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Case Report

Comprehensive Management Conferring an Improved Outcome for a post –COVID-19 Patient With Congestive Heart Failure: A Case Report

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ABSTRACT

Background: The COVID-19 pandemic has been unresolved for 3 years. Numerous individuals have survived COVID-19 but suffered from a variety of problems or symptoms for several months. This report presents our experience regarding a post–COVID-19 patient with exacerbated symptoms of congestive heart failure and congestive hepatopathy.

Result: Two months after being diagnosed with COVID-19, a 47-year-old man presented to Universitas Islam Indonesia Hospital with significant shortness of breath and abdominal enlargement. Cardiomegaly, increased jugular venous pressure, hepatomegaly, ascites, and bilateral edema of the legs were all indicators of congestive heart failure. Cardiomegaly was discovered on a chest X-ray. Electrocardiography revealed ischemic heart illness with an old myocardial infarction. Aspartate transaminase (911 U/L), alanine transaminase (820 U/L), and total bilirubin (4-6 mg/dL) were increased. Ultrasonography of the abdomen demonstrated congestive hepatopathy with ascites. Echocardiography showed hypokinetic cardiac wall movements and a 21% decrease in the ejection fraction. He had a comorbidity of uncontrolled diabetes mellitus. The symptoms subsided after therapy with furosemide, spironolactone, dobutamine, nebivolol, sacubitril, valsartan, L-ornithine-L-aspartate, insulin, and a broad spectrum of antibiotics. After 10 days of hospitalization with comprehensive management, the patient's condition improved.

Conclusions: Our post–COVID-19 patient suffering from congestive heart failure with a poor ejection fraction and severe congestive hepatopathy had an improved outcome following our comprehensive management. ***(Iranian Heart Journal 2023; 24(3): 100-107)***

KEYWORDS: Post–COVID-19, Congestive heart failure, Ascites, Congestive hepatopathy, Management

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The COVID-19 pandemic started in December 2019 and afflicted in excess of 220 countries, with more than 649 million affected cases and over 6.6 million deaths around the world by December 2022. The case fatality rate of

COVID-19 is 1.2%. [1] COVID-19 patients with comorbidity of cardiovascular disease and cardiovascular risks, such as diabetes mellitus and hypertension, have more significant risks of developing severe COVID-19 and mortality. [2] COVID-19 can affect nearly all organs in the body, including cardiac tissues, resulting in myocarditis, myocardial infarction, cardiomyopathy, and acute decompensated heart failure during or after hospitalization through a hyperinflammation mechanism. [3]

CASE REPORT

A 47-year-old man came to Universitas Islam Indonesia Hospital with chief complaints of dyspnea, enlargement of the abdomen and scrotum, and leg edema. He had been diagnosed with COVID-19 two months earlier and received treatment at a hospital in the Kulon Progo district of Yogyakarta, Indonesia. After recovering, the patient was discharged from the hospital. Several weeks later, he developed shortness of breath, abdomen and scrotum enlargement, and bilateral leg edema. He returned to the previous hospital; nonetheless, as there was no improvement in his condition, he came to Universitas Islam Indonesia Hospital for treatment. He had been diagnosed with type 2 diabetes mellitus 2 years earlier but had never consumed medication regularly.

The physical examination revealed a blood pressure of 104/84 mm Hg, a heart rate of 99 beats per minute, a respiration rate of 25 times per minute, a body temperature of 36.6 ℃, and an oxygen saturation level of 97% in room air. Slight hypotension and slightly decreased oxygen saturation were reported. Jugular venous pressure was elevated. The thorax examination showed symmetrical movements of the chest wall, normal tactile fremitus, normal percussion, and normal vesicular sounds and basal rales. Cardiomegaly was present, with normal first and second heart sounds. Additionally, ascites was diagnosed during the abdomen examination; however, the liver and the spleen could not be touched. There was edema and enlargement of the scrotum, as well as edema in both legs. Mild icteric was also detected in the sclera.

Electrocardiography (fig 1) displayed an old myocardial infarction (Q wave) in the anteroseptal area and left axis deviation.

The chest X-ray (fig 2) showed that the lungs were normal, but there was gross cardiomegaly.

