID-19 as a worldwide pandemic on March 11th, 2020.[1] COVID-19 has a variety of manifestations, ranging from asymptomatic disease to acute illness that can affect several organs including the heart, kidneys, liver, and brain, although being primarily associated with the respiratory system.

Patients with comorbidities such as cardiovascular are major risk factors for severity and mortality. According to a recent epidemiological study, coronavirus can result in acute myocardial damage in 7.2%, arrhythmia in 16.7%, and shock in 8.7% of cases.[2] Renal involvement is also frequent in the course of COVID-19. Patients with renal impairment, defined as an increase in baseline serum creatinine, an increase in baseline blood urea nitrogen, proteinuria and hematuria, and acute kidney damage, had a substantially higher death risk, according to Kaplan-Meier analysis.[3] Incidence and severity of AKI occurred in 1835 patients (46%), and 347 (19%) of patients with a diagnosis of AKI requiring dialysis in New York.[4]

A spectrum of related disorders between the cardiovascular and renal systems where acute and chronic disorders of one organ system can cause dysfunction in other organs. SARS-Cov-2 binds to ACE-2 in humans as an entry point for human pneumocytes.[5] The expression of ACE-2 in the heart and kidneys explains the association of COVID-19 with the renal and cardiovascular systems.[6] Special pharmacological considerations are necessary for the treatment of COVID-19 individuals with cardiovascular and

renal associations. But, there is limited data about COVID-19 patients who also suffered from Acute Coronary Syndrome (ACS) and AKI, especially with the limitation of facilities.

CASE DESCRIPTION

We present the case of a 62-year-old male patient, admitted to the hospital for having shortness of breath for 3 days, and was progressive 1 day before admission. Shortness of breath is felt during activity or at rest. He also had 3 day-history of a dry cough, runny nose, weakness, and a day before admission, he vomited 1 time associated with nonspecific abdominal pain. He also complained of chest pain 1 day before admission, the pain spread to the back, left arm, and epigastrium, and did not subside with rest. He denied complaints of fever**.** He was a smoker and had no history of cardiovascular disease, diabetes mellitus, hypertension, or any kidney disease.

On the examination, his heart rate was 104 beats per minute (pm), respiratory rate 28 times pm, O2 Saturation was 95%, the temperature was 36.7°C, and blood pressure was 150/100 mmHg. Clinical examination revealed the patient had symmetric bilateral chest retraction and bilateral crackles in both lungs. There was no gallop, murmur, or pedal edema at the examination. Besides the clinical examination, the patient's initial laboratory investigations revealed his random blood glucose was 167 mg/dL and decreased renal function with blood ureum at 89 mg/dL, and creatinine at 2.2 mg/dL. The chest x-ray showed parahilar and paracardiac consolidation at the right lung, and the heart was cardiomegaly (Figure 1). The ECG showed left ventricular hypertrophy and T inversion in lead I, aVL, and V4-V6 (Figure 2), and the cardiac troponin I was 0.15 ng/ml, slightly above the normal limit indicating anteroseptal ischemia of myocardium.

Based on these findings, he was diagnosed with community-acquired pneumonia with NSTEMI. The initial treatment given to the patient was supportive and symptomatic. At the ER, he was initially treated with oxygen 3 lpm, parenteral nutrition, anti-vomiting and Proton Pump inhibitor drug, isosorbide dinitrate 5 mg sublingual, loading clopidogrel 300 mg, aspirin 160 mg, furosemide bolus intravenous 60 mg, subcutaneous fondaparinux 2.5 mg/0.5ml, ramipril 2.5 mg, atorvastatin 40 mg, combination of ipratropium bromide and salbutamol sulfate nebulizer 2,5 ml and acetylcysteine 200mg for his shortness of breath. Because of the suspicion of bacterial superinfection, the patient was given an intravenous antibiotic, cefoperazone 2 grams. PCR test was performed on a nasopharyngeal swab, and returned positive for SARS-Cov-2. And he had antiviral therapy favipiravir 1600 mg 2 times a day on 1st day, and continued with favipiravir 600 mg 2 times a day from 2nd until 5th day and Hydrocortisone 100 mg iv for 5 days.

During his admission, an additional blood test showed leukocytosis with White blood cells count slightly above the normal limit, 11.55 x 10^3 uL, and the differential count shifted to the left. He also developed acute liver injuries as his Alanine transaminase (ALT) level was 65 U/L.

