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Efficacy of multielectrode Array catheter for ventricular tachycardia ablation in a patient with electrical storm

Ventricular arrhythmias are one of the major causes of mortality in patients with coronary artery disease. In this group of patients, ventricular tachycardia (VT) is produced by a reentrant mechanism due to the presence of a fibrotic scar, and may appear even decades after myocardial infarction (MI). (1) Despite the important advances in the treatment of MI, the risk of VT in the population with coronary artery disease is still an important issue in clinical practice. Antyarrhythmic agents and implantable cardioverter defibrillator (ICD) devices are the cornerstone in the management of ventricular arrhythmias. (2) These therapies are far from being infallible and may present significant adverse events. (3) Patients implanted with an ICD may experience significant physical and psychological deterioration due to repetitive delivery of shocks.

Catheter ablation of VT may have a significant impact in patient's quality of life by alleviating symptoms, reducing the requirement of antiarrhythmic drugs and the number of ICD shock deliveries. However, this procedure may result difficult because the arrhythmia is poorly tolerated in many occasions and hemodynamic decompensation make collection of point-by-point electrograms impossible.

The use of the multielectrode Array catheter as a diagnostic tool is becoming more frequent in our environment. (4) This noncontact device consists of 64 electrodes deployed on an inflatable mesh that is suspended in the selected heart chamber to treat the arrhythmia. The catheter records electrograms from multiple sites within a single cardiac chamber and is capable of determining the precise site of origin of the arrhythmia from a single run of tachycardia. We report the case of a 69-year-old male patient treated in our hospital, with history of hypertension, dyslipidemia, and former smoking. His usual creatinine levels were 1.4 mg/dl (creatinine clearance measured by MDRD of 56 ml/min/1.73 m2). Eighteen-years ago he presented myocardial infarction of the inferior wall and received medical treatment. In 2011 he complained of progressive angina and underwent myocardial revascularization surgery requiring implantation of four bypass grafts. At that time, the echocardiogram revealed severe left ventricular dysfunction with abnormal contraction in the inferior and posterior wall. Six months later he was admitted to the emergency department with sustained VT with hemodynamic instability. An ICD device was implanted. During follow-up, three appropriate shocks were delivered due to VT: Amiodarone 400 mg/day and carvedilol 25 mg bid were initiated.

The patient was admitted to the emergency department due to nausea, dizziness, abdominal pain and dyspnea of one-week duration. He reported two shock ICD deliveries. He denied fever or fever-like symptoms. He did not complain of angina and did not modify his usual medication.

He was evaluated by a cardiologist due to heart failure and signs of low cardiac output. At the physical examination, the patient looked severely ill, with heart rate of 140 beats per minute, tachypnea, hypotension, poor distal perfusion and oxygen saturation of 75% breathing room air. He did not present fever. Crackles were heard over both lung bases and middle fields.

The electrocardiogram showed a regular tachycardia of 140 beats per minute with wide QRS complexes (Figure 1), that was interpreted as sustained VT with hemodynamic instability. As the ICD was programmed to treat VT with heart rates >150 beats per minute, therapy with external cardioversion with a 200 J biphasic shock was decided.

The laboratory tests were within normal ranges. The chest X-ray showed signs of pulmonary congestion. Interrogation of the ICD device showed that the patient had presented three appropriate shocks to treat VT within the past 48 hours.

Furosemide was administrated and a negative fluid balance was achieved; amiodarone and lidocaine were given intravenously. Coronary angiography was performed to rule out acute coronary syndrome, and no new coronary artery stenoses were observed. Blood and urine cultures were negative. The patient presented several episodes of VT with hemodynamic instability and other symptomatic, short-lasting episodes despite the treatment. During hospitalization, three ICD shocks were delivered in the range of VT detection.

A diagnosis of electrical storm in a drug-refractory patient with hemodynamic instability was made and VT catheter ablation was indicated using a multielectrode Array catheter. Ventricular tachycardia was induced by programmed stimulation from the right ventricle (RV) (Figure 2) causing hemodynamic instability and was rapidly reverted by electrical shock. Substrate mapping and propagation mapping could be performed using a very few tachycardia beats, allowing the identification of the site of arrhythmia origin. A scar was identified in the anterolateral region of the RV. The area of slow tachycardia conduction was de-

tected along one of the edges of the scar (Figure 3). Radiofrequency energy was delivered to that area using a 4-mm ablation catheter. Then, the VT could not be induced after programmed stimulation and isoproterenol infusion. Over the next 7 months of follow-up, VT was not detected by ICD interrogation. The patient is currently treated with beta blockers and amiodarone 100 mg/day.

