中文題目：急性心肌梗塞後所發生的 Torsades de pointes

英文題目：Torsades de pointes occurred during the subacute phase of acute myocardial infarction

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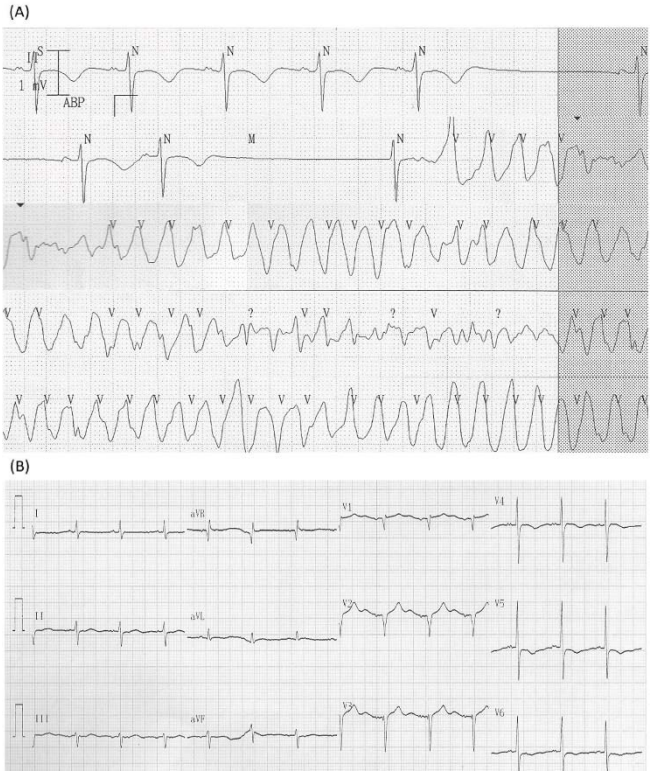
Introduction

Torsades de pointes (Tdp) is a polymorphic ventricular tachycardia associated with a prolongation of QT interval which was first described by Dessertenne in 1966.[1] Prolongation of QT interval in patients with acute myocardial infarction (AMI) is not rare. However, Tdp induced after AMI is not often.

Case Presentation



An 81-year-old male presented with chest tightness and cold sweating. AMI was diagnosed by ST-segment elevation in the leads of V1 through V3 in 12-lead electrocardiogram (ECG). The corrected QT (QTc) interval was normal at that time. The primary percutaneous transluminal coronary angioplasty (PTCA) was performed, which revealed a lesion with 99% of stenosis in the left main coronary artery, and another lesion with 60% stenosis in the left anterior descending artery. A bare metal stent was implanted in the left main trunk.
However, three days after the PTCA, an episode of Tdp lasting for 8.6 seconds followed by a pause was noted on the ECG monitor (Figure A). The QTc interval significantly prolonged to 529 ms in the 12-lead ECG and the serum electrolytes levels were all within normal ranges. Due to the suspicion that frequent Tdp episodes were induced by myoischemia, the second PTCA for left anterior descending artery lesion was performed. The QTc interval was shorten to 411 milliseconds (Figure B) and no more Tdp was recorded after the second PTCA.

Discussion

The prolongation of QT interval reflects a delay in myocardial repolarization caused by multiple factors.[2] The QT interval prolongation after myocardial infarction is a well described phenomenon and some reports have demonstrated that post-myocardial infarction patients with prolongation of QT interval are at a significantly high risk of sudden cardiac death.[3]

Tdp preceded a sudden slowing of heart rate was referred to as “pause-dependent Tdp”. Halkin et al. have made a survey of pause-dependent Tdp following AMI and concluded that the incidence of Tdp following AMI is 1.8% and most of Tdp occurred 3 to 11 days after infarction.[4] The prolongation of QT interval could be recovered and no more Tdp occurred in these patients during following up period. Similar to the report described, the prolongation of QT interval and Tdp occurred on the 4th day after infarction and the QT interval was recovered after completely relieving myoischemia in our

patient. No more Tdp occurred after discharged. This may suggest that myoischemia could be an important trigger of pause-dependent Tdp after AMI.

Crotti et al. investigated the genetic substrate in the patients with Tdp after myocardial infarction.[5] They found that K897T polymorphism is associated with an increased risk of Tdp developing in the subacute phase of acute myocardial infarction.

In conclusion, we presented a patient with Tdp occurred in the subacute phase of AMI. No more Tdp occurred after the myoischemia was completely relieved. The results from this patient and previous studies may suggest that myoischemia and genetic substrate play important roles in the genesis of Tdp occurred in the subacute phase of AMI.

Reference

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