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

Alfonso Jurado Román,* Belén Rubio Alonso,
Roberto Martín Asenjo, Rafael Salguero Bodes, María López Gil, and Fernando Arribas Ynsaurriaga

Servicio de Cardiología, Hospital Universitario 12 de Octubre, Madrid, Spain

*Corresponding author:
E-mail address: alfonjroman@hotmail.com (A. Jurado Román).
Available online 2 August 2011

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doi: 10.1016/j.rec.2011.05.018

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). 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. 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

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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).
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 hyper-
chomic 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, septoa-
pical 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
sten, 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. The incidence of helicoidal ventricular
tachycardia in myocardial infarctions is close to 2%, 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. TdP ventricular tachycardia
associated with prolonged QT 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.

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 myocardic membrane potentials in the
different levels of the myocardium, the middle myocardial layer in
particular, with consequent uncoupling and dispersion of the
repolarization. Other studies have proposed the role of genetic
causes, such as in other long QT syndromes. A mutation has been
detected on the SCNSA 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.

Montse Vilaseca-Corbera, Gabriel Vázquez-Oliva, Cristina Campoamor-Cela, Alberto Zamora-Cervantes, Joan Bassanyanes-Vilarrasa, and Rita Massa-Puig

*Unidad de Cardiología, Hospital Comarcal de Blanes, Corporació Salut Maresme Selva, Blanes, Girona, Spain
bServicio de Medicina Interna, Hospital Comarcal de Blanes, Corporació Salut Maresme Selva, Blanes, Girona, Spain
Unidad de Hemodinámica Cardíaca, Servicio de Cardiología, Hospital Josep Trueta, Girona, Spain
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 transeosophageal 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 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 perianular 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 perianular 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 perianular 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.

Usually an abscess is defined as a thickened area or a mass within the myocardium or annular region with a nonhogeneous echogenic or echoluent appearance. In most studies the criterion used to define a perianular abscess included the notion of a thickened area ≥10 mm. 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 echoluent spaces near the aortic annulus in more advanced cases.

Mortality in patients with perianular abscess involving prosthetic aortic valves is up to 70% on medical therapy. 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 perianular complication. In the setting of suspected prosthetic valve IE, negative or inconclusive TEE