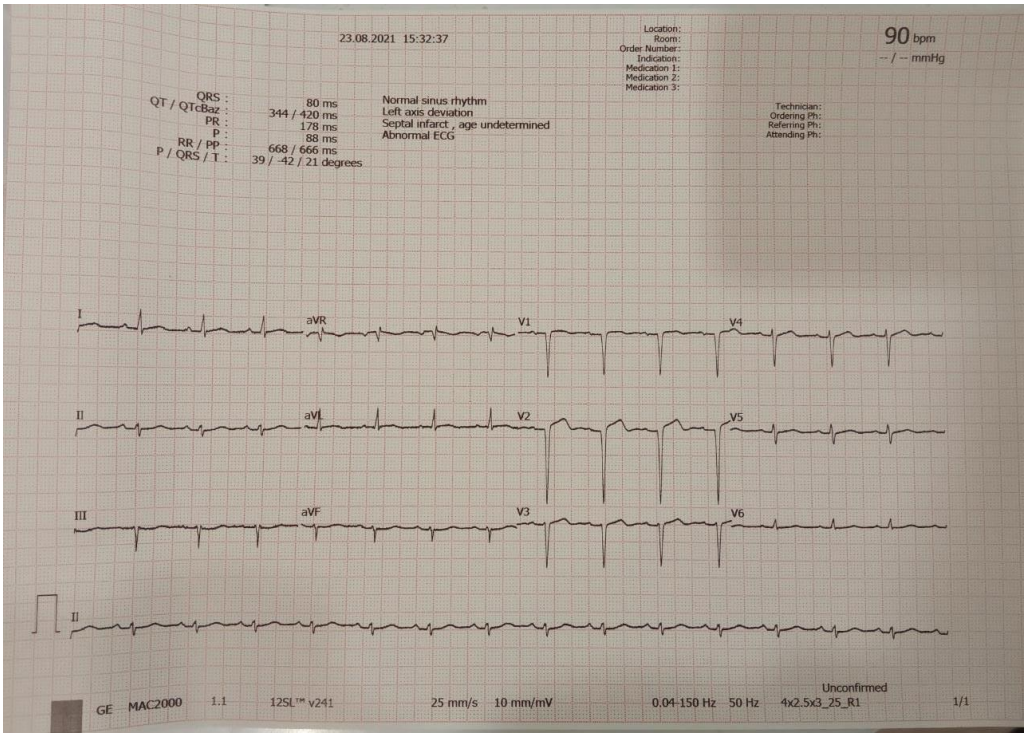

Figure 1: The image presents the patient's electrocardiogram.

