Brugada-Like ECG Associated with Primary Cardiac Lymphoma

Nobuaki Sato, Masahiro Shimokawa, Hiromi Iwasaki, Toru Maruyama* and Koichi Akashi

Department of Medicine and Bioisysmec Science, Kyushu University Graduate School of Medical Sciences, Fukuoka, 812-8582, Japan

Abstract

A 64-year-old female patient with primary cardiac lymphoma showed ECG mimicking Brugada syndrome. Lymphoma invaded the right atrial and ventricular outer layer and projected to the right ventricular outflow tract (RVOT). ECG restored normal after chemotherapy and subsequent stem cell transplantation. This case indicates that cardiac lymphoma invading the outer layer of RVOT induced reversible Brugada-like ECG.

Keywords: Brugada-like ECG; Cardiac lymphoma; Right ventricular outflow tract

Introduction

Brugada-like electrocardiogram (ECG) is observed in hematological diseases and mediastinal solid tumor [1,2]. However, primary cardiac but not mediastinal tumor demonstrating Brugada-like ECG pattern is extremely rare. Here, we report a case of primary cardiac lymphoma (PCL) showing ECG findings similar to those described in Brugada syndrome, and consider the electrocardiographic and morphological correlation.

Case Report

A 64-year-old female complained of fatigue, palpitation and pitting edema. Her blood examination demonstrated no specific abnormalities except for elevation of LDH (426 IU/L) and soluble IL-2 receptor (7015 U/ml). ECG showed sinus rhythm with right axis, clockwise rotation except for elevation of LDH (426 IU/L) and soluble IL-2 receptor (7015 U/ml). ECG showed sinus rhythm with right axis, clockwise rotation except for elevation of LDH (426 IU/L) and soluble IL-2 receptor (7015 U/ml). Computed tomography suggested massive intracardiac tumor invading the right atrial and ventricular wall and projecting to the right ventricular outflow tract (RVOT). Histologic diagnosis of diffuse large B-cell lymphoma was obtained by tumor biopsy under intracardiac ultrasound guidance [3]. The patient was referred to our Department for treatment of PCL. The ECG abnormalities described above progressed furthermore (B), indicating rapid PCL proliferation. Therefore, combination chemotherapy with rituximab, pirarubicin, cyclophosphamide, oncovin and prednisolone (R-THPCOP) was started. In addition, autologous peripheral blood stem cell transplantation was conducted after the completion of 6 courses of R-THPCOP regimen. Two years after transplantation, there has not been any recurrence of PCL and ECG does not show any specific abnormalities (C) (Figure 1).

Discussion

Primary cardiac tumor is rare, showing an incidence of approximately 0.02% on accumulated autopsy series [4], and PCL accounts for only 1% of these tumors [5]. PCL sometimes alters the cardiovascular morphology. In this case, ECG demonstrated marked and progressive cardiac axis deviation and clockwise rotation (A, B). PCL tissue is not electrically excitable, and reversible right axis deviation implies massive PCL invading a significant amount of the right ventricle. Computed tomography demonstrated marked cardiac distortion around the longitudinal axis due to PCL proliferation, which was indicated by the reversed position of the ascending aorta and main pulmonary artery (D). This is assumed to be the main reason for the marked and reversible clockwise rotation.

Brugada-like ST-segment elevation is ascribed to the transmural voltage gradient across the RVOT due to differences in native cardiac action potential configuration. In this case, PCL involved outer RVOT (D, E), and the electrical force generated by the inner remaining RVOT may have been reflected by right precordial ECG. Moreover, expanding PCL resulted in a systolic blood pressure gradient (28 mmHg) and probably mechanical wall stress in RVOT (E).

In conclusion, this case of PCL demonstrated a reversible Brugada-like ECG. As a possible explanation for these phenomena, PCL is considered to cause a transmural voltage gradient and mechanical stress in the RVOT.

References


*Corresponding author: Toru Maruyama, Department of Medicine and Bioisysmec Science, Kyushu University Graduate School of Medical Sciences, Higashi-ku, Fukuoka, 812-8582, Japan, Tel: 81-92-642-5235; Fax: 81-92-642-5247; E-mail: maruyama@hs.kyushu-u.ac.jp

Received December 24, 2010; Accepted March 07, 2011; Published March 09, 2011


Copyright: © 2011 Sato N, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

