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 website.

The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC: Heart Failure* author instructions page.

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Acute Myocarditis With Ventricular Noncompaction in a COVID-19 Patient

As highlighted by Nianguo Dong (1), coronavirus disease-2019 (COVID-19) can cause myocardial injury. Putative mechanisms are acute coronary syndrome, demand ischemia, microvascular ischemic injury, cytokine dysregulation, or myocarditis (2). However, myocardial injury does not always correlate exclusively with COVID-19, and it may also reveal incidental cardiomyopathy. Thus, physicians must stay alert, and careful causative assessment is required in these patients. As contagious risk and patient instability may preclude initial work-up, reassessing the patient after the acute phase is of utmost importance. To illustrate, we report the case of an acute myocarditis with an underlying isolated ventricular noncompaction (IVNC) in a COVID-19 patient with heart failure.

A 27-year-old male without medical history was admitted for respiratory distress, and COVID-19 was diagnosed. High-sensitivity troponin I and N-terminal pro-B-type natriuretic peptide concentrations were elevated (100 ng/l and 9,300 pg/ml, respectively) suggesting myocardial involvement. In addition, echocardiography revealed an enlarged left ventricle with impaired left ventricle ejection fraction (LVEF) of 20%. The patient improved on a regimen of high-dose diuretic agents and noninvasive ventilation. He was discharged 9 days later with prescriptions for bisoprolol, furosemide, and spironolactone. One month later, his LVEF improved to 40% and showed normal filling pressure and cardiac output. Echocardiography revealed a 2-layered structure of the myocardium suggesting IVNC (3). CMR confirmed the IVNC diagnosis but also revealed acute myocarditis (**Figure 1**). Coronary computed tomography angiography results ruled out coronary artery disease. Patients with IVNC have variable prognosis, ranging from a prolonged asymptomatic course to severe cardiac disability. Prognosis is worse in patients hospitalized for heart failure, New York Heart Association functional classes III to IV, lower LVEF, and elevated LV filling pressures (4). Management involved treating the patient's heart failure, and genetic screening of both the patient and family members was conducted.

In this case, reassessing the patient after the COVID-19 acute phase allowed us to diagnose an underlying severe case of IVNC and subsequently initiate appropriate treatment and follow-up.

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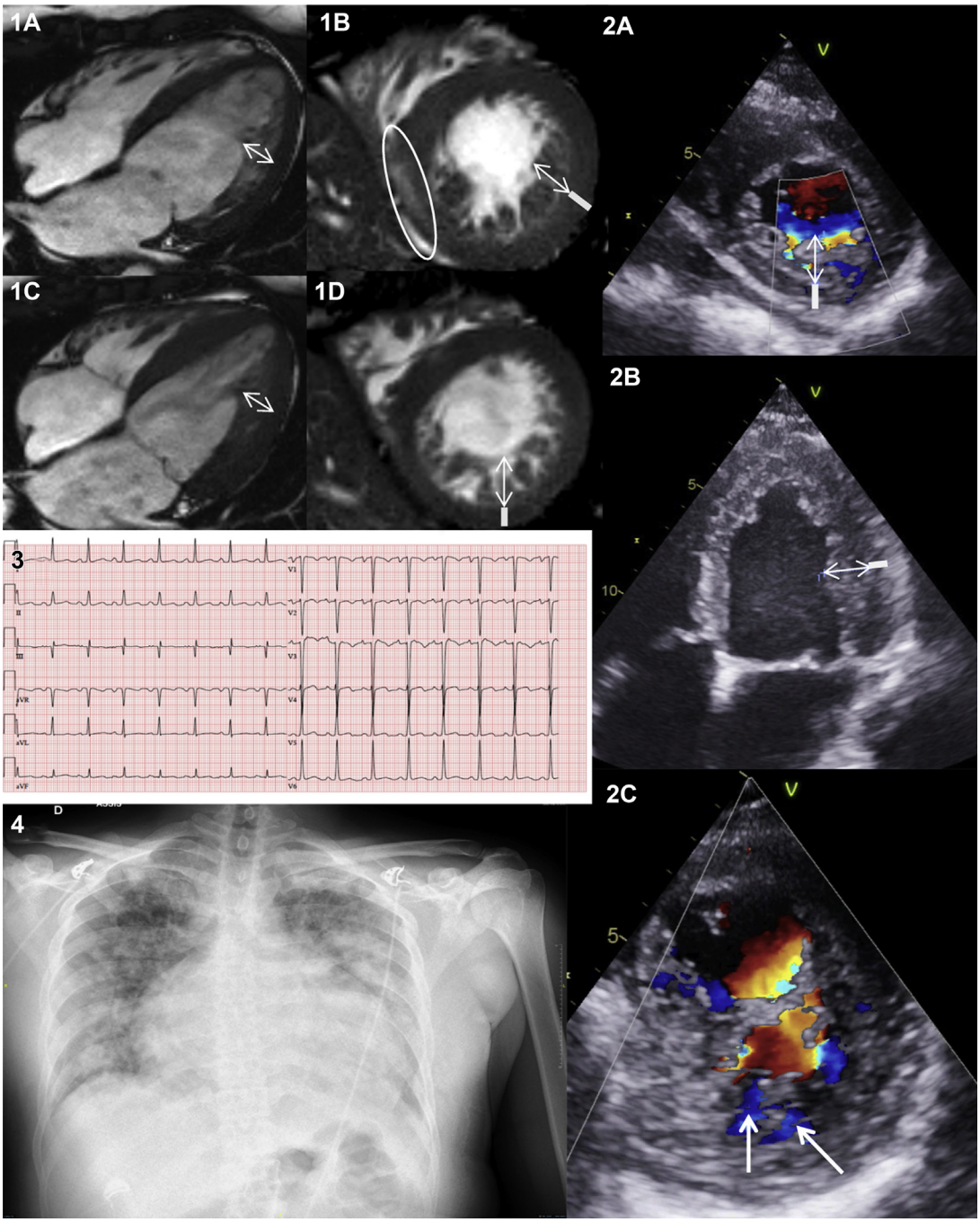
FIGURE 1 Multimodal IVNC and Myocarditis



CMR. **(1A and 1C)** Four-chamber and **(1B and 1D)** middle short-axis views. Subepicardial late gadolinium enhancement in cine T2- or T1-weighted images suggesting acute myocarditis **(open circle)**. Double-layered myocardium with a thin compacted epicardial layer **(open line)** and a thicker noncompacted endocardial band **(double arrows)** consisting of trabecular recesses suggesting IVNC. Transthoracic-echocardiography. **(2A and 2B)** Noncompacted endocardial layer **(double white-arrows)** and compacted layer **(white lines)**. **(2C)** Trabecular recesses deeply perfused in color Doppler **(open arrows)**. Electrocardiogram. **(3)** Sinus tachycardia, Q-wave and T-wave inversion in V_1 to V_3 leads, left axis deviation. Chest radiography. **(4)** Bilateral consolidation and significant heart enlargement. CMR = cardiac magnetic resonance; IVNC = imaging of ventricular noncompaction.