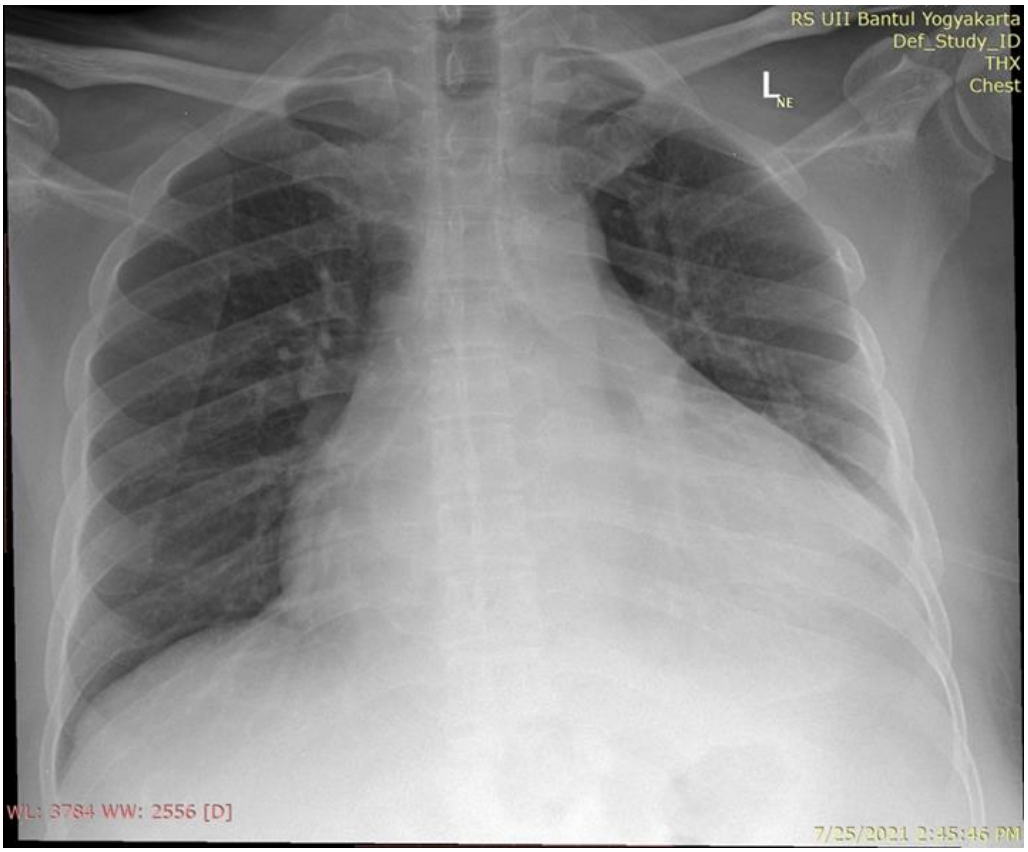

Figure 2: The image shows the patient's chest X-ray.

The routine blood examination (Table 1) revealed that almost all tests were normal, except the differential count of leucocytes. There was a domination of neutrophils and a decrease in lymphocytes with a high neutrophil-to-lymphocyte ratio.

The chemical blood examination (Table 2) showed a normal albumin level, a negative SARS-CoV-2 test, elevated aspartate and alanine aminotransferase levels, and modestly increased urea and creatinine levels.

Serological and coagulation function tests (Table 3) were performed to determine whether there was a hepatitis virus infection. The coagulation function was normal, and there was no indication of viral hepatitis infection.

The urine examination (Table 4) revealed that there was no proteinuria, but there was a urinary tract infection.

Echocardiography indicated left atrial dilatation, left ventricular dilatation, decreased left ventricular systolic function (the ejection fraction: 21%), segmental hypokinetic movements, diastolic dysfunction of the left ventricle (restrictive type), and mild mitral regurgitation. The echocardiography results demonstrated ischemic heart disease with a low ejection fraction.

Ultrasonography of the abdomen revealed ascites, hepatitis, and sludge in the gallbladder. The abdomen ultrasonography confirmed ascites.

Diagnosis and Treatment

The patient was diagnosed with post-COVID-19 congestive heart failure (functional class III), congestive hepatopathy, type 2 diabetes mellitus, and urinary tract infection. He received comprehensive treatment, encompassing oxygen administration with a nasal cannula (3 L/min), a diabetic diet (1700 calories with a low salt intake), 0.9% sodium chloride (8 drops/minute), insulin (NovoRapid: 6 units subcutaneously in 8 hours), furosemide (5 mg/h), spironolactone (25 mg twice a day), omeprazole (40 mg/24 h), HepaMerz (L-ornithine L-aspartate: 1 ampule/12 hours), Curcuma (3×1), cefotaxime (1 g/8 h), isosorbide dinitrate (5 mg as needed), dobutamine (3 µg/kg body weight/min), Uperio (sacubitril and valsartan: 2×25 mg), Nebilet (nebivolol: 2×5 mg), Laxadine Syrup (lactulose syrup), Dulcolax (as needed), and Aspar K (2×300 mg).

After 4 days of treatment, there was a significant reduction in ascites and edema of the scrotum and legs. The liquid balance was adjusted to be negative (at least 500 mL daily). Laboratory examination follow-ups (Table 5) showed improvements in some laboratory indicators, such as blood glucose and hepatic enzymes.

