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Sustained Ventricular Tachycardia as the first presentation of transthyretin amyloid cardiomyopathy: a case report

Taquicardia ventricular sostenida como primera manifestación de Cardiomiopatía Amiloide por Transtiretina: reporte de un caso

Taquicardia ventricular sustentada como primeira manifestação de Cardiomiopatia Amioloide por Transtiretina: relatório de um caso

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Sustained Ventricular Tachycardia as the first presentation of transthyretin amyloid cardiomyopathy: a case report

CONCEPTOS CLAVE:

Qué se sabe sobre el tema.

A frequent presentation of Amyloid Transthyretin Cardiomyopathy wild type (ATTR-CMwt) is heart failure and arrhythmias, but the prevalence of sustained ventricular tachycardia as the first manifestation is unknoun and is considered uncommon.

Qué aporta este trabajo.

We report an uncommom case of a 71 y-old man without heart failure who had a severe sustained ventricular tachycardia (SVT) as the first presentation of ATTR-CMwt. This case highlights that any type of life-threatening arrhytmia could be find and the mechanisms of SVT and the current indications for ICD are reviewed.

Divulgación

ATTR-CM is a toxic-infiltrative disease affecting the myocardial interstitium. Regular symptoms are related with heart failure, atrio-ventricular block, any type of arrhythmias or aortic stenosis. Life-threatening sustained ventricular tachycardia (SVT) is considered uncommon and its prevalence is unknoun, despite data from some registries. However, patients with this arrhythmia can suffer sudden cardiac death or they are not considered for ATTR-CM. In patients with SVT and septal thickness >12 mm ATTR-CM should be considered as a possible diagnosis.

Sustained Ventricular Tachycardia as the first presentation of transthyretin amyloid cardiomyopathy: a case report

Abstract

Keywords:
amyloidosis; heart; tachycardia; arrhythmia.

Transthyretin Amyloid Cardiomyopathy (ATTR-CM) was considered an uncommon disease until a few years ago, but advances in the epidemiology and non-invasive diagnostic tests have increased its timely detection. We report a 71 years-old man with history of hypertension and an incidental carcinoma of the left kidney detected 6 years ago, without heart failure who was performed cardiac magnetic resonance images (MRI) by suspicion of hypertrophic cardiomyopathy. Before his cardiologist be aware of the result, he suffered a severe sustained ventricular tachycardia (SVT) that required emergency cardioversion. Echocardiogram and cardiac MRI were suggestive for cardiac amyloidosis and the diagnosis was confirmed by scintigraphy with 99mTc-PYP (Perugini +3). Serum levels of light chains kappa and lambda were normal, and serum and urine immunofixation were negative; a genetic test had no variants, so supporting an ATTR-CM wild type. PET-CT did not detect metastasis of the renal tumor, but showed cardiac hypermetabolism and pericardial effusion. An implantable cardioverter defibrillator (ICD) was placed and after nine days a shock was delivered by the ICD due to a new event of SVT; in addition a Holter monitoring registered runs of asymptomatic atrial fibrillation. Etiologic treatment for ATTR-CM with Tafamidis 61 mg was started, amiodarone and rivaroxaban were added for control of arrhythmias and prevention of systemic embolism, respectively. After 14 months of follow-up, he is stable in class I NYHA. ATTR-CM is a complex disease, and the treatments should be indicated by a multidisciplinary team that consider the risks, benefits, and costs of each intervention.

Taquicardia ventricular sostenida como primera manifestación de Cardiomiopatía Amiloide por Transtiretina: reporte de un caso

Resumen

Palabras clave:
amiloidosis;
corazón;
taquicardia;
arritmia.

