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Case report

Successful treatment of fulminant myocarditis with biventricular mechanical circulatory support: A two-year follow-up

Jiri Maly [a,*], Zora Dorazilova [a], Milos Kubanek [b], Ivan Netuka [a,d], Martin Pokorny [a,c], Josef Besik [a], Jan Burkert [c], Ondrej Szarszoi [a]

[a] Department of Cardiovascular Surgery, Institute for Clinical and Experimental Medicine, Prague, Czech Republic
[b] Department of Cardiology, Institute for Clinical and Experimental Medicine, Prague, Czech Republic
[c] 2nd Faculty of Medicine, Charles University and University Hospital Motol, Prague, Czech Republic
[d] 2nd Department of Surgery, Department of Cardiovascular Surgery, First Faculty of Medicine, Charles University in Prague, Prague, Czech Republic

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ABSTRACT

Fulminant myocarditis (FM) is an inflammation of the myocardium characterized by progressive acute heart failure leading to cardiogenic shock that develops over several hours. In this article, we present a case of a female patient with acute fulminant lymphocytic myocarditis who was successfully treated with biventricular MCS.

1. Introduction

Fulminant myocarditis (FM) is an inflammation of the myocardium characterized by progressive acute heart failure that develops over several hours or days [1–3]. Compared with acute myocarditis, following differences are identified in FM, in addition to the rapid progression of the heart failure: common signs of viral infection preceding the manifestation of the disease by 2–4 weeks, more frequent disorders of the ventricular conduction, atrioventricular (AV) blocks and ventricular arrhythmias; laboratory results showing higher levels of cardiac-specific enzymes; and more frequent hepatorenal dysfunction. Echocardiography reveals the characteristic non-dilated left ventricle with thicker walls and reduced ejection fraction [4]. Diagnosis of FM by means of coronary angiogram excludes acute coronary syndrome, and the use of an endomyocardial biopsy (EMB) identifies and differentiates forms that are poorer in terms of prognosis, such as giant cell or necrotizing eosinophilic myocarditis, from benign forms such as acute lymphocytic or hypersensitivity myocarditis [3,5].

* *Corresponding author*. Department of Cardiovascular Surgery, Institute for Clinical and Experimental Medicine (IKEM), Vídeňská 1958/9, 140 21 Prague 4, Czech Republic. Tel.: +420 261 365 014; fax: +420 261 081 362.
E-mail addresses: jiri.maly@ikem.cz (J. Maly), mpok@medicon.cz (M. Pokorny).

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The prognosis of fulminant lymphocytic and hypersensitivity myocarditis has been significantly improved by the new developments in the drug treatment of acute heart failure and/or the implantation of a mechanical circulatory support (MCS) device, since, after the initial critical phase had been managed, these FMs have a benign prognosis, leading most commonly to complete recovery. In this report, we present a case of a female patient with acute fulminant lymphocytic myocarditis who required biventricular MCS in the initial phase of cardiogenic shock, accompanied by unstable ventricular tachycardia.

2. Case report

A 53-year-old, non-smoking female patient with a body mass index (BMI) of 27 kg/m^2, a history of thyroid disease, and no other significant medical history was referred to our clinic with cardiogenic shock accompanied by persistent ventricular tachycardia episodes. The patient was admitted to the respective cardiac clinic on February 17, 2011, at 3:00 p.m. in a precollapse state following 2 days of viral infection, with overall malaise, difficult breathing, and chest pain. The initial electrocardiogram (ECG) showed sinus rhythm with heart rate of 84 beats/min, left anterior hemiblock, right bundle branch block, and a higher-degree intermittent AV block; laboratory results detected positive troponin I levels (>40 μg/L). Coronary angiography, performed to exclude a coronary event, showed regular findings. However, the transthoracic echocardiogram (TTE) showed dysfunction of the non-dilated left ventricle (LV): LV end-diastolic diameter (LVEDD) = 52 mm; wall thickness of interventricular septum (IVS) = 10 mm; posterior wall thickness = 11 mm; ejection fraction (LVEF) = 35% and signs of dyssynchrony. The patient was transferred to our facility on February 18, 2011, at 2:00 p.m. because of cardiogenic shock, requiring a combination of inotropic support (norepinephrine 0.1 μg/kg/min, dobutamine 5 μg/kg/min), ongoing slow ventricular tachycardia of 130/min (Fig. 1), and incipient alteration of organ function. The patient was conscious, short of breath at rest, hypotensive (85/40 mmHg) and displayed signs of peripheral vasoconstriction. Chest fluoroscopy demonstrated signs of interstitial lung edema and non-dilated heart shadow. Echocardiography revealed reduced systolic LV function (LVEF = 20%, LVEDD = 56 mm, IVS = 9 mm), moderate functional mitral regurgitation, slightly reduced right ventricle (RV) function, and increased filling pressures in both ventricles. Laboratory results revealed significantly increased levels of B natriuretic peptide (BNP) (1595 ng/L) and troponin I (45.9 μg/L), aspartate aminotransferase (AST) = 4.72 μkat/L, alanine transaminase (ALT) = 1.95 μkat/L, creatinine = 80.4 μmol/L, urea = 10.0 mmol/L, C-reactive protein (CRP) = 30.2 mg/L, glycemia = 7.9 mmol/L, hemoglobin (Hgb) = 117 g/L, and lactate = 2.2 mmol/L. The condition was diagnosed as FM with a rapid progressive syndrome of low cardiac output, and the urgent implantation of short-term mechanical cardiac support (MCS) was indicated. The CentriMag ventricular assist system (Levitronix LLC; Waltham, Mass) was used.