Table 1: Routine blood examination results of the patient

Item	Level	Normal Value	Interpretation
Hemoglobin	17.1 g %	13.2-17.3	normal
Erythrocyte	5.79 million/µl	4.4-5.9	normal
Hematocrit	48.9 %	40-52	normal
Thrombocyte	182 thousand/µL	150-450	normal
Leucocyte	6.46 thousand/µL	3.8-10.6	normal
Neutrophil	79.7 %	28-78	increase
Lymphocyte	10.5%	25-40	low
Monocyte	9.8 %	2-8	increase
Eosinophil	0%	2-4	low
Basophil	0%	0-1	normal
Neutrophil-to-lymphocyte ratio	7.57	1-3	increase
MCV	84.5 FL	80-100	normal
MCH	29.5 pg.	26-34	normal

Table 2: Chemical blood laboratory examination results of the patient

Item	Level	Normal Value	Interpretation
Total Protein	6 mg/dL	6-8	normal
Globulin	2,3 mg/dL	2-3.4	normal
Albumin	3.7 mg/dL	3.4-4.8	normal
Antigen SARS-CoV2	Negative	Negative	normal
PCR SARS- CoV2	Negative	Negative	normal
Random blood glucose	255 mg/dL	75-140	high
Aspartate aminotransferase	911 U/L	<35	high
Alanine transaminase	820 U/L	<31	high
Urea	102 mg/dL	(17-50)	slight increase
Creatinine	1.37 mg/dL	(0.75-1.35)	slight increase

Table 3: Serological hepatitis viral infection and coagulation functions in the laboratory examination of the patient

Item	Level	Normal Value	Interpretation
HBsAg	Non-reactive	Non-reactive	Normal
Anti-HCV	Non-reactive	Non-reactive	Normal
PPT	22.8	11-15	Normal
APTT	29.8	28-40	Normal

Note: HBsAg, Hepatitis B virus surface antigen; HCV, Hepatitis C virus; PPT, Partial thromboplastin time; APTT, Activated partial thromboplastin time

Table 4: Urine examination results of the patient

Item of the Urine Test	Level	Normal Value	Interpretation
pH	6	6-7	normal
Specific gravity	1.02	1.01-1.025	normal
Nitrite	Negative	Negative	normal
Protein	Negative	Negative	normal
Glucose	Negative	Negative	normal
Ketone	Negative	Negative	normal
Urobilinogen	0.2	0.1-1.0	normal
Leucocyte	+3	Negative	abnormal
Blood	+2	Negative	abnormal
Leucocyte	40-45/sfv	0-5	abnormal
Erythrocyte	>100/sfv	<2	abnormal
Bacteria	Positive	Negative	abnormal
Yeast	Negative	Negative	normal

Table 5: Follow-up of the patient's laboratory examination

Item	Level	Normal Value	Interpretation
Blood glucose (day 4)	110 mg/dL	75-140	Normal/controlled
Aspartate aminotransferase (day 4)	419 U/L	<35	improved
Alanine transaminase (day 4)	461 U/L	<31	improved
Aspartate aminotransferase (day 8)	125 U/L	<35	improved
Alanine transaminase (day 8)	120 U/L	<31	improved

After 10 days in the hospital, the patient was discharged. Shortness of breath was alleviated, ascites was minimized, and edema of the scrotum and legs was eliminated. Body weight was reduced from 70 kg to 58 kg, while waist circumference was reduced from 112 cm to 98 cm.

We provided outpatient treatment, consisting of furosemide (40 mg 1-1-0), insulin (NovoRapid 3×6 units subcutaneously), spironolactone (2×25 mg), lansoprazole (1×30 mg), Curcuma (3×1), cefixime (2×200 mg), Uperio (sacubitril and valsartan: 2×25 mg), Nebilet (nebivolol: 2×5 mg), Laxadine Syrup, and Aspar K (2×300 mg).
A follow-up visit to the outpatient clinic revealed that the significant shortness of breath had vanished. Congestive heart failure symptoms, such as ascites and edema of the scrotum and legs, were eliminated.

