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Case report

Electrical storm due to myocarditis in post-infarct patient: When two diseases meet

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ABSTRACT

This case presents an episode of electrical storm (ES) in post-myocardial infarction patient results from myocarditis. ES was interrupted by aneurysmectomy and additional isolation of VT origins by radiofrequency catheter ablation. The histological evaluation of aneurysm material proved acute myocarditis. The key findings indicating acute inflammation in myocardium was an increased level of peripheral inflammatory biomarker IL6 and auto-antibodies to beta1-adrenergic receptor (β1-AAbs). Gated SPECT with phase images analysis turned out to be appropriate imaging strategy in visualizing potentially reversible causes of ventricular arrhythmias such as myocarditis. Taking together our findings might add some information on the pathogenesis and predisposing factors of ventricular arrhythmias especially in the cases of electrical storm.

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Introduction

Electrical storm (ES) is a life-threatening syndrome that is defined by three or more episodes of sustained ventricular tachycardia (VT) or ventricular fibrillation (VF) or appropriate shocks from implantable cardioverter-defibrillator (ICD) within 24 h. The incidence of ES varies from 4% to 28% in different studied populations. According to the MADIT-II substudy, patients, who experienced ES, showed a 7.4-fold higher risk of death compared with those without [1]. It is evident that ES is associated with worse patient's outcome, however it is still unclear whether their poor prognosis is a direct consequence of ES or it is simply an epiphenomenon of advanced structural heart disease [2].

A lot of attempts have been made in order to identify the possible predictors of ES. Undoubtedly that it is strongly associated with progressive heart failure (HF) [2]. Streitner F. et al. showed that episodes of ES in patients with HF could be predicted by elevated serum concentrations of inflammatory biomarkers such as hs-CRP, NT-pro BNP and IL-6 [3]. On the other hand the deterioration of HF itself can be accompanied by elevation of inflammatory biomarkers as well as anticardiac antibodies [4,5]. At the same time increased cytokine level and persistence of autoantibodies to beta1-adrenergic receptor (β1-AAbs) is usually observed in the serum of patients with inflammatory cardiac pathology [5]. These biomarkers can directly or indirectly worsen left ventricular (LV) function and so trigger VTs [5,6] as well as reflect the ongoing inflammation in myocardium.

One of clinical manifestations of myocarditis is new-onset ventricular arrhythmias. Such arrhythmias are often related to myocardial damage caused by toxic or viral as well as autoimmune injury. Moreover inflammatory affection of myocardium can additionally trigger arrhythmias also in patients with pre-existing arrhythmogenic focuses as we observed in presented case.

Case report

A 65-year old woman admitted to Intensive Care Unit of our Hospital on February 2013 because of recurrent VT episodes and multiple ICD shocks during the last three days.

The patient's history indicates that she survived from acute anterior myocardial infarction (MI) in 2001 and was clinically stable until the year 2012 on standard therapy. In July 2012 the patient suffered from recurrent anterior MI, which was complicated by sustained VT. That time the patient underwent coronary angiography followed by of two stents implantation in the left anterior descending artery. Two months later the patient suffered from several episodes of palpitations and presyncopes, that were documented on ECG as VTs. Clinical examination showed symptoms relevant to NYHA class II of heart failure. The echocardiography revealed a large anterior-septal hypokinesis with an apical aneurysm and significantly decreased left ventricular ejection fraction (LVEF) (27%). Patient underwent ICD implantation for secondary prevention of SCD and was stable until February 2013. It should be noted, that about two weeks before the onset of ES the patient had an episode of acute respiratory infection.

On present admission to the hospital patient was hemodynamically stable. She was on treatment with bisoprolol 5 mg/daily, amiodarone 200 mg/daily, clopidogrel, statins, aspirin, angiotensin converting enzyme (ACE) inhibitors and diuretics in appropriate doses. The ICD interrogation revealed multiple episodes of monomorphic VT with different QRS morphologies and maximal heart rate of 220 b.p.m. and a single episode of VF. Ventricular arrhythmias were successfully terminated by ICD.

Laboratory tests showed normal serum levels of thyroid hormones, potassium, magnesium, hs-CRP. The troponin test was negative. Polymerase chain reaction (PCR) revealed absent of herpes simplex 1–2 virus, human herpes 6 virus, Epstein-Barr virus, cytomegalovirus and parvovirus B19. The additional evaluation of inflammatory biomarkers found negative concentration of tumor necrosis factor alpha while the level of interleukin 6 (IL6) exceeded reference ranges and was 14.8 pg/ml. We also performed the evaluation of β1-AAbs in serum by enzyme-linked immunosorbent assay. The level of β1-AAbs was increase up to 7.2 (the normal values were considered to be less than 1), indicating possible autoimmune component of heart injury process.

Instrumental testing: The chest X-ray showed left ventricle enlargement, no signs of pulmonary edema. Dual-chamber ICD and electrodes were in normal position. Transthoracic echocardiography showed dilated left ventricle (end diastolic diameter (EDD) 6.7 cm, end diastolic volume (EDV) 268 ml) with extensive anterior-septal hypokinesis, huge apical aneurysm, accompanied by decreased LVEF (30%), moderate mitral regurgitation and mild lung pulmonary hypertension (pulmonary artery systolic pressure (PASP) 40 mmHg). Urgent coronary angiography did not show any coronary artery lesions including no evidence of restenosis in previously implanted stents.

Gated SPECT (MIBI) myocardial perfusion revealed significantly dilated left ventricle (EDV = 290 ml) with a clear non-perfused apical and anterior zones. Additional akinesis at non-perfused regions confirmed the diagnosis of anterior-apical aneurysm. The only lateral and posterior left ventricular walls were functionally preserved being resulted in LVEF of 24%, the summed rest score (SRS) of 49 and high volume of affected myocardium (up to 61%). The additional left ventricular perfusion protocols matching the wall motion abnormalities and phase images detected several dissociated foci of pathological asynchrony and active wall motion inside the aneurysm. The heterogeneous features of these foci makes possible to consider them as probable origins of ventricular arrhythmias (Fig. 1, study I).

According to 24-h Holter ECG almost all spontaneous VT episodes were preceded by bradycardia and have been usually triggered by R-on-T premature ventricular beats. Therefore as the first line of treatment strategy the baseline pacing frequency of ICD was increased from 55 to 70 b.p.m., the dose of bisoprolol was increased to 7.5 mg/daily. Additional continuous infusion of amiodarone (600–900 mg/daily) was started but that produced only temporary effect. Based on previous history of acute respiratory infection, above mentioned laboratory findings and gated SPECT (MIBI) data the inflammatory hypothesis of ES in our patient have been suggested. This made us to start glucocorticosteroid therapy