Patients with an ICD device are exposed to multiple electrical shocks delivered by the device with adverse effects on health outcomes. Each shock, appropriate or not, increases mortality. (5) In many occasions high dose of antiarrhythmic drugs are required and may generate significant adverse effects. (3)

Radiofrequency catheter ablation may produce significant improvement in the quality of life of these patients. The procedure requires the induction of the arrhythmia in the electrophysiology laboratory. The patient has to tolerate the arrhythmia to allow collection of the necessary electrograms to determine the possible origin of the tachycardia. In several occasions, the arrhythmia produces hemodynamic instability in elder patients or in those with ventricular dysfunction, turning impossible mapping with the usual methods.

The multielectrode Array catheter consists of 64 electrodes deployed on an inflatable mesh and is suspended in the selected heart chamber to treat the arrhythmia. The device generates >3000 virtual electrograms in a single cardiac cycle. The map is updated 1200 times per second. Due to these properties, the system can collect the sufficient information to generate voltage and propagation maps within a single cardiac cycle. In this way, the site of origin of non-sustained arrhythmias or of those with hemodynamic impact can be determined instantly. (6)

We have reported the case of a fully pharmacologically treated patient with ICD, with symptoms due to ventricular arrhythmia, with some episodes of non-sustained ventricular tachycardia and other tachyarrhythmic episodes with hemodynamic instability. In this patient, the multielectrode catheter was a fundamental tool for rapid identification of the site of origin of the arrhythmia within a couple of beats, reducing the risk of hemodynamic collapse in the electrophisiology laboratory.

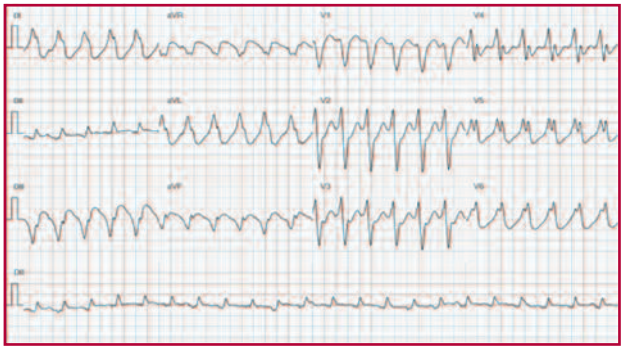

Fig. 1. 12-lead electrocardiogram showing regular tachycardia of 140 beats per minute with wide QRS complexes.

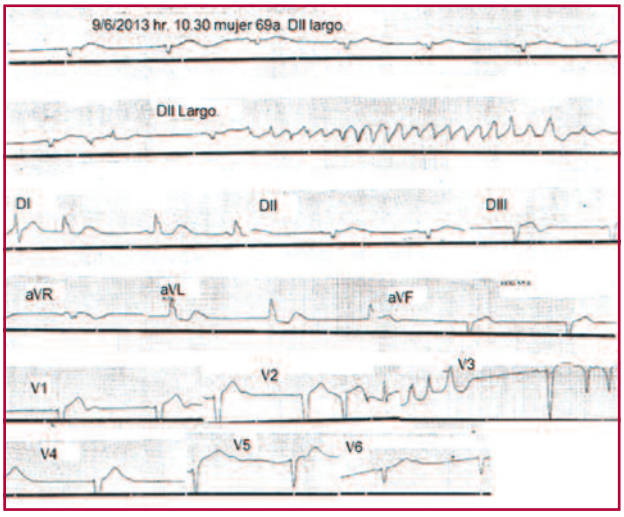

Fig. 2. Ventricular tachycardia induced in the electrophysiology laboratory with asynchronous right ventricular stimulation.

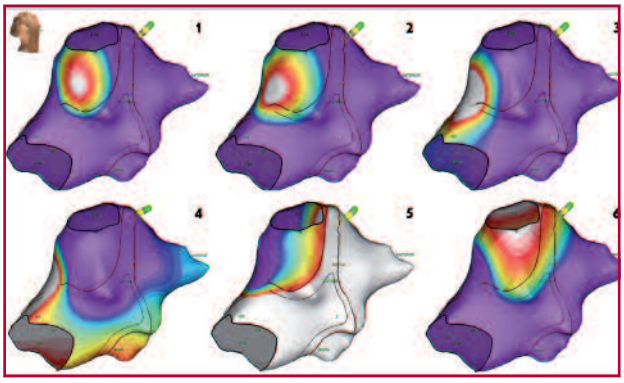

Fig. 3. Isopotential mapping showing the wavefront rotation area, as indicated by the white color. Slow conduction zone in the anterolateral area of the right ventricle (frames 1, 2 and 3).

