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CASE REPORT

Arrhythmic storm: Short-coupled variant torsade de pointes

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KEYWORDS
Polymorphic ventricular tachycardia;
Sudden cardiac death;
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Abstract A 49-year-old woman, with no relevant family history, was admitted in 1996 for arrhythmic storm with polymorphic ventricular tachycardia (torsade de pointes) which degenerated into ventricular fibrillation. Iatrogenic causes were excluded, the electrocardiogram (ECG) was normal and there was no structural heart disease. She refused cardioverter-defibrillator implantation. Treatment was begun with amiodarone, which she took irregularly.

She remained asymptomatic until 2014 when she was admitted for a new arrhythmic storm with torsade de pointes, refractory to antiarrhythmic therapy and aggravated by ventricular pacing (65 defibrillations). She had frequent ventricular extrasystoles (with short-coupled period <300 ms) preceding the tachycardia. After administration of isoprenaline infusion electric stability was maintained. In this setting and in the absence of structural heart disease or iatrogenic cause, a diagnosis of short-coupled variant torsade de pointes was established. A cardioverter-defibrillator was implanted and she was treated with verapamil, without recurrence of arrhythmias.

PALAVRAS-CHAVE
Taquicardia ventricular polimórfica;
Morte súbita cardíaca;
Variante *torsade de pointes* de acoplamento curto

Tempestade arrítmica: variante *torsade de pointes* de acoplamento curto

Resumo Mulher de 49 anos, sem história familiar de relevo. Em 1996 foi admitida no hospital por tempestade arrítmica: taquicardias ventriculares polimórficas tipo *torsade de pointes*, que degeneraram em fibrilhação ventricular. No estudo realizado foram excluídas iatrogenias, apresentava eletrocardiograma normal e ausência de cardiopatia estrutural. Recusou implantação de cardiodesfibrilhador. Ficou medicada com amiodarona, com cumprimento irregular. Assintomática até 2014, altura em que teve nova tempestade arrítmica com taquicardias ventriculares polimórficas, refratárias a antiarrítmicos e agravadas por *pacing* ventricular (65 desfibrilhações). Após introdução de isoprenalina em perfusão manteve estabilidade de

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ritmo, constatando-se extrassístoles ventriculares com período de acoplamento curto (<300 ms) a preceder as taquicardias. O diagnóstico de *short coupled variant torsade de pointes* foi estabelecido na ausência de iatrogenia ou cardiopatia estrutural. Implantou cardiodesfibrilhador e ficou medicada com verapamil, sem recorrência de eventos arrítmicos.

Case report

A 49-year-old woman, with no relevant family history and not taking any prescribed or illegal drugs, was admitted in 1996 for arrhythmic storm with polymorphic ventricular tachycardia (PVT) (torsade de pointes), with some episodes degenerating into ventricular fibrillation (VF) refractory to lidocaine, procainamide and propranolol and requiring several defibrillations. The 12-lead ECG was normal and electrolyte and metabolic abnormalities were excluded. Stabilization was achieved with amiodarone. During hospitalization she underwent transthoracic echocardiography (TTE) and cardiac catheterization, which were normal. Nonspecific characteristics were observed on endomyocardial biopsy. The electrophysiological study (EPS) performed under amiodarone (400 mg/day) was negative for inducible ventricular tachycardia. An implantable cardioverter-defibrillator (ICD) was proposed, but the patient refused. She was discharged under treatment with amiodarone and referred for an arrhythmia consultation.

During 18 years of follow-up she took amiodarone irregularly and with subtherapeutic doses (100 mg three times a week). Holter monitoring documented isolated premature ventricular contractions (PVCs), duplets and nonsustained PVT (all asymptomatic).

In 2014 she was readmitted for recurrent syncope due to PVT (torsade de pointes). The 12-lead ECG (Figure 1) and TTE were normal. Electrolyte and metabolic abnormalities and iatrogenic toxicological causes were excluded. She began intravenous therapy with magnesium sulfate and amiodarone without rhythm stabilization. Endotracheal intubation with mechanical ventilation was required in the context of multiple defibrillations. Lidocaine was added to amiodarone, also without success. A temporary pacemaker was implanted for ventricular pacing, but it proved to be arrhythmogenic (Figure 2A) and was promptly removed. Coronary angiography was normal. Veno-arterial extracorporeal membrane oxygenation (ECMO) was introduced because of hemodynamic instability, and removed after 24 hours due to lower limb ischemia. At this stage lidocaine was replaced by isoprenaline, maintaining amiodarone, with rhythm stabilization.

In an attempt to optimize therapy, isoprenaline was suspended at 48 hours and replaced by a beta-blocker in combination with amiodarone. This change triggered new episodes of nonsustained PVT. Isoprenaline was then

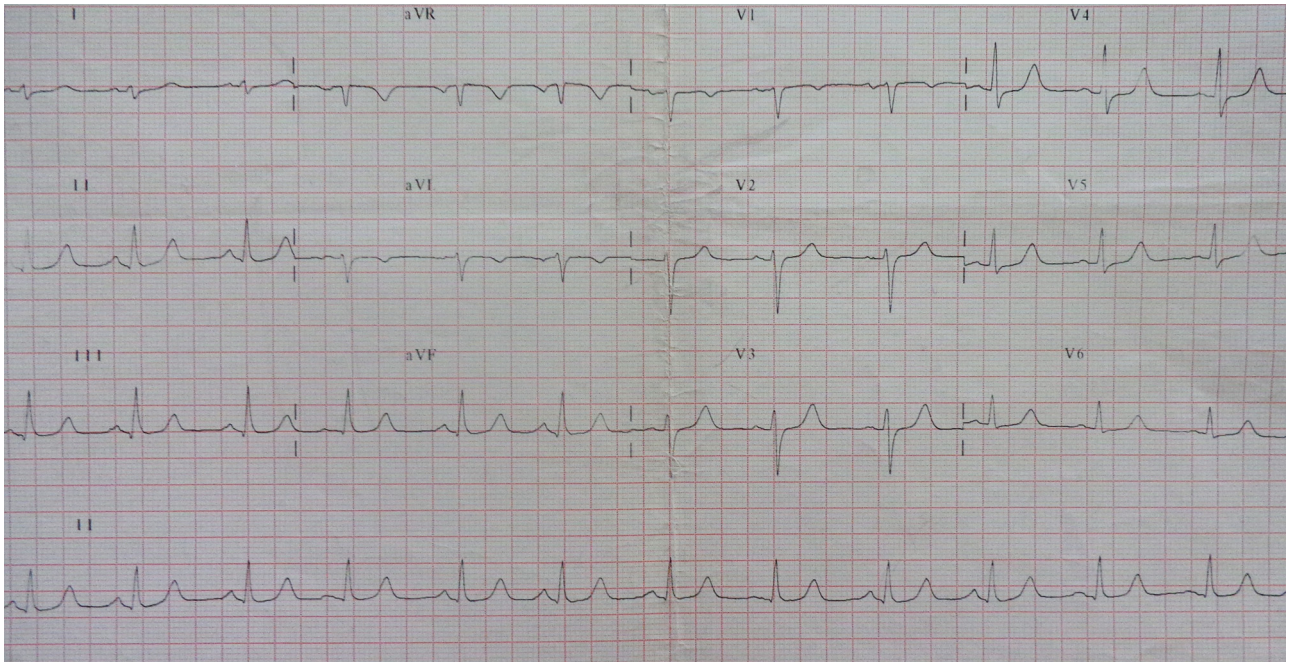

Figure 1 Normal ECG.