DISCUSSION

During their hospitalization, several COVID-19 patients acquired heart problems (7% of all COVID-19 patients and 22% of critically ill patients). [4] A prior study revealed that about 10% of patients with COVID-19 developed heart failure and myocardial damage. [5] Previous congestive heart failure in COVID-19 patients exerts a negative impact on hospitalization outcomes. COVID-19 is associated with a greater risk of developing heart failure in the future and may impair heart function, resulting in an increase in heart failure symptoms (acute decompensated heart failure). [6]
Our patient developed congestive heart failure 2 months after being diagnosed with COVID-19.
COVID-19 causes cardiovascular disease via 4 mechanisms: 1) a cytokine storm, causing epithelial dysfunction; 2) renin-angiotensin-aldosterone system dysfunction or abnormalities; 3) inflammation-induced disruption of atheroma plaques (plaque instability); and 4) hypercoagulability, causing disseminated intravascular coagulation. Our patient had severely impaired cardiac function since he had a low ejection fraction, severe congestive heart failure, and congestive hepatopathy. Adequate treatment aids in the improvement of the patient's condition. [7, 8]
A systematic review indicated that patients surviving COVID-19 may develop cardiovascular complications 3 to 6 months afterward. The symptoms include dyspnea, chest pain, diastolic dysfunction, late gadolinium enhancement, and elevated N-terminal pro–B-type natriuretic peptide, with patients having a higher risk of developing heart failure, arrhythmias, and myocardial infarction. [9]
The case fatality rate of COVID-19 patients with cardiovascular disease is 10.5%. [10] A previous study reported a rise in the incidence of cardiovascular disease during the COVID-19 pandemic, with the conditions including arrhythmias, ischemic and nonischemic heart diseases, myocarditis, pericarditis, thromboembolic events, and congestive heart failure. [11] The European Society of Cardiologists recommends the management of acute decompensated heart failure with diuretics, an angiotensin-converting-enzyme inhibitor (ACEI) or an angiotensin II receptor blocker (ARB), or a combination of sacubitril, an ARB, and a β-blocker. In acute decompensated heart failure, β-blockers should be administered at the lowest possible dose. Sacubitril, in combination with an ARB, is beneficial in reducing rehospitalization in patients with severe congestive heart failure. [12] ACEIs and ARBs may confer protective effects on individuals suffering from COVID-19. Still, data in this regard are inadequate, and further research is warranted. [5]
A prior report described a patient suffering from post–COVID-19 syndrome or long-COVID-19 hospitalized due to heart attack

and stroke. The patient was diagnosed with congestive heart failure and ischemic heart disease. [13]

Since the first year of the pandemic, researchers have reported that patients suffering from the initial infection of COVID-19 may develop cardiac injury even if they have no history of cardiovascular disease. [14]

Post-COVID-19 patients could suffer from long COVID-19 syndrome, which affects cardiac function. [15] We discovered congestive hepatopathy in our patient, who also had elevated liver enzymes. The administration of Curcuma and L-ornithine L-aspartate resulted in significant improvement. As comorbidity, our patient also had diabetes mellitus and urinary tract infection. The administration of insulin and a broad-spectrum antibiotic offered optimal blood glucose control. The physicians in charge of the patient came from the departments of internal medicine, anesthesia, critical care, and cardiology, and they worked well with dieticians and nurses. Comprehensive patient management enhanced the patient's condition significantly, and continuing treatment in outpatient care resulted in a better outcome. Previous research indicates that individuals with cardiovascular risk factors, such as diabetes and hypertension, fare the worst when exposed to COVID-19. [16] However, our patient survived COVID-19 while having the complication of severe congestive heart failure, which was successfully resolved with extensive care.

A previous study concluded that many patients with COVID-19 may develop heart failure and irregular heartbeats as a result of the inflammatory process in the heart. Comprehensive heart failure care has been successful in saving some individuals suffering from post–COVID-19 syndrome from heart failure. [17]

CONCLUSIONS

We herein described a post–COVID-19 male patient suffering from congestive heart failure with a poor ejection fraction and severe congestive hepatopathy with comorbidities, consisting of diabetes mellitus and urinary tract infection. Furosemide, sacubitril, valsartan, β-blockers, dobutamine, insulin, broad-spectrum antibiotics, L-ornithine L-aspartate, and Curcuma all contributed to the patient's improvement.

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Conflict of Interest: None

REFERENCES

1. WHO, *Coronavirus Disease*. 2022.

2. B. De Almeida-Pititto et al., "Severity and mortality of COVID 19 in patients with diabetes, hypertension and cardiovascular disease: A meta-analysis," Diabetol. Metab. Syndr., vol. 12, no. 1, pp. 1–12, 2020, doi: 10.1186/s13098-020-00586-4.

