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Case report

Mexiletine as a one effective alternative for antiarrhythmic drugs and ablation resistant electrical storm – A case report

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ABSTRACT

We present case of 78-year-old male transferred to our Department due to multiple episodes of malignant ventricular arrhythmias refractory to previous antiarrhythmic treatments (device settings optimization, amiodarone treatment, lidocaine infusion and optimal pharmacotherapy). Additionally, patient had ischemic end-stage heart failure and implanted CRT-D. We excluded secondary causes of electrical storm and due to inefficacy of medical therapy, we applied radiofrequency ablation. During 1st day after procedure life-threatening VT/VF recurred. As our last choice, we modified therapy and introduced mexiletine, what resulted in complete disappearance of complex ventricular arrhythmias.

In case of amiodarone inefficacy mexiletine may be considered effective alternative antiarrhythmic drug.

Introduction

Electrical storm (ES) is a state of cardiac electrical instability. Typically, it affects older men with coronary artery disease [1]. Amiodarone plus β-blocker is the most effective therapy to prevent ventricular tachycardia (VT) or ventricular fibrillation (VF) in patients with implanted cardioverter-defibrillator (ICD) [2]. However, while inefficient, alternative medications and methods might to be considered. We present a case of 78-year-old male patient in whom we managed to successfully use one of such alternative medications – mexiletine.

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Case report

We present a case of 78-year-old male transferred to Department of Cardiology due to multiple episodes of malignant ventricular arrhythmias refractory to previous antiarrhythmic treatments including device settings optimization, antiarrhythmic drug therapy (amiodarone, lidocaine) and optimal pharmacotherapy. The patient had history of biventricular, end-stage heart failure (HF) due to ischemic dilated cardiomyopathy, enlarged left ventricle up to 78 mm in diastole and systolic dysfunction with ejection fraction (LVEF) 15%, apex aneurysm with thrombus, permanent atrial fibrillation, third-degree atrioventricular block, hypertension, diabetes mellitus type 2 and chronic kidney disease. He underwent inferior wall myocardial infarction 24 years ago that was treated conservatively and primary PCI with stent implantation in the left anterior descending artery complicated by sudden cardiac arrest 8 years ago. Eight years earlier patient underwent ICD implantation (as the secondary prevention of sudden cardiac death (SCD)), which was followed by upgrade to Cardiac Resynchronization Therapy-Defibrillator (CRT-D) 4 years later.

Before transfer to our Department the patient was hospitalized due to infectious exacerbation of congestive HF (CHF). Prior to ES initiation for couple of week's patient suffered from respiratory tract infection, refractory to different courses of antibiotics. Sustaining elevation of infectious parameters clinically resulted in CHF exacerbation, what led to ES. During previous hospitalizations, multiple unsuccessful attempts to modify CRT-D settings were made, i.e. higher-rate detection threshold of VT, VT overriding, different antitachycardia pacing (ATP) algorithms or when unsuccessful or hemodynamically unstable ICD-initiated cardioversions. To achieve stabilization of the general state, the patient required continuous amiodarone administration and intravenous lidocaine infusion.

In our Department, we observed hemodynamically unstable cardiac arrhythmias – monomorphic, degenerating to polymorphic VT and VF (Image 1A) resulting in CRT-D interventions up to several dozen a day. Laboratory data were as follows: red blood cells – 4.73 $\times$ 10^6/μL; hemoglobin – 14.2 g/dL; white blood cells – 12.81 $\times$ 10^3/μL; estimated glomerular filtration rate – 38 ml/min/1.72 m^2; creatinine – 1.76 mg/dL; C-reactive protein – 52.2 mg/L; D-dimers – 2732 pg/ml; platelets – 188 $\times$ 10^3/μL; NT-proBNP value – 5711 μg/L; INR – 1.76, creatinine kinase (CK) – 52 IU/L; CK MB isoenzyme – 24 IU/L; troponin I – 0.063 IU/L. Potassium and magnesium level on admission were 4.6 mmol/L and 1.1 mmol/L, respectively. Initial corrected QT interval (QTc) was 430 ms. Due to elevated inflammation parameters, antibiotic therapy was continued. Before transfer to our Department the patient received amoxicillin with clavulanic acid (which do not prolong QTc). We switched the therapy to clarithromycin with ciprofloxacin. Additionally, optimal pharmacotherapy for CHF exacerbation, including angiotensin II receptor blocker, β-blocker, statin, spironolactone and furosemide in maximal tolerated doses, was continued.