Cardiomiopatía Amiloide por Transtiretina (ATTR-CM) fue considerada hasta hace poco tiempo una enfermedad poco frecuente, pero los avances en el conocimiento de su epidemiología y de los tests no invasivos han aumentado su diagnóstico oportuno. Presentamos un hombre de 71 años con historia de hipertensión arterial y un tumor renal a células claras operado 6 años antes, sin insuficiencia cardíaca a quien se realizó una resonancia magnética cardiaca por sospecha de miocardiopatía hipertrófica; antes de que su cardiólogo viera el resultado, presentó una taquicardia ventricular sostenida (TVS) severa que requirió cardioversión eléctrica de urgencia. Ecocardiograma y resonancia magnética cardiaca fueron sugestivos y el diagnóstico fue confirmado por centellografía con PYPTc99m (Perugini +3). Cadenas livianas kappa y lambda en suero e inmunofijación en sangre y orina fueron negativas y el test genético no mostró variantes, confirmando ATTR-CMwt. PET-CT no mostró metástasis del tumor renal pero detectó hipermetabolismo miocárdico y derrame pericárdico. Se colocó un cardio-desfibrilador implantable (CDI) y nueve días después tuvo una nueva TVS que fue detectada y tratada adecuadamente por el CDI. Además, el monitoreo Holter detectó eventos asintomáticos de fibrilación auricular. Se inició tratamiento etiológico de ATTR-CM con Tafamidis 61 mg y se agregó amiodarona para prevenir nuevos eventos de TVS y rivaroxaban para prevención de embolismo sistémico. A 14 meses de seguimiento el paciente permanece en clase I de NYHA. CM-ATTR es una enfermedad compleja y los tratamientos deberían ser indicados por un equipo multidisciplinario que considere los riesgos, beneficios y costos de cada intervención.

Taquicardia ventricular sustentada como primeira manifestação de Cardiomiopatia Amioloide por Transtiretina: relatório de um caso

Resumo

Palavras-chave:
amiloidose;
coração;
taquicardia;
arritmia.

A Cardiomiopatia Amiloide por Transtiretina (ATTR-CM) foi considerada até há pouco tempo, uma doença pouco frequente, mas os avanços no conhecimento da sua epidemiologia e dos testes não invasivos aumentaram o diagnóstico oportuno. Apresentamos um caso de um homem de 71 anos com antecedentes de hipertensão arterial e tumor renal a células claras com cirurgia de 6 anos de data, sem insuficiência cardíaca. Realizou-se ressonância magnética cardíaca por suspeita de miocardiopatia hipertrófica. Antes da revisão do estudo pelo seu cardiologista, o paciente apresentou taquicardia ventricular sustentada (TVS) severa que exigiu cardioversão eléctrica de emergência. Ecocardiograma e ressonância magnética foram sugestivas e o diagnostico foi confirmado com cintilografia com PYPTc99m (Perugini +3). Correntes claras kappa e lambda em souro e imunofixação em sangue e urina foram negativos e o teste genético não amostrou variações, confirmando ATTR-CM wild type. O PET-CT não mostraram metátese do tumor renal, mas detecto hipermetabolismo miocárdico e derrame pericárdico. Colocou-se um cardio-desfibrilador implantável (CDI) e nove dias depois apresentou uma nova TVS que foi detectada e tratada adequadamente pelo CDI. Além disso, o monitoreo-Holter detectou eventos assintomáticos de fibrilação auricular. Começou-se um tratamento etiológico de ATTR-CM com Tafamidis 61 mg e se adicionou amiodarona para prevenir novos eventos de TVS e rivaroxaban para prevenção de embolismo sistémico. Após 14 meses de seguimento, o paciente continua em tipo I de NYHA. CM-ATTR e uma doença complexa e os tratamentos deveriam ser indicados por uma equipe multidisciplinar que considere os riscos, benefícios e custos de cada intervenção.

