Post-COVID morphologically proved endocarditis: infective and nonbacterial forms

O. Blagova, E.A. Kogan, Y.U.A. Lutokhina, A.D. Kukleva, V.P. Sedov

I.M. Sechenov First Moscow State Medical University, Moscow, Russian Federation
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Purpose: To study clinical features of endocarditis and its possible mechanisms (infective and nonbacterial) in the long-term period after acute COVID-19.
Methods: Three patients (two male and one female, age 64, 39 and 46 years) diagnosed with postcovid endocarditis were included in the study. One patient had severe bilateral coronavirus pneumonia; the other two had only fever and weakness. The diagnosis of COVID-19 was confirmed by seroconversion. The time of admission after COVID-19 was from 4 to 7 months. All patients had study for anti-heart antibodies (AHA), EchoCG, Holter ECG, and endomyocardial biopsy (EMB) with PCR for SARS-Cov2 and cardiotropic viruses. The indication for EMB was suspected myocarditis. Blood cultures and procalcitonin levels were tested in one patient due to a prolonged febrile fever.
Results: Two variants of postcovid endocarditis have been diagnosed. The first variant was detected in two patients by EMB only. This patients had severe lymphocytic and giant cell myocarditis. In addition, EMB showed signs of lymphocytic endocarditis with infiltrates, marked thickening and fibrosis of the endocardium (Fig. 1). Some biopsy specimen were represented by fresh thrombotic masses, infiltrated with neutrophilic leukocytes. No intraventricular thrombus was detected on EchoCG and MRI. The second variant of postcovid endocarditis developed in a patient with bicuspid aortic valve and met the criteria of infectious endocarditis 2015: mobile vegetations on the valve with aortic regurgitation II, splenomegaly, irregular fever up to 39°C for six months, marked increase of CRP, procalcitonin and ferritin, hypochromic anemia, LV EF 25%. Blood culture was negative. After intravenous therapy with antibiotics and immunoglobulin, EMB confirmed the active lymphocytic myocarditis and only slight fibrosis of right ventricular endocardium. The bacteriological study of endocardium was negative. SARS-Cov-2 RNA was detected by PCR in myocardial biopsy specimens of two patients; the biopsy of one patient is in the study now. All patients had significantly elevated antibody titers to various cardiac antigens, but the level of antibodies to endothelial antigens remained completely normal. It is possible to suggest an active deposition of immune complexes in the endothelium. Two surviving patients receive steroid therapy (in case of IE with antibiotics).
Conclusions: SARS-Cov-2 infection induces the prolonged non-bacterial thromboendocarditis or infective endocarditis. In both cases, autoimmune mechanisms play a significant role, as evidenced by the simultaneous lymphocytic/giant cell myocarditis and high titers of AHA. Long-term persistence of coronavirus in the myocardium can also be considered as an etiological factor of endocarditis. In favor of this hypothesis is the parietal thrombosis in the absence of bacterial infection. Corticosteroids and anticoagulants should be considered for the treatment of postcovid endocarditis.

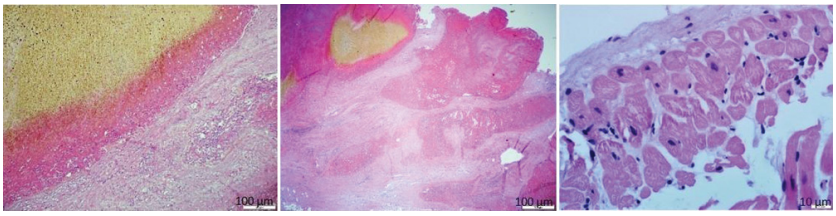

Figure 1. The EMB in in postcovid endocarditis