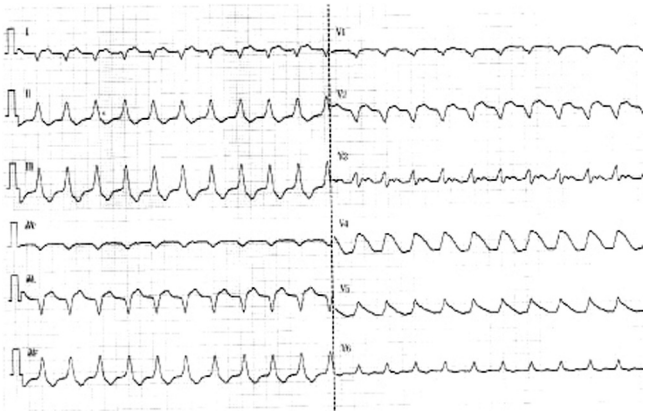

Fig. 1 – ECG at admission showing ventricular tachycardia 130/min.

The patient was transferred to the operating room at 4:00 p.m. in critical condition. Paroxysmal supraventricular tachycardia (SVT) (150/min.) resistant to repeated defibrillation occurred following anesthesia. Critical systemic hypotension required indirect and, following longitudinal sternotomy, direct heart massage through the anterior mediastinum. Bolus injections of norepinephrine were administered to maintain at least a minimum perfusion pressure. After heparin administration, extracorporeal circulation was introduced in a standard way. Vascular prostheses (Vascutek 8) were applied first to ascending aorta and then to the pulmonary artery; two tobacco-pouch sutures with pericardial pads were applied to free walls of both atria. Cannulas of the CentriMag left and right ventricular assist device (LVAD and RVAD, respectively) were introduced and the systems were voided of air. Gradually, the activities of both CentriMag devices were initiated (LVAD, 3700 rpm with cardiac output (CO) ≈ 5.5 L/min; RVAD, 3600 rpm with CO = 4.0–4.5 L/min). Extracorporeal circulation was used for 115 min, and protamine was administered; possible bleeding sources were reviewed; hemostasis was achieved; 3 drains were introduced; definite suture was postponed; vasopressor support by means of norepinephrine oscillated at approximately 0.3 μ/kg/min; and maximum lactate level was 6.3 mmol/L. Bleeding revision was performed on postoperative day (POD) 1, definite suture on POD 2, and extubation on POD 3 after discontinuation of inotropic support.

Myocardial RV biopsy confirmed acute lymphocytic myocarditis (20 lymphocytes/mm^2) accompanied with plaques of myonecroses and minimum interstitial necrosis (Fig. 2).

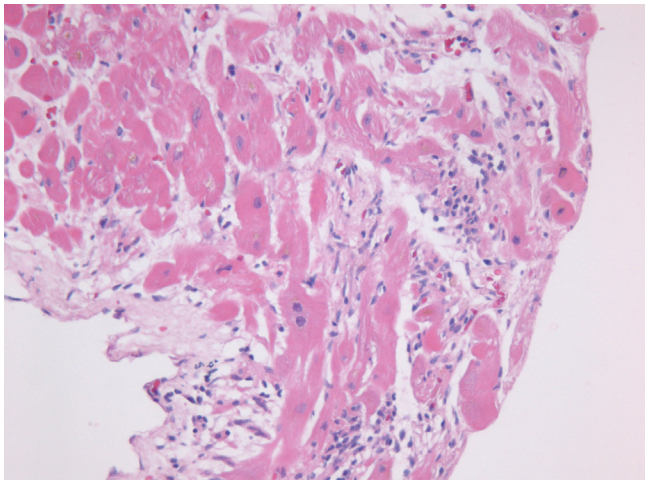

Fig. 2 – Biopsy showing interstitial inflammatory infiltrate.