# **Case Report**

# Reinfection with SARS-CoV-2 after Aortic Valve Replacement for Infective Endocarditis

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## **ABSTRACT**

Although the COVID-19 pandemic started over 2 years ago, the uncertainty regarding the management of this severe disease continues to prevail. We describe a patient with aortic valve endocarditis complicated by COVID-19 postoperatively. Given increased symptom severity during reinfection, it is important to highlight the need for the long-term follow-up of these patients since they might be prone to another episode of COVID-19. (*Iranian Heart Journal 2023; 24(3): 108-110*)

**KEYWORDS:** Cardiac surgery, COVID-19, Pneumonia, Reinfection

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Received: January 29, 2023	Accepted: March 15, 2023

espite extensive efforts to contain the virus, the number of COVID-19 rise. continues cases to and reinfection with SARS-CoV-2 is becoming a possibility. Patients previously strong infected with mild or asymptomatic disease could eventually become seronegative, losing the ability to protect themselves. Infective endocarditis (IE), with its high morbidity and mortality, continues to be a major concern for the healthcare system.<sup>2</sup> We describe a middle-aged man with severe COVID-19 reinfection concomitant with aortic valve endocarditis 7 months after the first infection.

#### **CASE REPORT**

A 40-year-old man with a history of hypertension was diagnosed with COVID-19 in early April 2022. At that time, he presented to the hospital with worsening shortness of breath, necessitating a high oxygen flow. The patient was hospitalized in the COVID-19 unit for a few days. Having had a clear improvement, he was discharged home.

Two months later, he presented again to the emergency ward with a fever and palpitations. On physical examination, he conscious with no neurological was symptoms. His body temperature was 38 °C, and there were audible aortic diastolic murmurs. Chest X-ray showed cardiomegaly with the normal appearance of both lungs. Hematological examinations showed hemoglobin of 9.3 g/dL, white blood cells of 5300/mm<sup>3</sup>, and a platelet count of  $128000/\text{mm}^3$ . The patient was transferred to the cardiology department to determine the etiology.

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Upon admission, he underwent transthoracic echocardiography, which showed severe aortic regurgitation with a dilated left ventricle, a preserved ejection fraction, and 2 mobile vegetation formations on the noncoronary aortic cusp measuring respectively 13 mm and 9 mm (fig 1A-1B). Cranial magnetic resonance imaging was performed as an extension study of IE, and it depicted an aneurysm in the right middle cerebral artery with a diameter of 11 mm associated with a right frontal ischemic lesion. Blood cultures were positive for Enterococcus faecalis with a high level of resistance to aminoglycoside antibiotics. The patient was initiated on an intravenous antibiotic regimen of ampicillin and ceftriaxone during 38 days without any improvement. Given persistent bacteremia, he then received linezolid for 28 days, which ameliorated the fever. The blood culture came back negative.

The patient was scheduled for aortic valve replacement 7 months after the first SARS-CoV2 infection. On arrival, he had no fever or respiratory signs. The surgery was carried out 8 days after the admission. Following a careful excision of the aortic cusps with skinny vegetation attached to the noncoronary cusp, the valve was replaced with a 25 mm bioprosthetic, and the patient was weaned from cardiopulmonary bypass without difficulty. However, he underwent re-exploration for a hemostasis revision without identifiable site bleeding. He was extubated 10 hours after reopening for bleeding, and presented an adequate postoperative recovery the first 48 hours.

The next day, the patient had a fever, difficulty in breathing, increased oxygen requirements, and a rapid progression of opacities in the thorax X-ray. The diagnosis of COVID-19 was confirmed by the reverse transcription polymerase chain reaction test. Fortunately, the patient's respiratory function gradually improved, and he was discharged home. At follow-up 6 weeks later, he had already returned to his daily activities and was completely symptom-free.

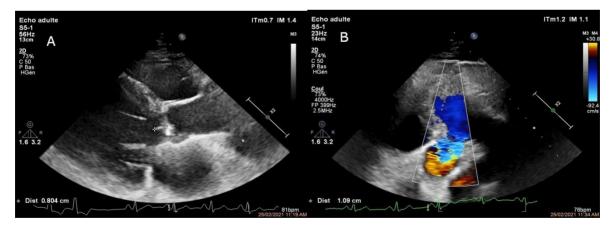


FIGURE 1: A- Transesophageal imaging shows vegetation on the non-coronary aortic cusp. B- Color Doppler demonstrates severe aortic regurgitation.

### DISCUSSION

With the rapid transmission of the coronavirus, we will have more cases of reinfection. The susceptibility of previously

infected patients to reinfection is still unclear. When any viral infection occurs, the levels of specific IgG antiviral antibodies rise a few weeks after symptom onset and persist for many years. Nonetheless, this is

not the case with coronavirus infections, and neutralizing immunoglobulins against SARS-CoV-2 persist for about 40 days.<sup>3</sup> This decrease in protective antibody titers after COVID-19 may explain reinfection. Patients with a more severe illness and prolonged viral shedding are more prone to developing protective immunity toward subsequent infections.<sup>1</sup> In our case, the patient's initial infection was moderate. while the reinfection occurred with severe symptoms, and it is unknown whether the patient developed any antibodies following his primary infection.

Other possibilities predisposing patients to reinfection include viral reactivation after a carriage of the prolonged virus or <sup>4</sup> In our immunodeficiency conditions. patient, reinfection occurred over a period of months after the first infection. 6 Accordingly, it is highly likely that the recovered and patient subsequently experienced reinfection. However. cardiopulmonary bypass may have reduced system, immune causing his severe symptoms and predisposing to reinfection.

COVID-19 also significantly impacts the hematopoietic system and hemostasis. Patients with COVID-19 exhibit coagulation abnormalities with longer prothrombin times and mild thrombocytopenia. <sup>5</sup> Our patient had mild thrombocytopenia, attributable to Nevertheless, heparin therapy. this thrombocytopenia may have been secondary to the coagulation disorder of COVID-19. Additionally, his postoperative bleeding might have been caused by thrombocytopenia cardiopulmonary or bypass-related alterations in hemostasis or even their combination.

The clinical presentation of IE and COVID-19 may be similar, rendering it difficult to distinguish between both diseases clinically.

The scenario becomes even more complicated when the 2 diseases are combined. There are a few reported cases of IE and COVID-19. Performing heart surgery on a COVID-19-positive patient may be very risky for the patient. The postoperative course could be modified by SARS-CoV-2 infection, and inversely the clinical course of COVID-19 infection could be altered by cardiac surgery. For such special patients, we must rely more on the prevention of transmission and the education of infected patients regarding the possibility of reinfection.

#### Acknowledgments: None

**Conflict of Interest:** The authors report no conflicts of interest.

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