

detected other associated factors that could prolong QTc (electrolyte abnormalities, bradycardia, etc.), which could enhance the proarrhythmic effect of the drug.⁵

In our patient, QTc prolongation showed a clear temporal relationship with amiodarone dosing both in terms of onset (24 h afterwards) and disappearance (1 week after the drug was discontinued).

Although slight hypopotassemia was observed, TdP did not cease when the levels were corrected, which suggests that although the condition could have enhanced the proarrhythmic effect of the drug it was not the main cause.

In conclusion, although amiodarone is considered safe for the treatment of ventricular arrhythmia, its arrhythmogenic potential should not be underestimated, particularly in women and in the presence of concomitant factors that could prolong the QTc. Careful monitoring of the QTc interval and these factors can lower the risk of proarrhythmia.

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Extreme QT Interval Prolongation and Helicoid Ventricular Tachycardia (Torsade de Pointes) in Non-ST-Elevation Acute Coronary Syndrome

Prolongación extrema del intervalo QT y taquicardia helicoidal (torsade de pointes) en el síndrome coronario agudo sin elevación del ST

To the Editor,

A prolonged corrected QT interval (cQT) during coronary ischemia is a well known sign that has even been incorporated into the parameters tested for the assessment of ischemic risk in acute coronary syndrome (ACS).¹ There is also a correlation

between a long QT and helicoidal ventricular tachycardia, or torsade de pointes (TdP), that has been described in this context.² Here, we present a case of ACS with a very prolonged QT interval with giant negative T-waves and a later development of TdP.

A 79-year-old woman sought emergency treatment for diffuse pain in the anterior thorax and dyspnea with 2 days evolution. She had a background of type-2 diabetes mellitus, systemic arterial hypertension, and rheumatoid arthritis. She was under treatment with metformin, vildagliptin, losartan, and indometacin. Upon admission to the hospital, the patient was dyspneic with a blood pressure of 219/96 mmHg, an O₂ saturation of 86%, and a regular pulse at 98 bpm, with notable bilateral basal crepitation. The abdomen was without abnormalities, with intact peripheral pulse. The initial electrocardiogram (ECG) (Fig. 1A) demonstrates a sinus

