Propranolol efficiency in prevention of sustained ventricular tachycardia in patients with implanted cardioverter-defibrillator: a case series

Patients with implanted cardioverter-defibrillator (ICD) often have justified ICD activations no matter if the indication for implantation was primary or secondary prophylaxis of sudden death. First line therapy in the prevention of recidivate ventricular arrhythmia in these patients is antiarrhythmic therapy, but if this is inefficient, arrhythmic substrate radiofrequency (RF) ablation is recommended. Ablation treatment is an accepted procedure in patients with ischemic heart disease, but it has rarely been used in patients with idiopathic and particularly polymorphic ventricular tachycardia (VT). It must be emphasized that acute ablation is relatively successful, but because of substrates progression, relapses of VTs are very frequent (35%) (1). Therefore, antiarrhythmic therapy remains an important therapy after the ICD implantation, before, and often after RF ablation (2).

We present the cases of five patients in whom the prevention of recidivate VTs was achieved only by an old nonselective beta-blocker propranolol (dose 20 × 40 or 2 × 80mg).

Patient 1: a 52 years old woman with ICD implanted as a secondary prevention after out-of-hospital cardiac arrest caused by ventricular fibrillation. There was no structural heart disease, coronarography was normal, and there was only arterial hypertension in patient’s history. QTc interval was within the reference range, there were no Brugada syndrome elements, and no sudden death in family history. During the first year after the implantation, ICD was activated for more than 10 times because of polymorphic VT in spite of medicaments therapy. On one occasion even an electrical storm occurred. After the implantation, bisoprolol maximum dose was given, later in combination with mexiletine, which was stopped as a result of intolerance. Combination with amiodarone was used for a short period of time but with no effects. Only after bisoprolol had been replaced with propranolol, there were no tachycardias and ICD stopped activating. Since then the patient has been followed-up for 5 years.

Patient 2: a 56 years old man with no family or individual heart disease history, or any other serious disease. ICD was implanted as secondary prophylaxis after relapsing syncope caused by VTs. The first therapy with amiodarone had not prevented multiple ICD activations, so it was replaced by a combination of bisoprolol and mexiletine. Despite the new therapy, ICD continued to activate for several justified occasions (5 times). Only after bisoprolol had been replaced with propranolol, ICD stopped activating. Since then the patient has been followed-up for 3 years. In the meantime, mexiletine therapy has been stopped (it is unavailable in Croatia), so now he takes propranolol only.

Patient 3: a 69 years old man with ICD implanted after recidivate sustained VTs. The patient had no structural heart disease or any signs of ischemia. Coronarography was normal, and there was only arterial hypertension in patient’s history. QTc interval was within the reference range, there were no Brugada syndrome elements, and no sudden death in family history. During the first year after the implantation, ICD was activated for more than 10 times because of polymorphic VT in spite of medicaments therapy. On one occasion even an electrical storm occurred. After the implantation, bisoprolol maximum dose was given, later in combination with mexiletine, which was stopped as a result of intolerance. Only the replacement of bisoprolol by propranolol led to a complete VT suppression. In the last 3 years, the patient has not had any ICD activation. In the meantime, mexiletine therapy has been stopped.

Patient 4: a 86 years old man. Two years ago, he had inferioposterior myocardial infarction with ST-eleva-
tion (STEMI) and a year after he was hospitalized for hemo-
dynamically unstable VT. Slightly reduced ejection fraction
(EF 45%-50%) was determined, caused by inferioposterior
hypokinesia. Coronarography showed an old collateralized
occlusion of the right coronary artery (RCA) and irrelevant
changes on other coronary arteries. Sustained VTs repeat-
ed daily, despite the use of different antiarrhythmics: biso-
prolol, lidocaine, amiodarone, and magnesium. After the
temporary electrode had been placed, conversion was
achieved by overdrive electrostimulation, but only occa-
sionally. However, electrical cardioversion had to be done
in most cases (6 times). Only the combination of propra-
nolol and lidocaine in high doses managed to suppress VT.
The patient had a successful RF ablation of VT with propra-
nolol therapy only. During the 2 years of follow-up, he has
had no arrhythmia. ICD has not been implanted.

Patient 5: a 60 years old man. After anteroseptal STEMI with
severely reduced left ventricular systolic function, the pa-
tient developed frequent VTs that responded neither to
combination of bisoprolol and lidocaine nor to overdrive
stimulation. The use of amiodarone induced a remarkable
extension of the QTc interval without suppressing arrhyth-
 mia. On most occasions, arrhythmia had to be stopped
by electrical cardioversion (5 times). Only after using pro-
pranolol instead of bisoprolol, arrhythmia was completely
suppressed. When propranolol had been cancelled, be-
cause of septic shock, VT relapsed. After the septic shock
had been stabilized, ICD was implanted and propranolol
was included to the therapy again. Further follow-up (1
year) has not recorded any ICD activation.

In our cases, non-selective propranolol was more effective
in suppressing severe VTs than newer selective beta block-
er bisoprolol. Unlike bisoprolol, propranolol blocks both
beta 1 and beta 2 receptors (20% of beta adrenergic re-
ceptors in the heart) and owing to liposolubility also pen-
etrates into the brain (3). Thereby, it can also result in the
central inhibition of the sympathetic nervous system. The
unique stability effect on the membrane of myocytes has
also been described (4). Previous studies showed that pro-
pranolol in combination with amiodarone offered the best
prevention of VT relapses (5). The reason why propranolol
is not widely used in patients with cardiomyopathy is the
lack of evidence-based data for these indications. Studies
in patients with cardiomyopathy have recently been con-
ducted using only newer beta-blockers (carvedilol, meto-
prolol, bisoprolol) (5). We believe it could be useful to con-
duct a study on propranolol, because propranolol possibly
has more beneficial effect on the survival of patients with
cardiomyopathy than selective beta blockers.

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