3. U. Landmesser, I. Lehmann, and R. Eils, "Hyperinflammation as underlying mechanism predisposing patients with cardiovascular diseases for severe COVID-19 ," Eur. Heart J., vol. 42, no. 18, pp. 1720–1721, 2021, doi: 10.1093/eurheartj/ehab191.

4. K. J. Clerkin et al., "COVID-19 and Cardiovascular Disease," Circulation, vol. 141, no. 20, pp. 1648–1655, 2020, doi: 10.1161/CIRCULATIONAHA.120.046941.

5. L. Italia et al., "COVID-19 and Heart Failure: From Epidemiology During the Pandemic to Myocardial Injury, Myocarditis, and Heart Failure Sequelae,"

Front. Cardiovasc. Med., vol. 8, Aug. 2021, doi: 10.3389/fcvm.2021.713560.

6. M. Linschoten et al., "Cardiac complications in patients hospitalised with COVID-19 ," Eur. Hear. Journal. Acute Cardiovasc. Care, vol. 9, no. 8, pp. 817–823, 2020, doi: 10.1177/2048872620974605.

7. M. Nishiga, D. W. Wang, Y. Han, D. B. Lewis, and J. C. Wu, "COVID-19 and cardiovascular disease: from basic mechanisms to clinical perspectives," Nat. Rev. Cardiol., vol. 17, no. 9, pp. 543–558, 2020, doi: 10.1038/s41569-020-0413-9.

8. V. Petrovic, D. Radenkovic, G. Radenkovic, V. Djordjevic, and M. Banach, "Pathophysiology of Cardiovascular Complications in COVID-19 ," Front. Physiol., vol. 11, no. October, pp. 1–11, 2020, doi: 10.3389/fphys.2020.575600.

9. M. S. Ramadan, L. Bertolino, and R. Zampino, "Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID- 19 . The COVID-19 resource centre is hosted on Elsevier Connect , the company ' s public news and information ," no. January, 2020.

10. S. Dan, M. Pant, and S. K. Upadhyay, "The Case Fatality Rate in COVID-19 Patients With Cardiovascular Disease: Global Health Challenge and Paradigm in the Current Pandemic," Curr. Pharmacol. Reports, vol. 6, no. 6, pp. 315–324, 2020, doi: 10.1007/s40495-020-00239-0.

11. Y. Xie, E. Xu, B. Bowe, and Z. Al-Aly, "Long-term cardiovascular outcomes of COVID-19 ," Nat. Med., vol. 28, no. 3, pp. 583–590, Mar. 2022, doi: 10.1038/s41591-022-01689-3.

12. T. A. McDonagh et al., "2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure," Eur. Heart J., vol. 42, no. 36, pp. 3599–3726, 2021, doi: 10.1093/eurheartj/ehab368.

13. P. Management and D. L. Goldenberg, "COVID-19 Long Haulers : A Look at Cardiovascular Risk," no. Cv, pp. 1–8.

14. L. Williamson, "What COVID-19 is doing to the heart, even after recovery," Am. Hear. Assoc. News, pp. 1–4, 2020, [Online]. Available: https://www.heart.org/en/news/2020/09/03/what-COVID-19 -is-doing-to-the-heart-even-after-recovery.

15. M. Chilazi, E. Y. Duffy, A. Thakkar, and E. D. Michos, "COVID and Cardiovascular Disease: What We Know in 2021," Curr. Atheroscler. Rep., vol. 23, no. 7, 2021, doi: 10.1007/s11883-021-00935-2.

16. S. A. Bae, S. R. Kim, M. N. Kim, W. J. Shim, and S. M. Park, "Impact of cardiovascular disease and risk factors on fatal outcomes in patients with COVID-19 according to age: A systematic review and meta-analysis," Heart, vol. 107, no. 5, pp. 373–380, 2021, doi: 10.1136/heartjnl-2020-317901.

17. D. R. Anderson, "Post-COVID syndrome : How ' s your heart ? By," pp. 1–6, 2021.