After the treatments, patient showed significant improvement after 5 days. One day before he was discharged, the laboratory investigation showed an improvement in the complete blood count and kidney function. He also had complete recovery of kidney function, he was no longer oliguric with creatinine level at 1.1 mg/dL and blood ureum at 34 mg/dL. After 7 days of admission, he was discharged home in good condition.

The following day, transthoracic echocardiography was performed and revealed left ventricular ejection fraction was 43 % with eccentric left ventricular hypertrophy and diastolic dysfunction grade II. There was normal right ventricular systolic function with tricuspid annular

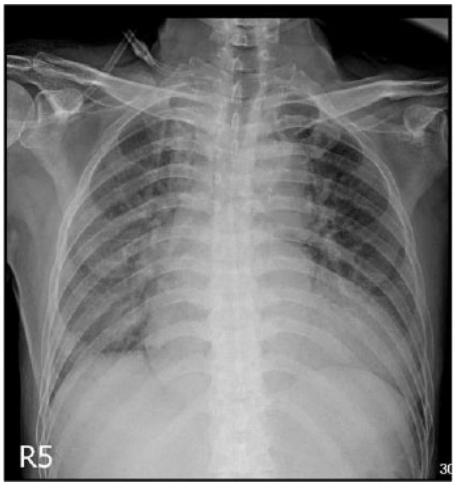

Figure 1. Chest X-Ray: Parahilar and paracardiac consolidation at the right lung, and cardiomegaly.

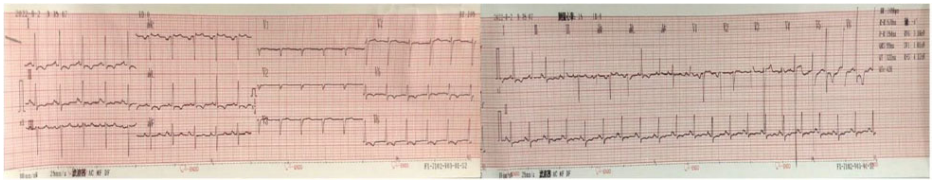

Figure 2. ECG: left ventricular hypertrophy and T inversion in lead I, AVL, and V4-V6.

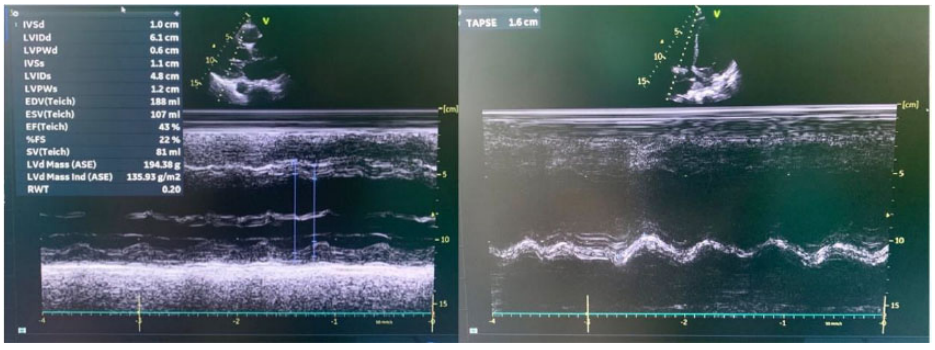

Figure 3. Transthoracic Echocardiography: LVEF 43 % with eccentric left ventricular hypertrophy and diastolic dysfunction grade II. And normal right ventricular systolic function with TAPSE 16mm.

plane systolic excursion (TAPSE) 16mm, moderate to severe regurgitation of mitral and tricuspid valve, with estimated right atrial pressure (eRAP) 8 mmHg (Figure 3). And 2 weeks after his discharge follow-up Echocardiography was performed for evaluation and shows no improvement and diagnosed with Congestive Heart Failure with NYHA Functional Classification 2.

DISCUSSION

The respiratory system is the primary organ affected by COVID-19 infection, however, multiple organ involvement also has been reported, including the heart, kidney, lives, and gastrointestinal tract.[3] Cardiorenal syndrome in Covid-19 patients requires certain pharmacological considerations of treatment. Fluid therapy, anti-viral therapy, renal replacement therapy (RRT), percutaneous coronary intervention (PCI), and anticoagulant prophylaxis/treatment need special attention in COVID-19 patients with Cardiorenal syndrome.

