Journal of Clinical and Medical Images

Case Report

ISSN: 2640-9615 | Volume 6

Complete Atrio-Ventricular Block Secondary to Focal Acute Myocarditis

Francesco Rotondi[1], Felice Nappi[1], Francesca Esposito[1*], Francesco Urraro[1], Fiore Manganelli[1], Paolo Vitillo[1] and Emilio Di Lorenzo[1]

[1]Department of Cardiology and Cardiovascular Surgery «D. Rotiroti» Avellino, Italy

[*]Corresponding author:

Francesco Rotondi,
Department of Cardiology and Cardiovascular Surgery «D. Rotiroti» Avellino, Italy,
Tel. +39.0825.203240,
E-mail: francesco.rotondi@tin.it

Received: 08 May 2022
Accepted: 30 May 2022
Published: 04 Jun 2022
J Short Name: JCMI

Citation:

Francesco Rotondi, Complete Atrio-Ventricular Block Secondary to Focal Acute Myocarditis. J Clin Med Img. 2022; V6(10): 1-3

Keywords:

Atrio-ventricular block; Acute myocarditis; Cardiac magnetic resonance

1. Abstract

We present a case of a 24 -year-old man with syncope due to transient complete atrio-ventricular block secondary to acute myocarditis involving only basal anterior interventricular septum. Atrio-ventricular block occurred with a localized left ventricular dysfunction assessed by cardiac magnetic resonance. Previous reports suggest that serious conduction disturbances in acute myocarditis are associated with severe myocardial necrosis. Conversely, in our patient only magnetic resonance allow us to detect a focal myocardial injury that may account for the transient disturbance of conduction system. The implantation of a permanent pacemaker in this setting may be delayed because of potentially reversible myocardial damage.

2. Introduction

High-degree heart block is an uncommon manifestation of acute myocarditis in adults with a normal left ventricular systolic function [1]. Here we present a case of a 24 -year-old man with syncope due to transient complete atrio-ventricular block (CAVB) requiring a temporary pacemaker implant, secondary to localized acute myocarditis involving only basal anterior interventricular septum, the myocardial region where atrio-ventricular conduction system is located.

3. Case Report

A 24-year-old man was transported to the emergency department of our hospital because he experienced a syncope. He did not report any symptoms except for a fever 3 day earlier. On examination, his heart rate was 32 beats per minute (bpm), blood pressure was 100/60 mmHg, oxygen saturation was 99 %, and body temperature 36.3°C. A 12-lead electrocardiogram (ECG) revealed a CAVB with wide QRS escape and ventricular rate of 30 bpm (Figure 1a).

Chest radiography was normal and transthoracic echocardiography found an ejection fraction of left ventricle = 50% without regional dyssynergy.

All laboratory-test results were normal except for a mild elevation in Troponin I levels (peak 0,95 ng/ml; nv = 0,00-0,30 ng/ml). A temporary transvenous pacemaker, programmed in VVI mode at a rate of 60 bpm, was placed in the right ventricle and the patient was admitted in coronary intensive care unit. Therapy with aspirin and omeprazole was started. Only in the following days appeared leucocytosis and a slight elevation of VES, PCR and NT-proBNP.

Five days after admission, ECG demonstrates complete resolution of CAVB (Figure. 1b). Cine cardiac magnetic resonance showed a slightly reduced global function of left ventricle (ejection fraction =47%) and a localized akinesia of the basal anterior interventricular septum (Figure 2). Gadolinium-enhanced imaging detected a focal late enhancement in the subepicardial layer of basal anterior septal wall, suggestive for acute focal myocarditis (Figure 3).

Predischarge 24-hour Holter monitoring did not show any significant hyperkinetic and /or hypokinetic arrhythmia. Fifteen days later he was still asymptomatic and was discharged. At three-month follow-up patient was completely asymptomatic without any complication or adverse event.

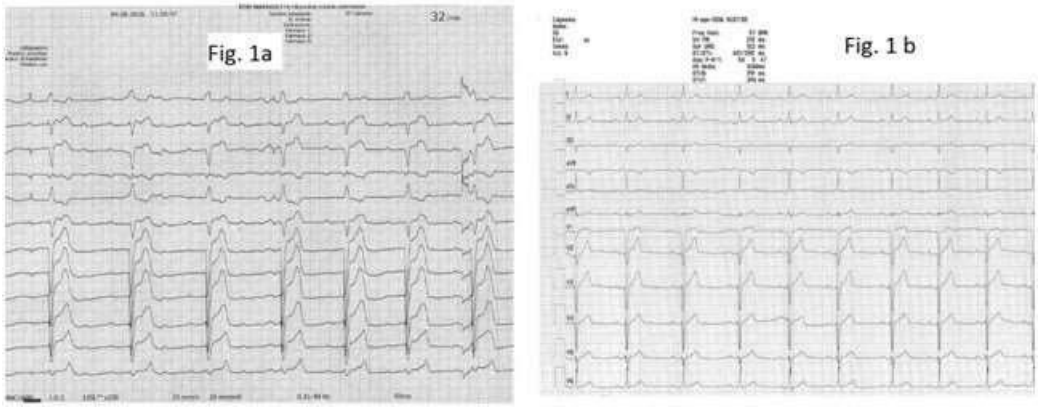

Figure 1. complete atrio-ventricular block (CAVB) with wide QRS escape and ventricular rate of 30 bpm (1a). Five days after admission, ECG demonstrates complete resolution of CAVB (1b).