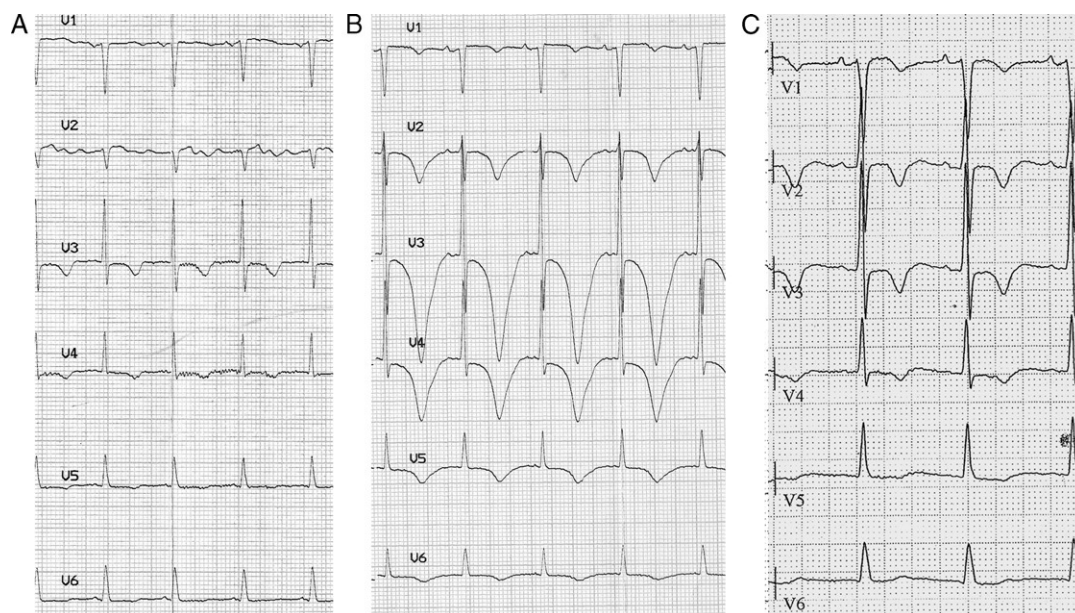


Figure 1. A, test results upon admission: RR, 640 ms; corrected QT interval, 510 ms. B, test results after 24 h: RR, 780 ms; corrected QT interval, 745 ms. C, test results after 13 days: RR, 760 ms; corrected QT interval: 504 ms. (Corrected QT intervals derived using Bazett's formula).

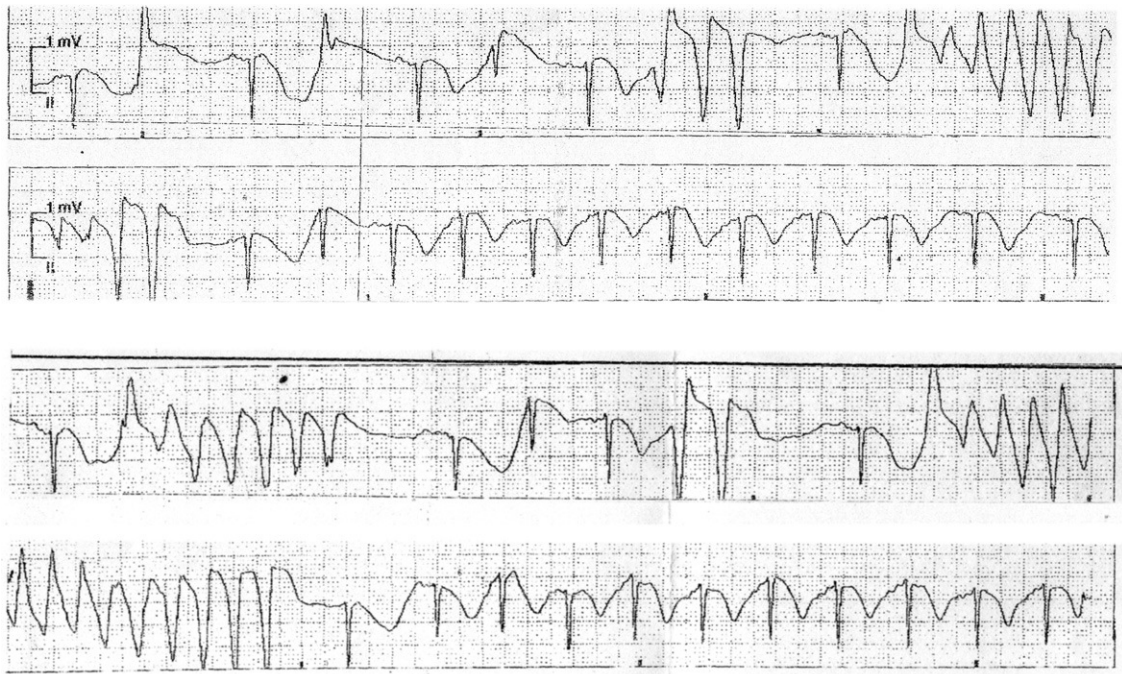


Figure 2. Torsade de pointes (electrocardiogram monitoring following Figure 1B).