In a meta-analysis and systemic review, acute cardiac injury was the most often reported cardiac complication of COVID-19 disease, occurring with a frequency of 25.3%.[7] In the acute phase of COVID-19, there have been reports of an association between the disease and the development of cardiovascular problems, but the mechanism is still not entirely unclear. Activation of the immune-inflammatory-procoagulant cascade, direct invasion by the virus, indirect damage due to the systemic inflammatory syndrome and cytokine storm, dysregulation of renin-angiotensin-aldosterone system, hypoxia-induced cardiac injury, microvasculature damage of the heart, stress-induced cardiomyopathy, and cardiac damage secondary to multi-organ failure is suspected to be the mechanism underlying the occurrence of cardiovascular complications in patients with COVID-19.[7]

ACE-2 receptors are extensively expressed in endotheliocytes and myocardial cells, which allows SARS-CoV-2 to infect the cardiovascular system and cause endothelial dysfunction, arterial or cardiomyocyte inflammation, plaque rupture, and transient heart attacks.[8] The virus attaches to the ACE-2 protein and invades host endothelium cells. Infected cells showed disruption of intracellular connections, cellular swelling, and loss of basal membrane integrity upon histological examination of lung tissue from COVID-19 patients. Additionally, cytokine storm is a significant factor. Tumor necrosis factor-alpha, interleukin-1, and interleukin-6 are released because of this microvasculature damage, which enhances tissue factor expression on endothelial cells and macrophages. High levels of these pro-inflammatory cytokines in Covid-19 patient's serum triggered the coagulation cascade and resulted in microvascular thrombosis, which makes covid-19 patients more susceptible to coronary plaque rupture, thrombogenesis, and acute stent thrombosis.[8,9]

Numerous biomarkers have been reported in COVID-19 individuals, for example, decreased number of fibrinogen and an increased number in D-dimer, which become markers of systemic hypercoagulable condition, these markers might be indicators the patients need anticoagulation therapy that can improve the prognosis of COVID-19 patients with cardiovascular complications. However, due to limited laboratory facilities, these 2 indicators could not be examined in the patient in this case. Additionally, the diagnosis of type 1 NSTEMI (plaque rupture) is challenging since a significant number of COVID-19 patients have increased troponin level, and regardless of whether COVID-19 patients have ischemic cardiac damage or not, the majority of experts believe that there is a tight correlation between the increase of troponin and the worse prognosis in these patients. A troponin rise in COVID-19 patients' myocardial damage is largely correlated with severe hypoxia, sepsis, and systemic inflammation, pulmonary thrombosis embolism, and also probably related to stress cardiomyopathy, myocarditis.[8]

According to a study that included 31 patients COVID-19 with suspected ACS, urgent PCI is frequently needed for ACS patients with SARS-CoV-2 infection to improve the prognosis in all but the most advanced patients.[10] Recently, patients with acute cardiac injury had a much higher risk of being admitted to the ICU.[7] All individuals should be treated medically with a full dosage of aspirin, a high-intensity statin, parental anticoagulation (heparin or low molecular weight heparin-LMWH), beta-blocker if hemodynamically stable, and nitrate treatment if they have ongoing chest pain. Patients with a global registry acute coronary event (GRACE) score above 140 or those with high-risk clinical characteristics such as refractory chest pain, unstable arrhythmias, heart failure, or hemodynamic instability may consider an invasive strategy.[11] In the present case, our patient had 120 for the GRACE score, and double anti-platetel therapy (DAPT) with clopidogrel 300 mg, aspirin 160 mg and anticoagulant with 2.5 mg of Fondaparinux was chosen for the therapy of his NSTEMI. He also had nitrates sublingual to reduce his chest pain.

In this case, our patient also developed heart failure after his admission, the development of heart failure in COVID-19 patients is thought to involve two different and overlapping pathways. The first is thought to be cytokine release causing myocardial inflammation, and the second is thought to be a direct viral infection that causes myocarditis.[12]

The cause of AKI can be classified into three broad groups: (1) pre-renal or hemodynamic (i.e., hypoperfusion to the kidney), (2) intrinsic (i.e., structural damage to the kidney), and (3) post-renal (i.e., obstruction of urinary outflow). It has been reported that the kidney is one of the organs that most expresses ACE-2 and that the renal cells with the highest expression are the proximal tubular cells and, to extent, the podocytes.[13,14] The pathophysiology of developed AKI in COVID-19 is thought to be multifactorial and involves cardiovascular comorbidity, direct effects of the virus on the kidney, local, and systemic inflammatory and immune responses, endothelial injury, and activation of coagulation pathways and the renin-angiotensin system.[13,15] COVID pneumonia can cause right ventricular failure and lead to renal congestion, while left ventricular dysfunction can lead to hypotension, decreased cardiac output, and hypoperfusion of the kidney. Severe COVID-19 can cause skeletal muscle damage leading to myoglobin release,

