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Case Report

Ablation of idiopathic ventricular fibrillation triggered by ventricular premature beat originating from myocardium of right ventricle: Case report

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ABSTRACT

We report a case of a 55-year-old woman with idiopathic ventricular fibrillation (VF) who suffered from recurrent implantable cardioverter-defibrillator shocks triggered by short coupled ventricular premature beat (VPB). This VPB was mapped and ablated from the myocardium of right ventricle close to the lateral tricuspid annulus.

<Learning objective: Triggering ventricular premature beat (VPB) in idiopathic ventricular fibrillation was reported to originate from the myocardium of right ventricular outflow tract or from Purkinje system. In this case, the origin of triggering VPB is the myocardium of right ventricle close to the lateral tricuspid annulus.>

Introduction

Idiopathic ventricular fibrillation (VF) is defined as a spontaneous VF in the absence of any congenital or acquired structural or electrical heart disease. Idiopathic VF can be caused by short coupled ventricular premature beat (VPB) [1,2]. We present a case of idiopathic VF triggered by VPB from an unusual origin.

Case report

A 55-year-old woman was admitted to intensive coronary care unit after out of hospital resuscitation due to VF. She was treated by mechanical ventilation for three days and hypothermia for the first 24 h. After three days, mechanical ventilation was gradually withdrawn. She was neurologically intact without cognitive impairment. Electrocardiogram (ECG) showed normal sinus rhythm with no evidence of acute ischemia, abnormal ST elevation, or early repolarization (Fig. 1a). QT interval was normal. Blood tests were normal. Echocardiography showed normal left ventricular and right ventricular (RV) function and normal valves. Coronary angiography revealed normal coronary arteries, and right ventriculography was normal. Flecainide test ruled out Brugada syndrome (Fig. 1b). Polymorphic ventricular tachycardia (PMVT) was induced during electrophysiological study (EPS) with pacing from RV apex at cycle length 400 ms and three extrastimuli. A diagnosis of idiopathic VF was made and a single lead implantable cardioverter-defibrillator (ICD) was implanted.

Two weeks after discharge, she was re-admitted because of recurrent ICD shocks. ICD interrogation showed frequent episodes of PMVT/VF preceded by monomorphic VPBs (Fig. 2a–c). Holter monitoring showed frequent (about 5% of heart beats distributed over 24 h) short-coupled monomorphic VPBs (coupling interval about 300–330 ms) and several episodes of non-sustained PMVT and episodes of VF preceded by the same VPB. All sustained arrhythmias were treated successfully by ICD shocks. Medical treatment including beta-blocker and amiodarone failed to suppress the VPBs or prevent VT recurrence. Thus, EPS was done in order to map and ablate the focus of these triggering VPBs.

Diagnostic and ablation catheters were inserted via the femoral vein and positioned in RV and His. Endocardial mapping of the RV was performed during VPBs using a 3.5-mm open irrigated tip catheter (Navistar Thermocool, Biosense-Webster, Diamond Bar, CA, USA) and Carto mapping system (Carto, Biosense-Webster). The earliest activation potential was recorded close to lateral tricuspid annulus 43 ms before QRS onset of the VPBs. No Purkinje potentials were recorded at this site (Fig. 3). The QRS morphology during pacing from that site showed a 12/12 match with the spontaneously occurring VPBs (Fig. 4). With the power control set at 35 W, radiofrequency energy was delivered at this site (Fig. 5). VPBs

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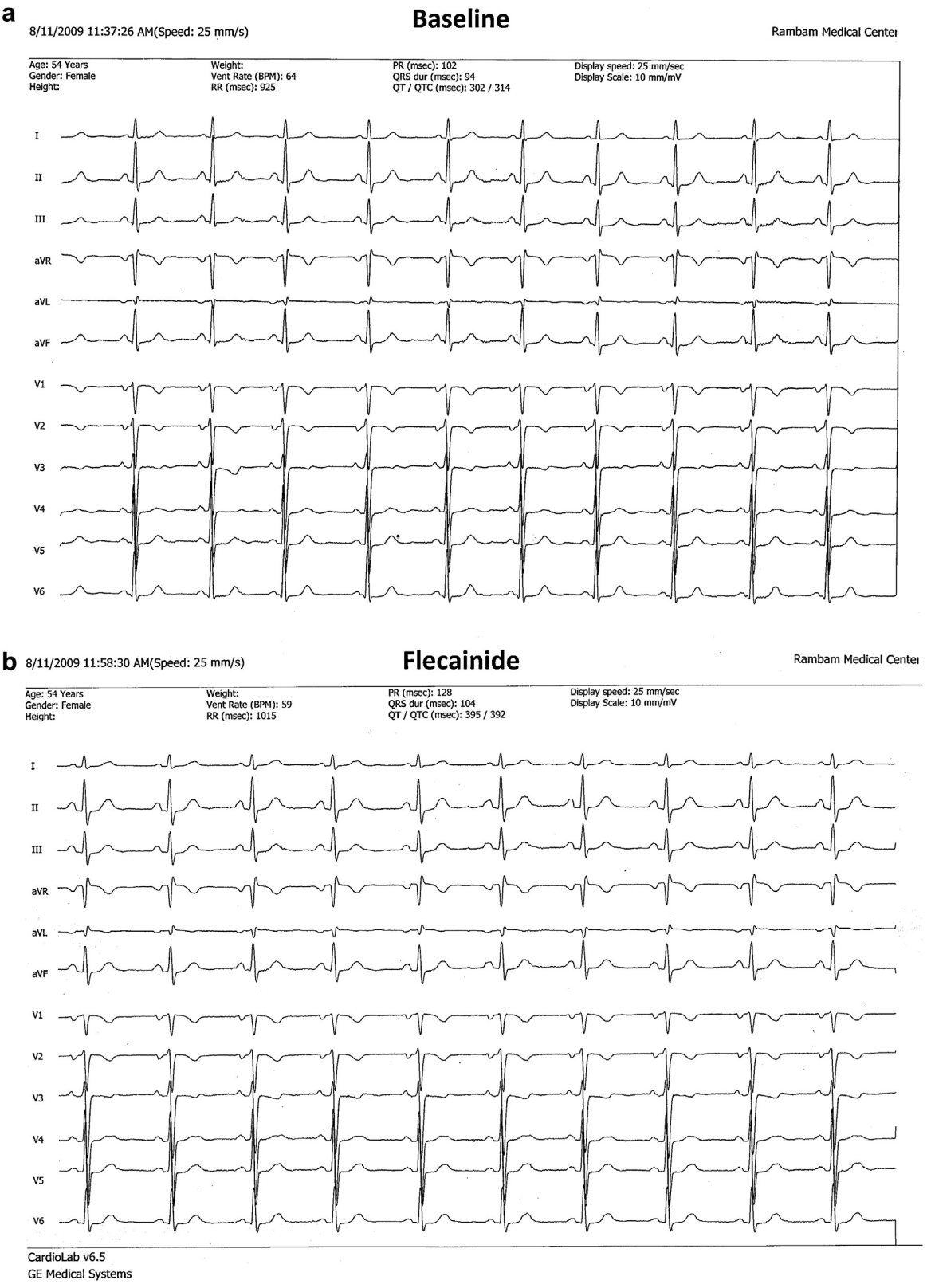

Fig. 1. (a) Electrocardiogram at baseline. (b) Flecainide challenge test (flecainide dosage was 150 mg) ruled out Brugada syndrome.