rhythm of 94 bpm, PR of 125 ms, QRS of 9 ms, without a significant decrease in ST, asymmetrical negative T-waves at V3 and V5, and a cQT of 510 ms. Laboratory analyses indicated normocytic hypochromic anemia, creatinine at 1.4 mg/dl, sodium at 138 mEq/l, potassium at 4.5 mEq/l, and magnesium at 1.8 mg/dl. The troponin T test was positive for myocardial injury. An echocardiogram indicated that the left ventricle had septal hypertrophy, septoapical hypokinesia and anteroapical akinesia, with an ejection fraction of 39%. The diagnosis was made of non-ST elevation left heart failure in the context of ACS, hypertension, and normocytic anemia. The patient was treated using double antiplatelet, anticoagulant, and vasodilator drugs. After 24 h, the patient was stable, with blood pressure at 140/85 mmHg and the control ECG (Fig. 1B) demonstrated giant negative T-waves from V2 to V5 with a cQT of 745 ms. We monitored the patient using ECG, revealing bouts of type TdP polymorphic ventricular tachycardia (Fig. 2), which ceased upon intravenous administration of 2 g magnesium sulphate. Serial measurements of markers for myocardial damage were congruent with a myocardial infarction, excluding other possible causes of the prolonged cQT.

A cardiac catheter test demonstrated a calcified lesion along 10% of the common arterial trunk and a lesion along 70% of the anterior descending artery. We treated the patient by implanting a stent, and later evolution was favorable. A control echocardiogram indicated that the patient did not suffer contractile deficit, and systolic function was normal. In the ECG taken 13 days later (Fig. 1C), the negative T-waves continued between V1 and V5, and the cQT remained at 504 ms. The QT interval normalized after 6 weeks.

The relationship between myocardial ischemia and a reversible prolongation of the QT interval has been shown during controlled coronary occlusion in angioplasty procedures, and it has been documented that prolongation of the cQT is the most concordantly altered electrocardiographic variable during the early phase of transmural ischemia.³ The incidence of helicoidal ventricular tachycardia in myocardial infarctions is close to 2%,^{2,4} and is most

closely associated with occasional extremely prolonged cQT. These cases have been discussed under the term of acquired long QT syndrome associated with infarction.⁴ TdP ventricular tachycardia associated with prolonged cQT infarction tend to be preceded by the (pause-dependent) short-long-short cycle phenomenon, and its appearance is greatest in the days directly following an infarction.⁴

As research continues to delve into the exact mechanisms by which the QT interval is prolonged in ACS, there is ever more information regarding its etiology and implications. The underlying structural mechanisms of prolonged QT intervals are related to the increase in electric heterogeneity due to ischemia-induced changes in the ion permeability of the sodium/potassium pump and an increase in the duration of myocytic membrane potentials in the different levels of the myocardium, the middle myocardial layer in particular, with consequent uncoupling and dispersion of the repolarization.^{4,5} Other studies have proposed the role of genetic causes, such as in other long QT syndromes. A mutation has been detected on the *SCN5A* gene, associated with altered sodium channels in one patient with an arrhythmic storm during an acute infarction. The polymorphisms known to cause the long QT phenotype have been observed between days 2 and 11 following an infarction, which provides the foundation for a genetic predisposition to prolonged QT intervals and the appearance of TdP.⁶

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Diagnostic Challenge of Annular Abscess in a Patient With Prosthetic Aortic Valve: Can F-Fluorodeoxyglucose Positron Emission Tomography Be Helpful?

Reto diagnóstico de un absceso anular en una paciente con válvula aórtica protésica: ¿puede ser útil la tomografía de emisión de positrones con F-fluorodesoxiglucosa?

To the Editor,

A 30-year-old woman was admitted because of asthenia and fever episodes (>39 °C) during the previous month. She had received implantation of a mechanical prosthetic aortic valve (St Jude Medical 21) in 2003. The patient underwent transthoracic echocardiography, which revealed normal native and mechanical valves. Vegetations or possible abscess were not observed. Because endocarditis was suspected, we proceeded with transesophageal echocardiography (TEE), which confirmed the absence of vegetation. However, in longitudinal view we noticed a thickened area of 3 mm at the level of the noncoronary sinus of Valsalva that was not accompanied by hypoechoic or gelatinous extra echoes that could have suggested the presence of an abscess (Fig. 1). Blood cultures were positive to *Streptococcus sanguinis*. The patient received antibiotic treatment with penicillin and gentamicin.