Introduction

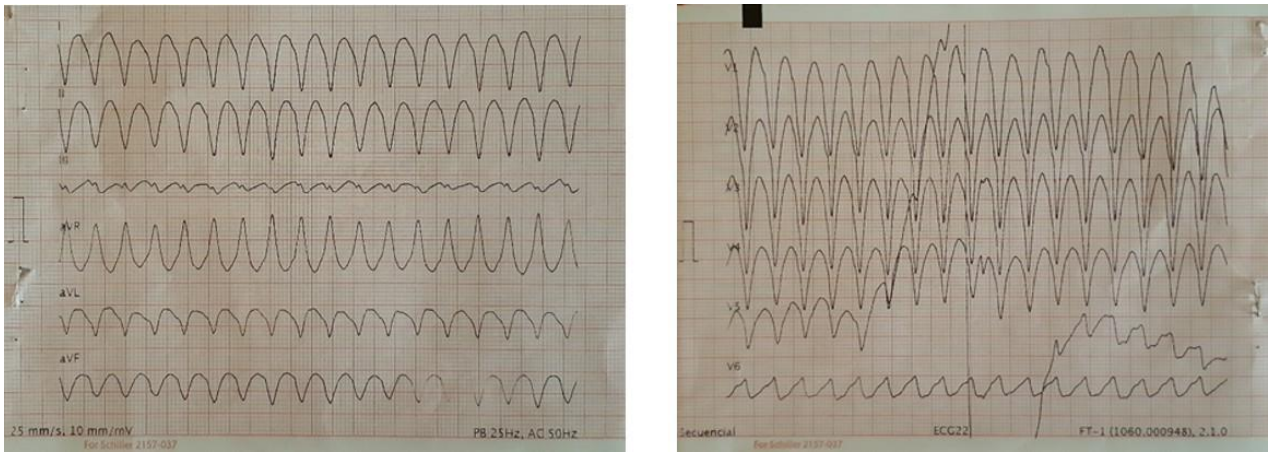
Transthyretin Amyloidosis Cardiomyopathy (ATTR-CM) is currently considered much more common than previously thought, mainly due to advances in its knowledge and in non-invasive cardiac imaging[1,2]. The clinical spectrum of ATTR-CM ranging from asymptomatic to autonomic dysfunction and syncope, heart failure with preserved ejection fraction, aortic stenosis, atrial fibrillation (AF) and conduction system disease, advanced atrioventricular block, and ventricular arrhythmias, but sustained ventricular tachycardia (SVT) are quite uncommon[1]. We report a patient without any previous symptom of ATTR-CM with a SVT as the initial presentation.

Case report

The patient provided written informed consent for this report.

A 71-years-old man woke up feeling unwell, pale, sweating, with neck pain and weakness, so his wife called the emergency service. On examination at home, blood pressure was 60/40 mmHg, and ECG showed sustained monomorphic ventricular tachycardia originated from the inferior wall of the right ventricle at 211 bpm. A biphasic electrical cardioversion was performed with 200 joules, resulting in sinus rhythm of 115 bpm with first-degree atrioventricular block and left bundle branch block, with a QTc of 333 ms (Figure 1).

A



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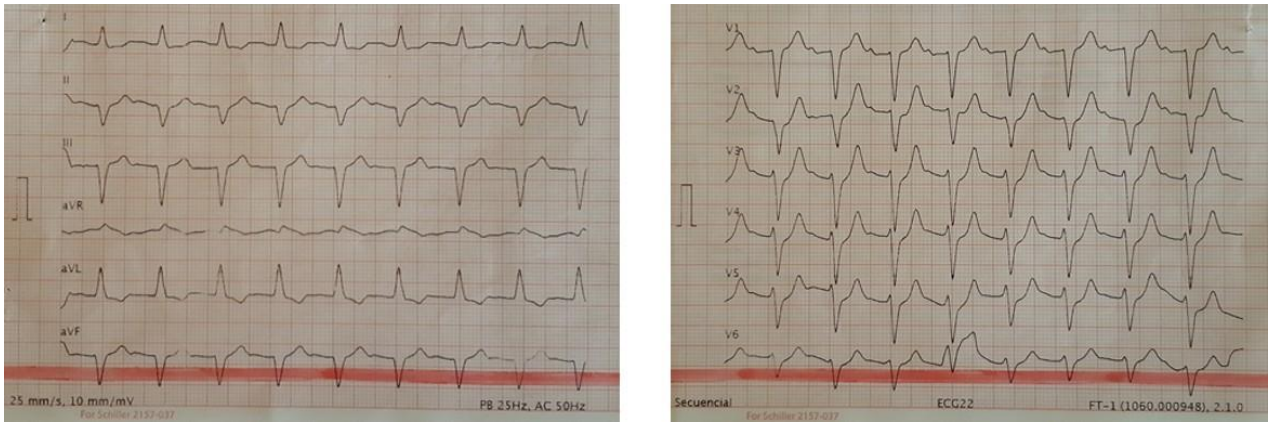

Figure N°1. ECG at home. A. Sustained ventricular tachycardia from the inferior wall of the right ventricle. B. Sinus rhythm after biphasic cardioversion with 200 joules emerging with first-degree atrioventricular block and left bundle branch block.

He was admitted to the hospital with a heart rate of 65 bpm, blood pressure 115/70 mmHg, oxygen saturation 97%, and respiratory rate 14 bpm. He had a history of hypertension and past smoking, but no chest pain or dyspnea.

