CLINICAL VIGNETTE

Fulminant herpes virus 6 myocarditis with complete atrioventricular block successfully treated with extracorporeal life support and ganciclovir

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Acute fulminant myocarditis is an inflammatory condition accompanied by hemodynamic compromise and associated with a high mortality rate. It is primarily caused by various infectious agents (mainly viruses, including the novel severe acute respiratory syndrome coronavirus 2 [SARS-CoV-2]).[1] In children, human herpes virus 6 (HHV-6) has been widely reported in fulminant myocarditis and dilated cardiomyopathy.[2]

A 5.5-year-old girl was admitted to the tertiary pediatric hospital with high fever, overwhelming weakness, and signs of peripheral hypoperfusion. Her electrocardiogram showed complete atrioventricular block with a variable ventricular rhythm at 80–220 bpm/min (FIGURE 1A and 1B). On echocardiography, the right ventricle (RV) was significantly enlarged, and the diameter of the left ventricle (LV) was normal. The contractility of both ventricles was globally decreased with ejection fraction (EF) of 30% to 35%. Laboratory tests yielded the following results on admission: troponin I, 160 000 ng/l (reference range <19 ng/ml); creatine kinase–myocardial band, 141 U/l (reference range <5.1 ng/ml); N-terminal fragment of the prohormone brain natriuretic peptide, 128 500 pg/ml (reference range <125 pg/ml); blood urea nitrogen, 95 mg/dl (reference range, 15–36.4 mg/dl); and creatinine, 1.4 mg/dl (reference range, 0.2–0.4 mg/dl). Despite primary treatment with inotropes and diuretics, the patient developed cardiogenic shock and multiorgan failure: aspartate aminotransferase 2373 U/l; alanine aminotransferase, 1156 IU; blood urea nitrogen, 161 mg/dl; creatinine, 2.4 mg/dl. She was intubated and sedated, received mechanical ventilation, as well as adrenaline and immunoglobulins were added. Computed tomography angiography confirmed an unobstructed coronary flow. Endocavital stimulation was unsuccessful. Due to progressive clinical and echocardiographic deterioration (EF, 14%; FIGURE 1C–1E), arteriovenous extracorporeal membrane oxygenation (ECMO) through sternotomy was used. Macroscopically, the myocardium was cyanotic (FIGURE 1F), biopsy of the right atrial myocardium revealed massive necrosis accompanied by hemorrhage and lymphocyte infiltration. A polymerase chain reaction test confirmed HHV-6 infection, so a 15-day ganciclovir therapy was started. The dose of inotropes was decreased stepwise. A slight improvement in LV contractility with decreased RV function was observed on echocardiography. Electrocardiography showed persisting complete atrioventricular block and signs of prior RV myocardial necrosis.

On day 10, the patient was successfully weaned off from ECMO, and milrinone and adrenaline were continued. Sedatives were slowly tapered. On day 21, the girl was extubated, and received respiratory support with high-flow nasal cannula. Intravenous treatment was gradually replaced with oral carvedilol, captopril, sildenafil, and aldactone. Therapy was continued in the cardiology department. Glucocorticoids were used. Ten months later, the LV was normal, with EF of 50%, and the RV remained

