Recurrence of torsades de pointes after catheter ablation of incessant ventricular bigeminy in combination with QT prolongation

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A 45-year-old woman, who had received a single-chamber implantable cardioverter defibrillator (ICD) due to ventricular fibrillation 5 years ago, was admitted for catheter ablation of incessant right ventricular outflow tract bigeminy. After successful ablation recurrent torsades de pointes were associated with a prolonged corrected QT interval (QTc). The 12 lead ECG after ablation showed sinus bradycardia and multiple PVCs with various morphologies, couplets and triplets. Genetic testing revealed a heterozygous missense mutation in the SCN5A-gene (p.Arg190Gln, Exon 5), consistent with long QT syndrome 3. DDDR pacing following implantation of an atrial lead prevented further ventricular tachyarrhythmias.

Case report

In 2005, the patient had received a single-chamber ICD after having survived ventricular fibrillation of unknown cause in another hospital.

Since 2009, an almost incessant ventricular bigeminy despite therapy with bisoprolol occurred. Several Holter electrocardiograms (ECGs) demonstrated >40 000 monomorphic premature ventricular complexes (PVCs) per 24 h. Left ventricular ejection fraction (LV-EF) decreased to 40% and the patient suffered from dizziness, fatigue, and pre-syncope. In 2010, the patient experienced one adequate ICD shock and another non-sustained ventricular tachyarrhythmia was recorded in the ICD storage. The stored electrogams showed ventricular bigeminy with the same morphology as that of the initiating PVCs.

After gradual withdrawal of bisoprolol, the patient underwent electrophysiological study without sedation. Incessant ventricular bigeminy was present and cardiac repolarization was prolonged with a QT interval of 480 ms (Figure 1A). Programmed right ventricular stimulation with up to triple extrastimuli did not induce any ventricular tachyarrhythmia. Radiofrequency (RF) ablation in the right ventricular outflow tract (RVOT) eliminated the ventricular ectopy (one RF application, 60 s, 30 W). During a 60 min waiting period after ablation, no PVCs occurred without or with intravenous orciprenaline. The 12 lead ECG after ablation showed sinus rhythm at a heart rate of 63/min with a QTc interval of 520 ms (Figure 1B).

Bisoprolol (5 mg daily) was re-administered to the patient before discharge from the hospital. After 3 days, she was re-admitted due to an adequate ICD shock. ECG recordings showed sinus bradycardia and multiple PVCs with various morphologies, couplets and recurrent torsades de pointes requiring multiple defibrillations despite the administration of intravenous and oral beta-blockers, diazepam, potassium, and magnesium (Figure 1C). RVOT PVCs with the initial QRS morphology were not detected. The device was upgraded to a dual-chamber ICD after implantation of an atrial lead and DDDR-60 pacing was initiated besides continuation of beta-blocker therapy. During the 6 months of follow-up, the patient remained free from further ventricular tachyarrhythmias and symptomatic improvement was noted. Echocardiographic determination revealed normalization of LV-EF.

Discussion

Frequent RVOT PVCs may be a possible cause of LV dysfunction and heart failure and focal RF ablation produces clinical benefits in these patients.1 A recent report points out to focal RF ablation as a potential valuable tool in controlling arrhythmogenesis by elimination of RVOT PVCs that trigger torsades de pointes in long QT patients.2 Schoonderwoerd and van Gelder have recently reported on a patient with a long QT syndrome, sinus bradycardia and ventricular ectopy who was successfully treated with a dual-chamber pacemaker.3 Our patient remained free from further episodes of ventricular tachyarrhythmias after dual-chamber pacing was initiated. Meanwhile, a SCN5A-mutation (p.Arg190Gln, Exon 5, heterozygous) consistent with long QT-syndrome 3 was found. A former resting ECG of our patient from 20 years ago already showed intermittent QTc prolongation at rest. The elimination of the incessant ventricular bigeminy as one arrhythmogenic foci may have facilitated the development of pause-dependent torsades de pointes associated with QTc prolongation. The case report highlights the risk of arrhythmogenesis after ablation of ventricular bigeminy in case of QTc prolongation. A dual-chamber ICD is indicated in selected patients with long QT-syndrome in order to allow for DDDR pacing when there are untreatable pause-dependent torsade de pointes despite adequate beta-blocker therapy or to enable beta-blocker therapy in case of severe bradyarrhythmia.

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References

CASE REPORT

Intramural inflammation as a cause of transient ST-segment elevation in a patient of cardiac sarcoidosis

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Cardiac magnetic resonance demonstrated myocardial damage within the left ventricle (LV) in a patient with cardiac sarcoidosis. During inflammation, ST-segment elevation was observed in her ECG. The ST-segment deviation was attenuated following disappearance of the abnormal Gallium-67 uptake in the LV, suggesting transmural LV voltage gradient was a cause of the ST-segment elevation.

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