He was being treated for lithiasis in the left renal pelvis with complete fragmentation of the calculus by flexible ureteroscopy and placement of a pigtail. Six years earlier, he had undergone a partial nephrectomy of the left kidney for incidental clear cell papillary carcinoma (Grade II Furham), and periodic follow-up showed no evidence of recurrence. The patient had a recent cardiac magnetic resonance imaging (MRI) suspected by his cardiologist of hypertrophic cardiomyopathy performed before his SVT, which showed wall thickening of the left ventricle (LV), late gadolinium enhancement with diffuse subendocardial signal

increase with nearly transmural extension at the basal segments, preserved systolic function (LVEF 55%), normal morphology and motility of the right ventricle (RV) (RVEF 58%) with diffuse signal intensity increased in all its walls, left atrial dilation with signal increased in both the right and left atrium and in the interatrial septum, consistent with amyloid infiltration (Figure 2).

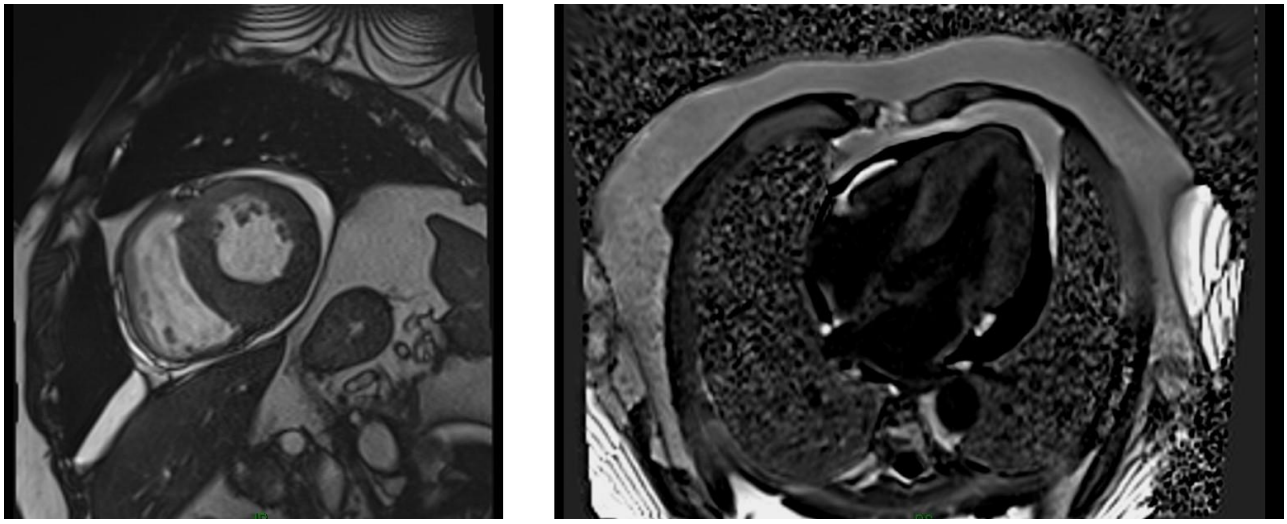

Figure N°2. Cardiac magnetic resonance imaging showing severe thickness of interventricular septum (left), and late gadolinium enhancement with increased diffuse subendocardial signal at the basal segments of the left and right ventricles and interatrial septum

Admission laboratory showed creatinine 5 mg/dL, which decreased to 1.5 mg/dL within 48 hours, hemoglobin 13.8 g/dL, hs-cTnT 98 ng/L, NT-proBNP 2622 pg/mL, sodium 134 mmol/L, potassium 4.5 mmol/L, and chloride 98 mmol/L. Echocardiogram showed interventricular septum thickness of 22 mm, left ventricular posterior wall thickness 14 mm, LVEF 61%, grade II diastolic dysfunction, average global longitudinal strain -11% with "cherry-on-top" pattern, biatrial dilation, and moderate pericardial effusion (Figure 3). A cardiac scintigraphy with 99mTc-PYP showed Perugini grade 3, without vascular pool confirming the diagnosis of cardiac amyloidosis (Figure 4).