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Cardiac tamponade in disseminated cryptococcosis

Cryptococcosis is an opportunistic fungal disease caused by an encapsulated yeast, Cryptococcus neoformans (CN). The perfect state of the basidiomycetes is Filobasidiellia neoformans, which has two varieties: neoformans and bacillispora. Five serotypes and two different varieties have been identified: CN var. neoformans (A, B and AD) and CN var. gattii (B and C). (1)

Cryptococcosis is an important cause of infection in patients with human immunodeficiency virus; it has no sex predilection though it is more common in men between 30 and 60 years, and, to a less extent, in children.

Exposure to pigeon feces or air-conditioning systems contaminated with bird droppings is a risk factor. Several infectious agents produce infective pericardial effusion; CN is one of them, with mortality between 15% and 30%. (2, 3)

We present a case report of cardiac tamponade in disseminated crytococcosis (DC).

A 46-year-old male patient was admitted to the intensive care unit due to rapidly progressive dyspnea and consciousness deterioration. He had a history of dilated cardiomyopathy due to acquired immunodeficiency syndrome (AIDS) under antiretroviral therapy. He had had a previous hospitalization a short time ago with DC diagnosis, meningeal and lung involvement, severe hypoacusia and kidney dysfunction. At admission, the patient was confused and did not present fever (36.2 °C); his blood pressure was 70/40 mm Hg, he had jugular engorgement, poor distal perfusion and tachypnea (25 breaths per minute). Crackles were heard over both lung bases and a grade 1/6 systolic murmur was heard over the tricuspid area.

The electrocardiogram indicated sinus rhythm with a heart rate of 110 beats per minute and low QRS voltage in the frontal and precordial leads. The chest X-ray showed cardiac enlargement and diffuse redistribution of blood flow in both pulmonary fields. In the laboratory tests, erythrocyte sedimentation rate was elevated (58 mm), the hematocrit was 30%, BUN 120 mg/dl, creatinine levels 2.2 mg/dl, and creatinine clearance 38.57 ml/min.

Doppler echocardiography showed mild left ventricular dilatation, normal wall thickness, global hypokinesia and severe left ventricular dysfunction with an ejection fraction of 24%. The left atrium was moderately dilated, with an estimated area of 29 cm2. The dimensions of the right chambers were normal and the right ventricle had mild ventricular dysfunction. Opening of the trileaflet aortic valve was normal. The mitral valve had mild regurgitation and restrictive left ventricular filling pattern. The presence of mild tricuspid regurgitation allowed the estimation of systolic pulmonary artery pressure of 50 mm Hg. A severe circumferential pericardial effusion was observed and the pericardial layers were separated by 19 mm (posterior effusion), 17 mm (lateral effusion) and 6 mm (anterior effusion). Echocardiographic signs of cardiac tamponade were present, with early diastolic collapse of the right ventricular anterior wall and diastolic collapse of the right atrium, dilated inferior vena cava of 32 mm with inspiratory collapse <50% (Figures 1 and 2).

The patient required endotracheal intubation, mechanical ventilation and hemodynamic support with vasoactive drugs. He underwent emergency pericardiocentesis, and after 400 ml of blood stained fluid were drained, the hemodynamic parameters experienced immediate recovery.

Further examination of pericardial fluid was positive for CN antigen, and quantitative analysis was performed by latex test.

Microscopic examination with India ink stain and pericardial fluid culture were positive for CN.

The infectologists indicated fluconazole 200 mg for life because of the unfavorable outcome and disseminated form; the patient is currently taking this medication.

The incidence of cryptococcosis in AIDS patients varies among the different regions of the world, from 50-10% in Western Europe and in the United States, to 20% or more in Central Africa and Southeast Asia. In Argentina, the incidence would be 4.6% according to official public health agencies. (4) Infection is acquired by inhalation of air-borne yeast cells present in nature that easily reach the pulmonary alveoli. Neither person-to-person nor animal-to-human respiratory transmission has not been documented, but transmission via organ transplantation has been reported. Physicians should suspect this disease to make the diagnosis. (1)

Pericardial involvement occurs as pericarditis, asymptomatic pericardial effusion or cardiac tamponade. The prevalence of pericardial involvement ranges between 3% and 37%. (5)

The typical signs and symptoms of heart failure (HF) may be difficult to recognize in a population of