which induces kidney damage through pigment cast formation, causing tubular obstruction and tubular toxicity related to iron release.[15] According to Kidney Disease Improving Global Outcomes (KDIGO), AKI is defined as any of the following: (1) an increase in serum creatinine (SCr) by ≥0.3 mg/dL (≥26.5 μmol/L) within 48 h; or (2) an increase in SCr ≥ 1.5 times of baseline within the prior 7 days; or (3) urine volume < 0.5 mL/kg/hour for 6 h. AKI can be also staged for severity according to KDIGO: stage (1) increase in SCr to 1.5–1.9 times baseline or by ≥0.3 mg/dL; stage (2) increase SCr to 2.0–2.9 times baseline; stage (3) increase SCr to 3.0 times baseline or increase in serum creatinine to ≥4.0 mg/dL (≥353.6 mmol/L) or the initiation of renal replacement therapy. According to several case studies, the incidences of AKI in COVID-19 patients varied. In China, the studies showed AKI occurred in 5% to 29% of cases within a median of 7 to 14 days after admission, although reports from the United States showed higher rates, ranging from 37% to 57% in COVID-19-positive patients.[16] In our case, the most probable cause of AKI would be pre-renal and intrinsic factors which associated with angiotensin-converting enzyme to dependent pathway and cause mitochondrial dysfunction and acute tubular necrosis. COVID-19 related immune response dysregulation also generates a cytokine storm and the development of microemboli microthrombi in the context of hypercoagulability.[15]

The same mechanism like cardiovascular complications, sepsis, cytokine storm, and direct cellular damage, all play a role in kidney injury in COVID-19 patients.[17] The proximal tubular epithelial cells and podocytes in the kidneys have the greatest levels of ACE-2 in the kidney and become the entry site of SARS-CoV-2. Therefore, the most plausible mechanism for AKI development is the direct infection of kidney cells by the SARS-CoV-2 virus. Other possible mechanisms are abnormal immune responses associated with SARS-CoV-2 known as a cytokine storm. Interleukin-6 is the most crucial cytokine contributing to COVID-19 ARDS complications. AKI in cytokine storm conditions is thought to be caused by intrarenal inflammation, increased vascular permeability, volume depletion, and cardiomyopathy. Cardiomyopathy can cause stasis in the renal veins, resulting in renal hypotension and hypoperfusion, leading to a reduction in the glomerular filtration rate.[16]

There is no specific treatment for AKI caused by COVID-19. Fluid treatments (balanced crystalloids), reducing the use of nephrotoxic medications, and, maintaining blood pressure with the use of vasopressors in the case of hypovolemia, and good oxygenation are needed to prevent AKI progression. However, for COVID-19 patients with hemodynamically unstable, volume overload, especially those with persistent hypoxemia, should be considered for renal treatment goals, including supportive therapy and RRT if necessary.[16,18] Fluid therapy is the traditional cornerstone of AKI prevention and treatment, but patients with AKI are also prone to develop fluid overload. This case may present classical findings associated with volume overloads including dyspnea in the ER, pulmonary rales, and the chest x-ray showing cardiomegaly therefore we choose to do a restriction fluid and add diuretics. In the DOSE-AHF (Diuretic Optimization Strategy Evaluation in Acute Decompensated Heart Failure) study, the authors found that patients with acute heart failure may benefit from an initial bolus strategy.[19]

Drug treatment for COVID-19 patients also slows the progression of COVID-19 and lowers the risk of developing AKI.[16] In individuals with renal disease, kidney elimination of certain drugs or their metabolites may raise the risk of toxicity of kidney. Therefore, some medications may require dose modifications depending on the level of kidney dysfunction and their potential renal adverse effect. Corticosteroids reduce proinflammatory cytokines and suppress the "cytokine storm" that causes clinical morbidity and mortality. Corticosteroids were strongly advised for severe or critical COVID-19 patients and preferably be started seven days after the onset of symptoms. And no dose adjustment is required for patients with renal complications.[20]