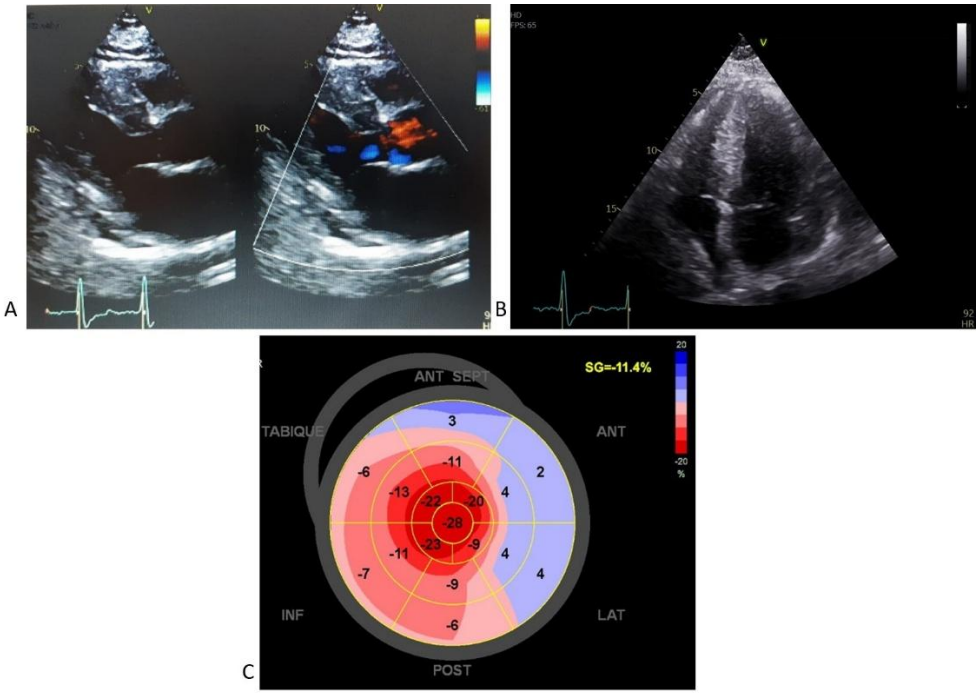

Figure N° 3. Echocardiogram. A. Parasternal long axis showing severe thickness of interventricular septum and posterior wall. B. Four chamber view showing interventricular septum thickened, biatrial dilation and moderate pericardial effusion. C. Global longitudinal strain -11% with "cherry-on-top" pattern.

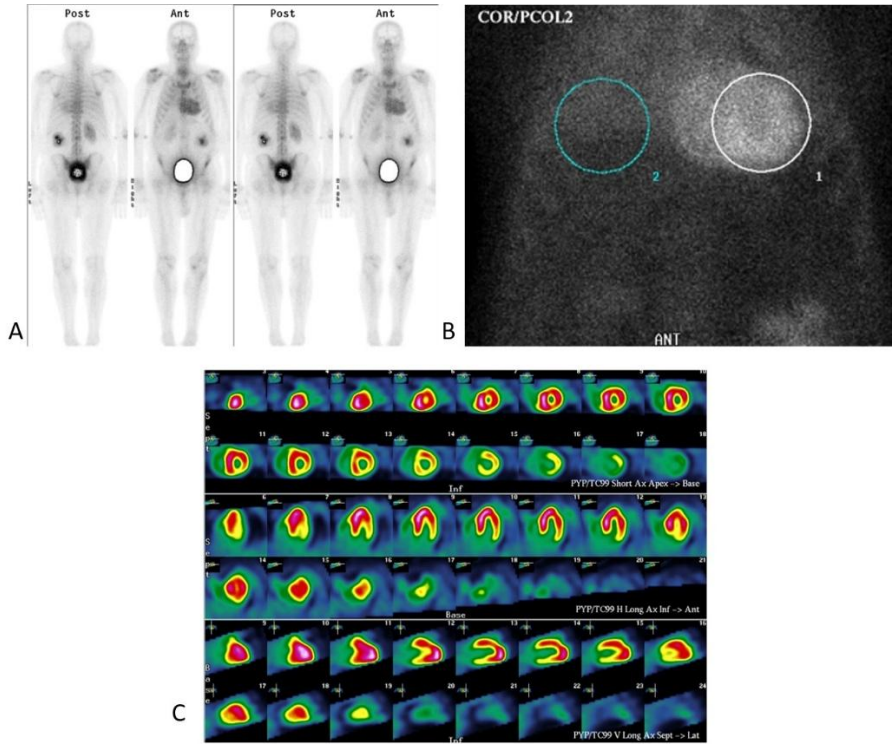

Figure N° 4. Cardiac scintigraphy with 99mTc-PYP. A. Planar whole-body bone scan in the anterior and posterior projection demonstrating intense cardiac uptake. B. 99mTc-PYP imaged at 1 hour after injection showing myocardial uptake greater than ribs (visual score grade 3). C. Additional scan was conducted at 3 hours to assess for blood pool washout.

