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Case Study
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Coronavirus Disease with Heart Failure: A Case Report

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Abstract

In this report, we describe the case of a 78-year-old man with no history of lung disease who presented with heart failure 2 weeks after the diagnosis of Coronavirus Disease (COVID-19). After the administration of anti-virals and diuretics, nutritional treatment for myocardial infarctions, and other treatments, the patient tested negative for severe acute respiratory syndrome coronavirus 2, and his heart function significantly improved. Thus, we postulate that COVID-19 can lead to decreased heart function; attention must be paid to changes of inflammation and heart function, which must be actively treated.

Introduction

The Coronavirus Disease (COVID-19) outbreak is an unprecedented global public health challenge. On January 30, 2020, the World Health Organization (WHO) declared the disease caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) a Public Health Emergency of International Concern (PHEIC), later officially upgrading it to a global pandemic. As of April 29, 2020, more than 3,000,000 confirmed cases from over 177 countries and more than 50,000 deaths have been documented worldwide. In some patients, septic shock occurs rapidly, eventually followed by multiple organ failure. Apart from acute respiratory failure, patients experience serious complications such as acute cardiac injury, which has also been observed in a large number of patients [1,2]. Therefore, we report a case of COVID-19 focusing on the treatment process for patients with new coronary heart disease and heart failure.

Case Presentation

History of presentation

A 78-year-old male native of Wuhan city was admitted to the hospital with a chief complaint of an "intermittent fever for 1 week." Upon presentation, he reported intermittent chills, cough, chest tightness, headache, and fatigue. A SARS-CoV-2 RNA test with a throat swab specimen showed positive results. The patient was febrile (37.8°C) with a heart rate of 96 beats per minute. His arterial oxygen saturation (SaO_2) was 97% on 3 L of supplemental oxygen. Physical examination demonstrated coarse crackles in the bilateral lower lung fields.

Medical history

His medical history included hypertension.

Investigations

Routine blood examination showed increased N-Terminal pro-Brain Natriuretic Peptide (NT-pro BNP) levels; decreased leukocyte and lymphocyte counts; decreased hemoglobin level; and increased Lactate Dehydrogenase (LDH), TNF-a, and IL-6 levels (Table 1). The patient received intermittent oxygen inhalation and supportive treatment. After active diuresis, nutrient treatment for myocardial infarction, and other treatments, the patient's heart failure symptoms improved significantly. His NT-pro BNP and electrolyte levels also normalized.

Management

After the patient was admitted to the hospital, he received lopinavir and ritonavir tablets, Lianhuaqingwen capsules to control body temperature, compound methoxyphenamine capsules to treat cough, and amlodipine besylate for hypertension. After the patient developed heart failure, we administered furosemide for diuresis, coenzyme Nicotinamide Adenine Dinucleotide (NAD+) and vitamin C as nutrient treatment for the myocardium, and symptomatic treatment. At the time of submission of this report, the patient had been discharged.

Discussion

The most common laboratory abnormalities found in COVID-19 patients include decreased

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Table 1: Laboratory parameters in the present case.

	Day 1	Day 2	Day 3	Day 5	Normal range
Leucocytes 10^9/L	4.59			2.54	3.5-9.5
Lymphocyte 10^9/L	0.31			0.79	1.1-3.2
Neutrophil 10^9/L	4.19			1.15	1.8-6.3
Hemoglobin (g/L)	111			112	130-175
LDH (U/L)	248			210	135-225
Prothrombin time (second)				13.8	11.5-14.5
D-Dimer (ug/ml)				2.14	<0.5
Fibrinogen (g/L)				4.27	2.0-4.0
Total protein (g/L)	76			80.3	64-83
Albumin (g/L)	30.1			31.9	35-52
AST (U/L)	24			18	<40
ALT (U/L)	22			15	<41
Serum creatinine (umol/L)	56			59	45-84
Serum kalium (mmol/L)	5.73	4.6			3.5-5.5
Serum sodium (mmol/L)	128.9	137			136-145
Serum calcium (mmol/L)	2.13	2.21			2.2-2.5
Glucose (mmol/L)	5.41				4.11-6.05
C-reactive protein (mg/L)	5.2		2.6		<1
Interleukin-1β (pg/mL)	6.5		5.7		<5
Interleukin-6 (pg/mL)	64.45		42.24		<7
Interleukin-8 (pg/mL)	6.6		13		<62
Interleukin-10 (pg/mL)	7.1		5.8		<9.1
TNF-α (pg/mL)	12.7		10.5		<8.1
NT-BNP (pg/mL)	512			197	<285

lymphocyte, neutrophil, and leukocyte counts, as well as elevated serum C-reactive protein and LDH levels [1,3].

Present data regarding COVID-19 patient's show significantly increased levels of plasma pro-inflammatory cytokines including IL1-β, IL-8, IL-6, and TNF-α [4]. Increasing evidence suggests that enhanced production of a large number of inflammatory cytokines can directly or indirectly cause cardiac injury [5]. These findings are consistent with the findings of our case.

Cardiovascular diseases are the most common comorbidities in patients with COVID-19. Available data indicate that COVID-19 patients are often diagnosed with hypertension (15% to 30.4%), coronary artery disease (2.5% to 8%), or other cardiovascular diseases (4% to 14.6%) [6]. In addition, patients with concomitant cardiovascular diseases have a worse prognosis and require admission to the Intensive Care Unit (ICU) more often than patients without such comorbidities [2,3,7]. In one study, among 120 critically ill patients infected with SARS-CoV-2, 27.5% had elevated NT-pro BNP concentrations and 10% had increased cardiac troponin levels [8]. These findings indicate that cardiovascular injury may affect systemic stability and should not be ignored. Whole-genome sequencing analysis showed that the SARS-CoV-2 virus is a novel virus different from the SARS virus and Middle East Respiratory Syndrome virus [9]. However, there are some clinical similarities. There is no effective treatment or vaccine for COVID-19 pneumonia currently, which is mostly, treated using supportive treatment [9-11]. In our case, early treatment mainly involved antiviral, anti-inflammatory, and symptomatic treatment. Later, the increased expression of many inflammatory factors (TNF-α, IL-6) caused systemic inflammatory reactions, which damaged the myocardium was also damaged, eventually causing heart failure. NT-pro BNP levels increased significantly. At this time, while the patient was receiving anti-inflammatory drugs, we performed diuresis and administered coenzyme I for anti-oxidative stress in addition to other treatments. Subsequently, the patient's inflammation and heart failure symptoms improved, and other parameters returned to the normal range. After myocardial injury, we used NAD+ (Coenzyme I 30 mg), which can specifically activate sirtuin activity, promote myocardial metabolism, improve anti-oxidative stress, reduce inflammation, and correct heart failure [12].

Conclusion

While administering anti-virals, attention should be paid to the presence of heart failure symptoms and the amount of fluid infusion. When COVID-19 is accompanied with heart failure, diuresis and nutritional treatment for the myocardium are required.

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