Some antiviral therapy is also needed for COVID-19 patients. Some investigational agents including remdesivir were being tested for antiviral treatment of COVID-19.[21] Remdesivir therapy for up to 5 days, which can be prolonged for up to 10 days, is an option for patients with moderate or severe COVID-19. If remdesivir is unavailable, the administration of antivirals can be adjusted to medications that are accessible in healthcare centers, such as favipiravir (200 mg), which is given in loading doses of 1600 mg every 12 hours orally on the first day and 600 mg twice a day after that (2nd to 5th day).[22] Favipiravir is RNA-dependent RNA polymerase (RdRp) inhibitor that is currently under investigation for SARS-CoV-2 antiviral therapy. According to several meta-analyses, favipiravir has some advantages over other antivirals. This drug has a wide safety margin and can improve the symptoms earlier than other antivirals. Renal side effects of favipiravir have not yet been reported. No clear data are provided about dosage modification for patients with renal impairment. But, because it's primarily excreted by kidney, favipiravir is not advised to be used in individuals with estimated glomerular filtration rate (eGFR) <30 ml/min.[20,23]

For our case, cefoperazone was also given because the laboratory examination showed leukocytosis, as an empirical antibiotic for secondary bacterial infection in COVID-19 patients. As there is no evidence for a specific superior empirical treatment strategy in patients with COVID-19 and bacterial pneumonia, we recommend to follow local and/or national guidelines for the antibacterial treatment of pneumonia. As an example, in the Dutch Working Party on Antibiotic Policy guideline on community-acquired pneumonia (CAP), preferred regimens depend on the severity of the disease: for mildly and moderately severe CAP amoxicillin is recommended, for patients with severe CAP at the general ward a second or third-generation cephalosporin is recommended.[24,25]

CONCLUSION

In conclusion, we report a case of an Indonesian male patient diagnosed with AKI, COVID-19, and NSTEMI. The disease courses and treatment

options for the patient were significantly quite challenging. Patients at risk for AKI and those with AKI should have kidney function monitored closely by SCr concentration and urine output. Careful assessment of volume status and hemodynamics should be undertaken and treated with intravenous fluids, diuretics, or others means of hemodynamic support as indicated.

DISCLOSURE

Funding
None.

Conflict of Interest
The author reports no conflicts of interest in this work.

Author Contribution
Indry Agatha Rihi Pake involved in concepts, clinical studies, literature search, data acquisition, and manuscript preparation. Putu Eka Dianti Putri involved in design, literature search, data acquisition, manuscript preparation, and manuscript editing. Agung Pradnyana Suwirya involved in concepts, definition of intellectual content, manuscript review, and guarantor. Desak Nyoman Desy Lestari involved in design, definition of intellectual content, manuscript review, and guarantor.

Acknowledgments
None.

Patient Consent
Patient had received signed written informed consent regarding publication of medical data in jorunal article with confidentiality to personal information.