Coronary angiography showed no obstructive lesions, only a muscular bridge in the middle third of the anterior descending artery with moderate reduction of the lumen during systole. A PET-CT with FDG-F18 looking for metastasis of the renal tumor was negative, but diffuse hypermetabolism in the myocardium and pericardial effusion was detected (Figure 5). Serum and urine immunofixation tests were negative, and serum measurement of light chains was: kappa 35.81 mg/L (reference range 3.3-19.4) and lambda 17.79 mg/L (reference range 5.71-26.3 mg/L), with a kappa/lambda ratio of 2.01 (reference range 0.37 to 3.1 for chronic renal failure). The hematologist concluded that the values did not correspond to light-chain amyloidosis (AL), and the genetic test did not detect sequence variants, so ATTR-CM wild-type was confirmed.

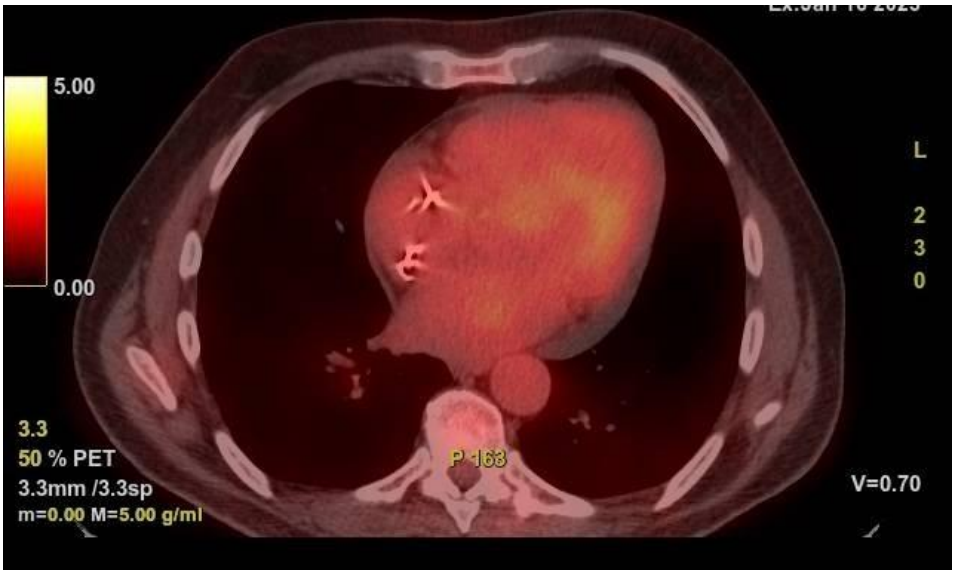

Figure N° 5. Positron emission tomography-computed tomography (PET-CT) scan with FDG-F18 showing diffuse hypermetabolism in the myocardium and pericardial effusion.

An implantable cardioverter-defibrillator (ICD) was placed, and Tafamidis 61 mg was started. The patient was discharged in stable condition, and nine days later had another SVT recorded and treated with an appropriate ICD shock; amiodarone was added and after that he did not have any further VT episodes. Before starting Tafamidis a 6-minute walk test was performed, and he covered 76% of the theoretical distance with a score of 0 on the Borg scale and O2 sat 98% without significant changes. On the 24-hour Holter monitoring, the patient had sinus rhythm with supraventricular extrasystoles and self-limited asymptomatic AF episodes, without ventricular ectopic activity, so oral anticoagulation with Rivaroxaban 20 mg was initiated. After fourteen months of follow-up, he is stable without new shocks from the ICD and NYHA class I.