Because the TEE result was regarded as inconclusive, we decided to perform F-fluorodeoxyglucose positron emission

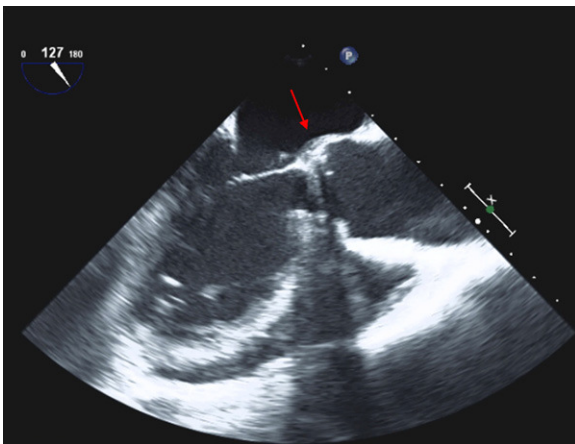


Figure 1. Transesophageal echocardiogram showed a thickened area of 3 mm at the level of the noncoronary sinus of Valsalva without hypoechoic or gelatinous extra echoes that suggest the presence of an abscess.

tomography/computed tomography (FDG-PET/CT) and to repeat TEE a week later. The FDG-PET/CT scan showed a major FDG uptake at the level of the mechanical aortic valve that suggested the diagnosis of periannular abscess (Fig. 2, Video). A second TEE did not reveal any changes. Follow-up blood cultures 5 days after initiation of antibiotics were negative; however, a low-grade fever persisted.

Based on the assumption that the FDG-PET/CT image was compatible with a diagnosis of periannular abscess and that patients with this diagnosis usually have a high mortality on medical therapy, it was decided to perform an exploratory surgery. The prosthetic valve was removed and the Ross procedure was done. Surgically excised tissue was sent for microbiology and pathology analysis that confirmed the diagnosis of periannular abscess and the presence of the *S. sanguinis*. Postoperative echocardiography revealed a well-functioning aortic valve. More than 6 months after surgery, the patient is doing well and follow-up blood cultures are negative.

In clinical practice, the diagnosis of infective endocarditis (IE) is often difficult, and both overdiagnosis and underdiagnosis are observed. Echocardiography represents the central role in the evaluation of patients who have a clinical presentation suggestive of IE. In the majority of published studies, transthoracic echocardiography and TEE sensitivity ranges between 40% and 63% and between 87% and 100%, respectively. Perivalvular abscesses are particularly common in prosthetic valve IE, since the annulus is the usual primary site of infection. This serious complication has been reported in up to 40% of patients with native aortic valve IE and the incidence is higher in patients with prosthetic aortic valve IE.¹

Usually an abscess is defined as a thickened area or a mass within the myocardium or annular region with a nonhomogeneous echogenic or echolucent appearance.² In most studies the criterion used to define a periannular abscess included the notion of a thickened area ≥ 10 mm.³ However, this definition may lack sensitivity for the diagnosis of abscess since the echocardiographic appearances of aortic root abscesses ranged from a diffusely thickened aortic root in early cases to multiple echolucent spaces near the aortic annulus in more advanced cases.⁴

Mortality in patients with periannular abscess involving prosthetic aortic valves is up to 70% on medical therapy.¹ The presence of an aortic root abscess is usually an indication for urgent surgery; a rapid and accurate diagnosis is essential if perioperative morbidity and mortality are to be reduced and surgical repair facilitated. In a recent study, 57% of patients with prosthetic valve endocarditis who needed urgent surgery presented some type of periannular complication.⁵ In the setting of suspected prosthetic valve IE, negative or inconclusive TEE