Biventricular apical ballooning in patient with COVID-19

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COVID-19 disease due to severe acute respiratory syndrome Coronavirus 2 (SARS-CoV₂) has different types of cardiovascular manifestations. Detectable high sensitivity cardiac troponin I and abnormal CMR has been reported in 71% and 78% of recently recovered COVID-19 patients, respectively [1]. Cardiac injury associated with COVID-19 illness has been attributed to cytokine storm, viral myocarditis as well as ischemia due to microvascular and macrovascular dysfunction [2].

Herein, we present a 57-year-old male, known case of hypertension and dyslipidemia, under treatment with losartan and atorvastatin, presented with dyspnea and dry cough for 5 days. Low-grade fever (oral temperature of 37.9 °C), stable vital signs without hypoxemia or respiratory distress, was observed, so he was discharged after describing alarm signs. After 10 days, he presented with complaint of retrosternal chest pain and dyspnea for 2 days. He was afebrile, ill and agitated with hypoxemia (SPaO₂ of 77%), tachypnea (respiratory rate: 28/min), tachycardia (HR:115 bpm), hypotension (blood pressure: 90/65), cold sweat and rales in half of the lung fields, representing acute pulmonary edema. Electrocardiogram revealed sinus tachycardia with mild dynamic ST segment depression in precordial leads. Oxygen therapy via nasal mask, intravenous furosemide, inotrope, anticoagulant, and oral antiplatelets was administered while chest CT scan demonstrated ground glass opacities

compatible with COVID-19 and congestion (Fig. 1a). No significant coronary artery stenosis was observed during catheterization (Fig. 1b), but transient bradycardia led to temporary pace maker insertion. Transthoracic echocardiography showed akinesia in mid-to-apical segments of both ventricles compatible with biventricular apical ballooning syndrome (Takotsubo cardiomyopathy) (Fig. 1c,d, Supplementary Video 1). Laboratory data showed positive oropharyngeal swab for CoV-2, lymphopenia (952 per μ L) without leukocytosis (6800 per μ L), elevated cardiac troponin I (183 ng/L, normal < 50) and C-reactive protein (18.9 mg/L, normal < 5). His clinical course was unremarkable with mild biventricular residual systolic dysfunction on 6 week follow-up.

Stress cardiomyopathy also known as Takotsubo cardiomyopathy or apical ballooning syndrome is known as acute coronary syndrome presentation accompanied by dynamic ST-segment changes and myocardial dysfunction in a non-coronary distribution (3). Takotsubo cardiomyopathy has been reported during SARS-CoV₂ infection in the setting of preexisting coronary artery disease along with normal coronary angiogram [3, 4]. Male predominance was proposed in Takotsubo cardiomyopathy associated with COVID-19, despite usual forms of stress cardiomyopathy [5].

This biventricular variant of Takotsubo cardiomyopathy in a male patient is a unique form of cardiac injury during COVID-19 disease with favorable outcome.

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Declarations

Conflict of interest Azin Alizadehasl, Azam Soleimani, Mohammad Mehdi Peighambari, Atousa Mostafavi declare that they have no conflict of interest.

Human rights statements All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1964 and later versions.

Fig. 1 **a** Mid thoracic slice of chest CT scan showed ground glass peripheral opacities accompanied by congested lung tissue. **b** Left coronary angiography revealed normal epicardial coronary arteries,

pace maker lead is seen in RV apex too. **c, d** Transthoracic apical four and two chamber views show akinesia in mid to apex of both ventricles associated with ballooning appearance

Informed consent Informed consent was obtained from the patient for being included in the study.

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