Discussion

A case of ATTR-CMwt with SVT as the first clinical presentation with no history of heart failure or arrhythmias, was initially suspected of having hypertrophic cardiomyopathy, but cardiac MRI performed before the event had suggestive signs of amyloidosis. Before his cardiologist be aware of MRI, the patient suffered a SVT that threated his life and the diagnosis was confirmed during hospitalization. Cardiac amyloidosis typically presents with nonspecific symptoms and heart failure, conduction disorders or low QRS voltage on the ECG, in people over 60 years-old[1-3]. However, severe arrhythmias may be key for the diagnosis. In addition, the PET-CT showed cardiac hypermetabolism and pericardial effusion supporting the toxic-infiltrative nature of the amyloid fibers on the myocardium. Westin et al. reported an increase in the incidence of cardiac amyloidosis (CA) in people over 65 years from 0.88 per 100,000 persons/year (p/y) in the period from 1998 to 2002 to 3.6 per 100,000 p/y in 2013-2017, and 62% had heart failure, cardiomyopathy, atrial fibrillation, or pacemaker placement as their first diagnosis[4]. Chen et al. reported on 12,139 patients diagnosed with amyloidosis, an incidence of 6.54 per 100,000 p/y and CA of 0.61 per 100,000 p/y. The risk of developing VT in the amyloidosis group compared to those without amyloidosis was 7.9 ([HR: hazard ratio] 95% CI 4.49-13.9), while the risk of developing VT in the CA group compared to those without amyloidosis was 153.3 ([HR] 95% CI 54.3-432.7), and in patients with CA, the incidence of VT was significantly higher than in patients with amyloidosis without cardiac involvement (HR 53.9; 95% CI 19.9-146.4, P <0.001); they also had a higher risk of cardiovascular death[5]. In the multivariable analysis, new-onset VT was an independent predictor of cardiovascular death in patients with amyloidosis (HR 1.50; 95% CI 1.07-2.12, P <0.02)[6]. It is noteworthy that no ICD was placed during the follow-up of more than 10 years[5]. Higgins et al. reported 47.5% of VT in 472 patients with CA, and it was significantly associated with mortality[6]. Of 5,585 inpatients, Thakkar et al. reported that 36.1% were hospitalized with arrhythmias, atrial fibrillation being the most frequent (72.2%) followed by VT (14.9%)[7]. No CA registry with VT events differentiated between ATTR-CMwt and AL, so the exact prevalence of VT in each of them is unknown[8-10]. Cases of SVT have been reported especially for AL, and there are few reported in ATTR-CMwt. Oladiran et al. reported a similar case to ours in a 71-year-old man with ATTR-CMwt who presented with SVT, which is considered less frequent than non-sustained VT[11]. The severity of the progression and mortality of AL is greater than ATTR-CM, and although the cause is not yet clear, it could be attributed to the rapid deposition of fibrils and the toxic effects of amyloid light chain that produce an increase in reactive oxygen species, oxidative stress with alterations in intracellular calcium, impairing the contraction and relaxation of myocytes, lysosomal, mitochondrial and microvascular dysfunction[12]. In ATTR-CM, the same process is probably similar but slower. The possible arrhythmogenic substrate was studied combining ECG images with cardiac MRI: the ventricular conduction is slow and spatially heterogeneous, repolarization is slow and spatially dispersed, and epicardial potentials are of low

amplitude. Patients with AL have a lower total amyloid volume than ATTR-CM, electrophysiological abnormalities are more notable: lower epicardial signal voltage, higher degree of signal fragmentation, and greater dispersion of repolarization. In all patients with CA, an inverse correlation between T1 and signal amplitude was observed, and the higher the extracellular volume, the greater signal and repolarization time fragmentation[13].

The indication for ICD for primary prevention is controversial: the European Consensus does not routinely recommend ICD for primary prevention, but it is recommended for secondary prevention[3]. The American Guidelines for the prevention of Sudden Death recommend an individualized decision due to the shortage of data to make a recommendation[14] and the Heart Rythm Society recommends the placement of an ICD for those who survived a cardiac arrest if a survival of >1 year is expected, without differentiating ATTR-CM from AL; on the other hand, for AL in which non-sustained VT was confirmed, prophylactic ICD is recommended if a survival of >1 year is expected[15].

In our patient, the indication for ICD was based on the presence of SVT with severe hemodynamic deterioration that could evolve to ventricular fibrillation (VF) and sudden death. In fact, the event recurred after the ICD placement, and an appropriate discharge was provided by the device. The combination of an antiarrhythmic (amiodarone), a transthyretin stabilizer (tafamidis), an oral anticoagulant (rivaroxaban) for prevention of systemic embolism, and ICD for additional episodes of VT/VF, are treatments that should be indicated by a team that considers the risks, benefits, and costs of each intervention[2].

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Limitaciones de responsabilidad:

La responsabilidad del trabajo es exclusivamente de quienes colaboraron en la elaboración del mismo.

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Este artículo es original y no ha sido enviado para su publicación a otro medio de difusión científica en forma completa ni parcialmente.

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