

Qtc Prolongation after a Negative Dipyridamole Pharmacological Stress Study

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Abstract

QTc interval prolongation after dipyridamole administration was reported only in patients who had positive pharmacological stress test results. We report a case of QTc prolongation after dipyridamole administration with no evidence of myocardial ischemia.

Case presentation

A 73 year old female patient, with hyperlipidemia treated with statins and a history of low grade lymphoma for which she underwent splenectomy at 1998. No further treatment was needed. The patient underwent a coronary angiography in 1997 due to chest pain, which was summarized as normal coronary angiography.

At May 2013 the patient was examined in the outpatient clinics for atypical chest pain and effort dyspnea. An echocardiography showed normal sized heart chambers, normal systolic function of both left and right ventricles, mild aortic and mitral regurgitation, and normal pulmonary artery pressure. No evidence of left ventricular hypertrophy. Laboratory tests were normal, including kidney function tests, liver function tests, and electrolytes. For further evaluation she was referred for a myocardial perfusion imaging with pharmacological stress using dipyridamole.

Her baseline electrocardiography showed normal sinus rhythm, heart rate of 55/minute, PR interval of 160 msec, narrow QRS complex, and QTc interval (calculated using the Bazett formula) of 417 msec (Figure 1).

The patient weighed 80 kg and accordingly received 45 mg dipyridamole infused during 4 minutes. At one minute through the infusion of dipyridamole her ECG showed normal sinus rhythm, QTC was 440, no other changes compared to the baseline ECG was seen.

She completed 2 minutes walk in a low velocity on a treadmill according to the protocol. Her heart rate raised to 90 beats per minute and her QTc interval was 582 msec (Figure 2) at 2.50 minutes after the cessation of the dipyridamole infusion. The gradual prolongation of QTc interval was noticed during dipyridamole infusion and walking on treadmill. At 3 minutes past the cessation of the infusion her QTc interval was 550 msec, and at 5 minutes past the infusion it was 423

msec. Notably, Tc99m-MIBI was injected about 3 minutes after the cessation of dipyridamole infusion.

There were no other ischemic changes in the ECG along the test and during monitoring after the test. Her myocardial perfusion imaging was negative for perfusion defects and was concluded as normal (Figure 3). It also showed Normal left ventricular size, normal left ventricular function with an ejection fraction of 68%.

Discussion

Dipyridamole is an indirect acting coronary vasodilator used for pharmacological stress tests for studying coronary blood reserve and for revealing myocardial areas supplied by stenotic coronary arteries. It inhibits cellular reuptake of adenosine causing inappropriate accumulation of adenosine, which represents the main cause of arteriolar vasodilation determining a myocardial flow mal distribution [1].

Guideri et al [2]. Reported a significant prolongation of the QTc interval after dipyridamole and adenosine administration only in patients who had positive test results indicating myocardial ischemia [2]. In patients with unstable angina pectoris, QTc interval prolongation represented an independent marker of risk, and was associated with the presence of more severe coronary artery disease and poor short-term clinical outcome [3]. In other study QT dispersion increased during peak infusion of dipyridamole with a time course mirroring that of development of contractile abnormalities in patients with positive dipyridamole stress test findings [4].

In this case report, QTc prolongation was not associated with any sign of ischemia in contrast to the study of Guideri et al. [2]. QTc interval prolongation was suggested to be a consequence of an altered repolarization of the cell in ischemic myocardial tissue due to local electrolyte changes rather than to a direct Pharmacological effect

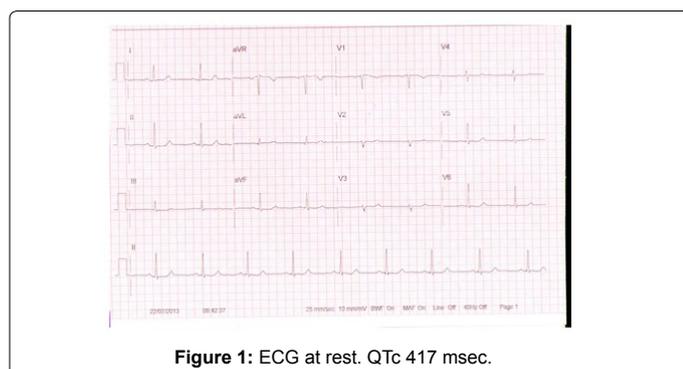


Figure 1: ECG at rest. QTc 417 msec.

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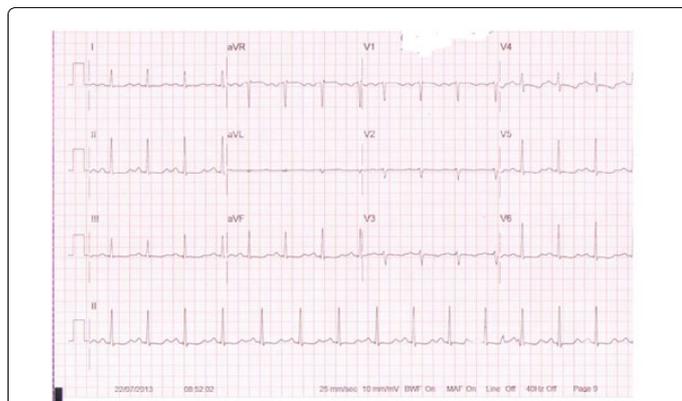


Figure 2: Maximal QTc (582 msec) prolongation after the end of dipyridamole infusion.

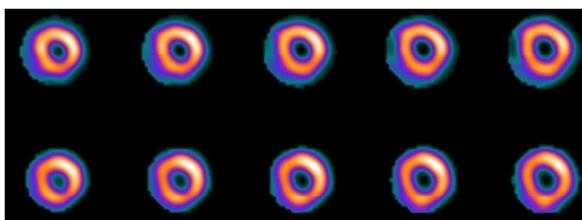


Figure 3: Myocardial perfusion SPECT was performed using a single day single isotope pharmacological stress test protocol using 0.21 MBq (8 mCi) Tc99m-MIBI for rest and 0.68 MBq (25 mCi) for stress. SPECT short axis slices (upper row–stress, lower row–rest) demonstrate normal and homogenous tracer distribution in all left ventricular walls.

of dipyridamole or adenosine [2]. The reason for QTc prolongation in this case is not clear. In addition, it is not clear why maximal QTc prolongation occurred at 2.50 minutes after the cessation of dipyridamole infusion. It could be related to unknown direct effect of dipyridamole unrelated to the presence or absence of myocardial ischemia.

False negative result or balanced ischemia as a possible explanation for QTc interval prolongation cannot be ruled out. However, we think that the patient most probably had no ischemia because of clinical, electrocardiographic and imaging finding during stress test and absence of cardiac events during follow-up. Thus, coronary angiography was not undertaken recently. In addition the myocardial perfusion imaging is more accurate and valuable in demonstration/exclusion of myocardial perfusion abnormalities compared to coronary angiography.

We like to emphasize that this was a single case, for which we have no good explanation, and its significance is not yet known.

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