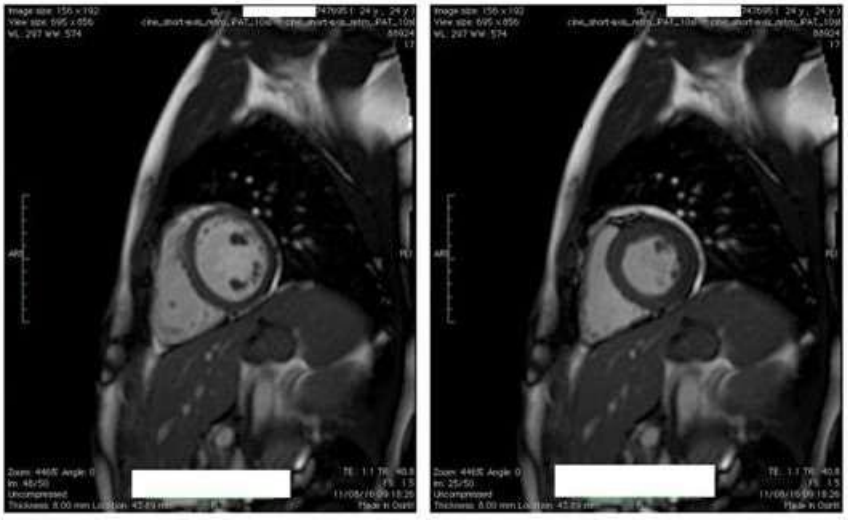

Figure 2. Cine cardiac magnetic resonance showed a slightly reduced global function of left ventricle (ejection fraction = 47%) and a localized akinesia of the basal anterior interventricular septum.

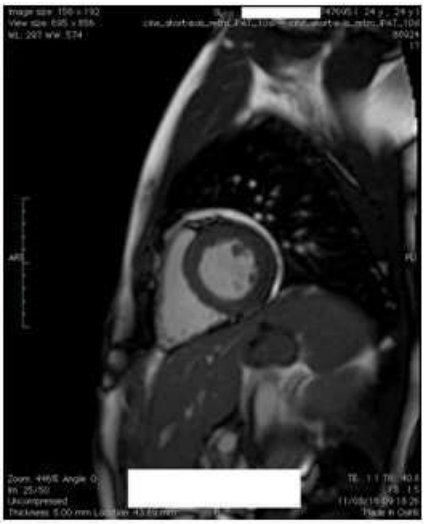

Figure 3. Gadolinium-enhanced imaging detected a focal late enhancement in the basal anterior septal wall, suggestive for acute focal myocarditis.

4. Discussion

Acute myocarditis (AM) is an inflammatory disease of the myocardium. There are several causes of AM, including viral infection, toxins, drugs, hypersensitive immune reactions. AM is a challenging diagnosis due to the heterogeneity of clinical presentations [2,3]. The most characteristic feature of our case was that AVCB occurred with a localized left ventricular dysfunction assessed by cardiac magnetic resonance. Previous reports suggest that advanced conduction disturbances in acute myocarditis are associated with extensive myocardial necrosis [4,5]. Conversely, in our patient, CMR detected a focal myocardial injury that may account for the transient disturbance of atrio-ventricular conduction system. Although endomyocardial biopsy (EMB) is the "gold standard" for definite diagnosis of AM, its use on a widespread basis is not feasible in routine clinical practice. Due to limited availability and procedural risks of EMB, current guidelines recommend its use only in selected clinical scenarios. Moreover, data are lacking as far as concerns EMB adjunctive value in defining therapy and patient prognosis. In our patient CMR, due to its unique ability of an "in vivo" tissue characterization, was able to highlight the presence of kinetics' abnormalities and inflammatory correlates in the interventricular septum, likely the substrate of the affected conduction system. Therefore, a sudden appearance of a cardiac conduction disorder in a young patient should rise the clinical suspicion of myocarditis even in the absence of a large left ventricular dysfunction. Furthermore, our case suggests that the implantation of a permanent pacemaker in this setting may be delayed because of potentially reversible myocardial damage in AM.

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