Cardiac arrhythmias were refractory to antiarrhythmic drugs and caused further deterioration of patient's clinical status. On the 3rd day in our Department we tried to ablate VT, which originated from LV (Image 1B). Before and during the procedure the patient received vancomycin, as prophylaxis. Programmed ventricular stimulation induced monomorphic VT, cycle length 430 ms with alternans. There were two dominating PVC morphologies and they were considered the ablation's target. After procedure programmed ventricular stimulation did not induce VT. Early post-procedure period was uneventful. On the 1st day after intervention we observed aggravation of life-threatening, hemodynamically unstable, cardiac arrhythmias. Patient received additionally amiodarone p.o., continuous i.v. infusion of midazolam and xylocaine in up titrated doses. We withdrew metoprolol and introduced carvedilol and gave fractioned s.c. doses of morphine. Additionally, we made temporary attempt to inactivate ATP. Due to *Clostridium difficile* infection the antibiotic therapy was switched one more time – to vancomycin p.o.

Four days after radio-frequency ablation due to sustaining life-threatening condition we decided to withdraw amiodarone (QTc = 420 ms) and introduced therapy with mexiletine. The initial dose was 400 mg 3-times per day and was up-titrated up to 400 mg 4-times per day. After mexiletine

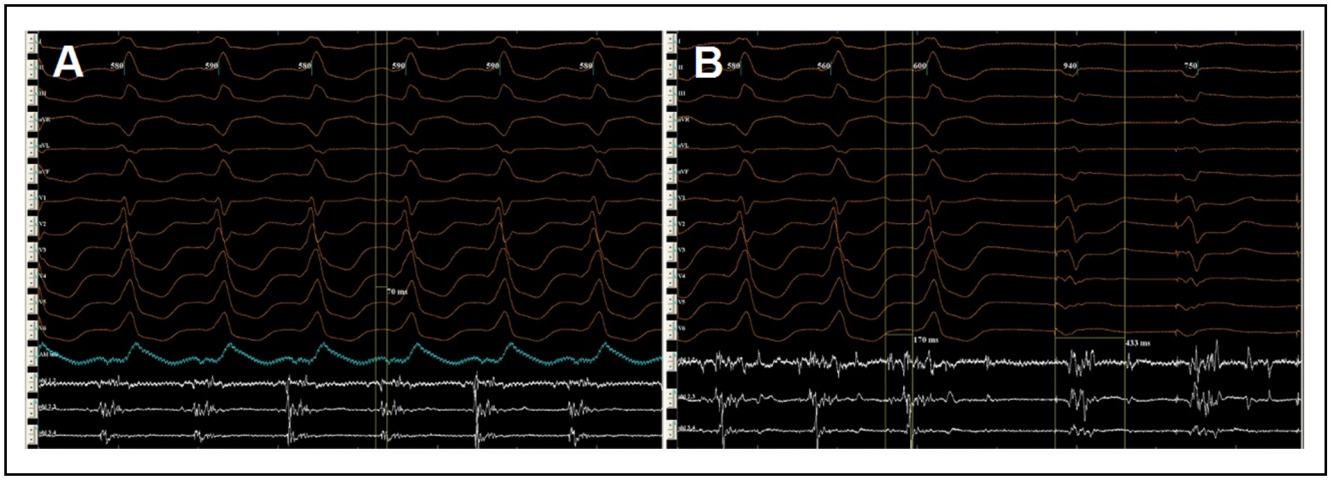

Image 1 – (A) Monomorphic ventricular tachycardia before ablation; (B) attempt at ablation substrate of ventricular tachycardia.

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