REFERENCES

1. Liu YC, Kuo RL, Shih SR. COVID-19: The first documented coronavirus pandemic in history. Biomed J. 2020;43(4):328-333. doi:10.1016/j.bj.2020.04.007
2. Wang D, Hu B, Hu C, et al. Clinical Characteristics of 138 Hospitalized Patients With 2019 Novel Coronavirus–Infected Pneumonia in Wuhan, China. JAMA. 2020;323(11):1061-1069. doi:10.1001/jama.2020.1585
3. Cheng Y, Luo R, Wang K, et al. Kidney disease is associated with in-hospital death of patients with COVID-19. Kidney Int. 2020;97(5):829-838. doi:10.1016/j.kint.2020.03.005
4. Chan L, Chaudhary K, Saha A, et al. AKI in Hospitalized Patients with COVID-19. J Am Soc Nephrol. 2021;32(1):151-160. doi:10.1681/ASN.2020050615
5. Beyerstedt S, Casaro EB, Rangel ÉB. COVID-19: angiotensin-converting enzyme 2 (ACE2) expression and tissue susceptibility to SARS-CoV-2 infection. Eur J Clin Microbiol Infect Dis. 2021;40(5):905-919. doi:10.1007/s10096-020-04138-6
6. Gheblawi M, Wang K, Viveiros A, et al. Angiotensin-Converting Enzyme 2: SARS-CoV-2 Receptor and Regulator of the Renin-Angiotensin System: Celebrating the 20th Anniversary of the Discovery of ACE2. Circ Res. 2020;126(10):1456-1474. doi:10.1161/CIRCRESAHA.120.317015
7. Momtazmanesh S, Shobeiri P, Hanaei S, Mahmoud-Elsayed H, Dalvi B, Malakan Rad E. Cardiovascular disease in COVID-19: a systematic review and meta-analysis of 10,898 patients and proposal of a triage risk stratification tool. Egypt Heart J. 2020;72(1):41-49. doi:10.1186/s43044-020-00075-z
8. You H, Zhao Q, Dong M. The Key Genes Underlying Pathophysiology Correlation Between the Acute Myocardial Infarction and COVID-19. Int J Gen Med. 2022;15:2479-2490. doi:10.2147/IJGM.S354885
9. Wolsky RM, Bateman CT. A Myocardial Infarction Following a Mild Case of COVID-19 in a 26-Year-Old Male. Cureus. 2022;14(7):e27026. doi:10.7759/cureus.27026
10. Othman F, Abid AR, Alibrahim M, et al. Non-ST Segment Elevation Myocardial Infarction in a Patient with COVID-19. HEART VIEWS. 2020;21(3):5-12.
11. Watson RA, Johnson DM, Dharia RN, Merli GJ, Doherty JU. Anti-coagulant and anti-platelet therapy in the COVID-19 patient: a best practices quality initiative across a large health system. Hosp Pract. 2020;48(4):169-179. doi:10.1080/21548331.2020.1772639
12. Walsh MN, Sorgente A, Fischman DL, Bates ER, Grapsa J. The COVID-19 Pandemic and Cardiovascular Complications. JACC Case Rep. 2020;2(9):1235-1239. doi:10.1016/j.jaccas.2020.06.017
13. Diebold M, Zimmermann T, Dickenmann M, et al. Comparison of Acute Kidney Injury in Patients with COVID-19 and Other Respiratory Infections: A Prospective Cohort Study. J Clin Med. 2021;10(11):2288-2296. doi:10.3390/jcm10112288
14. Perico L, Benigni A, Remuzzi G. Should COVID-19 Concern Nephrologists? Why and to What Extent? The Emerging Impasse of Angiotensin Blockade. Nephron. 2020;144(5):213-221. doi:10.1159/000507305
15. Legrand M, Bell S, Forni L, et al. Pathophysiology of COVID-19-associated acute kidney injury. Nat Rev Nephrol. 2021;17(11):751-764. doi:10.1038/s41581-021-00452-0
16. Głowacka M, Lipka S, Młynarska E, Franczyk B, Rysz J. Acute Kidney Injury in COVID-19. Int J Mol Sci. 2021;22(15):8081-8096. doi:10.3390/ijms22158081
17. Chu KH, Tsang WK, Tang CS, et al. Acute renal impairment in coronavirus-associated severe acute respiratory syndrome. Kidney Int. 2005;67(2):698-705. doi:10.1111/j.1523-1755.2005.67130.x
18. Chávez-Valencia V, Orizaga-de-la-Cruz C, Lagunas-Rangel FA. Acute Kidney Injury in COVID-19 Patients: Pathogenesis, Clinical Characteristics, Therapy, and Mortality. Diseases. 2022;10(3):53-62. doi:10.3390/diseases10030053
19. Claure-Del Granado R, Mehta RL. Fluid overload in the ICU: evaluation and management. BMC Nephrol. 2016;17(1):109-117. doi:10.1186/s12882-016-0323-6
20. Jonny, Violetta L, Kartasasmita AS, Amirullah Roesli RM, Rita C. Pharmacological Treatment Options for Coronavirus Disease-19 in Renal Patients. Uribarri J, ed. Int J Nephrol. 2021;2021:1-9. doi:10.1155/2021/4078713
21. Wong CKH, Au ICH, Cheng WY, et al. Remdesivir use and risks of acute kidney injury and acute liver injury among patients hospitalised with COVID -19: a self-controlled case series study. Aliment Pharmacol Ther. 2022;56(1):121-130. doi:10.1111/apt.16894
22. Setiati S, Azwar MK. COVID-19 and Indonesia. Acta Med Indones. 2020;52(1):84-89.
23. Mohamadi Yarijani Z, Najafi H. Kidney injury in COVID-19 patients, drug development and their renal complications: Review study. Biomed Pharmacother. 2021;142:111966. doi:10.1016/j.biopha.2021.111966
24. Olson G, Davis AM. Diagnosis and Treatment of Adults With Community-Acquired Pneumonia. JAMA. 2020;323(9):885-892. doi:10.1001/jama.2019.21118
25. Wiersinga WJ, Bonten MJ, Boersma WG, et al. Management of community-acquired pneumonia in adults: 2016 guideline update from the Dutch Working Party on Antibiotic Policy (SWAB) and Dutch Association of Chest Physicians (NVALT). Neth J Med. 2018;76(1):10-18.