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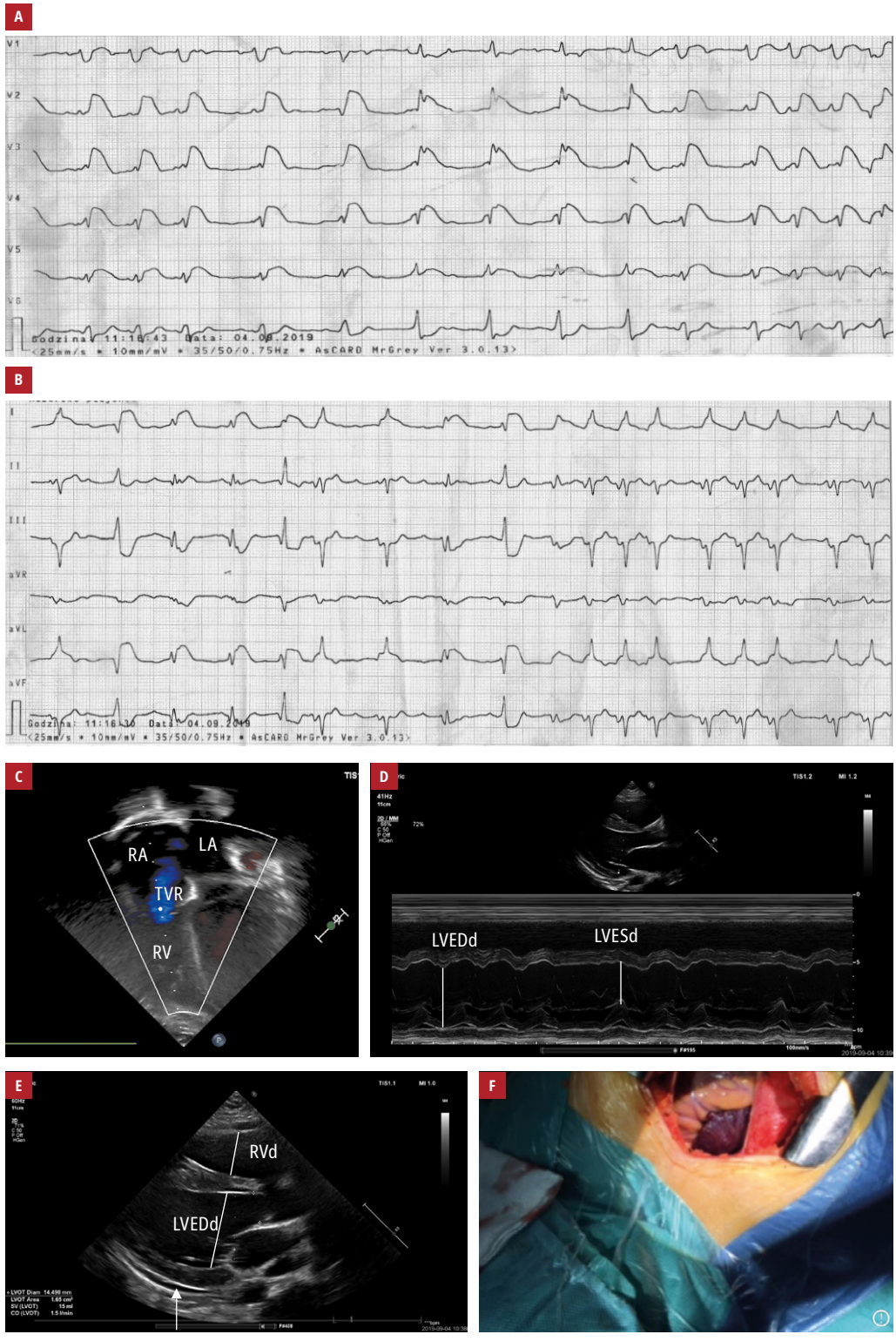

FIGURE 1 **A**, **B** – electrocardiograms showing sinus tachycardia and short runs of atrial tachycardia with variable atrioventricular conduction and signs of atrioventricular block, escape beats likely originating in the junction, and an extensive ST-segment elevation; **C** – 4-chamber view showing the dilated right atrium and both ventricles and tricuspid valve regurgitation; **D**, **E** – M-mode (**D**) and 2-dimensional (**E**) long-axis views showing dilated ventricles, dyskinesia, and paradoxical interventricular movement as well as decreased cardiac output and hydropericardium (white arrow); **F** – cyanotic myocardium of the right atrium
Abbreviations: LA, left atrium; LVEDd, left ventricular end-diastolic dimension; LVESd, left ventricular end-systolic dimension; RA, right atrium; RV, right ventricle; RVd, right ventricular diastolic dimension; TRV, tricuspid valve regurgitation

enlarged: the RV end-diastolic volume index was 153 with EF of 28%. Complete heart block persisted at a ventricular rate of 70 bpm. Despite that, the girl's medical status was generally good. She showed good exercise tolerance and no neurological deficits.

In fulminant myocarditis with progressive cardiogenic shock, ECMO is a life-saving procedure and intravenous immunoglobulins are commonly administered.[3] When viral etiology is confirmed, antiviral therapy can be beneficial. There have been incidental clinical reports suggesting that ganciclovir effectively improves myocardial function in cytomegalovirus myocarditis.[4] There is little evidence indicating that ganciclovir can abolish viral transcription also in the case of HHV-6–induced heart failure.[5] In our patient, despite the presence of laboratory and histological markers of massive myocardial necrosis, a relatively quick recovery was achieved. Ganciclovir can also be an efficient adjunct in the management of HHV-6 myocarditis.

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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