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Clinical case report based study

Torsade de pointes as a reperfusion arrhythmia following intravenous thrombolytic therapy

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ABSTRACT

Many types of cardiac arrhythmias have been noted following acute myocardial infarction. Polymorphic ventricular arrhythmias (polymorphic ventricular tachycardia and ventricular fibrillation) related to an acute myocardial infarction generally strike during the hyperacute phase, are clearly related to ischaemia and are not associated with a long QT interval time.

Pause-dependent Torsade de pointes has been reported following acute myocardial infarction and this arrhythmia generally occurs 3–11 days after the onset of acute myocardial infarction and none has been reported during the hyperacute phase. Torsade de pointes – a specific ventricular tachycardia with specific characteristics has been described in hypokalemia, hypomagnesaemia, during Quinidine therapy, and while using phenothiazines and tricyclic antidepressants. It is reported following liquid protein diet, brady-arrhythmias [especially III° AV Block], sick-sinus syndromes.

Torsade de pointes either pause-dependent or pause-independent occurring directly as a reperfusion arrhythmia during intravenous thrombolytic therapy has not been reported in the literature to the best of the authors knowledge.

Here, an episode of Torsade de pointes as a direct consequence of reperfusion following thrombolytic therapy in a patient of acute myocardial infarction is described.

1. Case report

A 55-year male presented with central chest pain of one-hour duration and electrocardiogram showed acute antero-septal myocardial infarction (Fig. 1). The patient was a smoker with a recent history of mild hypertension and was not on any medications for hypertension or for any other ailments. Examination revealed a heart rate of 75 beats/min, B.P.110/76 mm Hg without any cardiovascular abnormalities. He was in Killip's class I. Electrocardiographic monitoring was begun. Intravenous nitroglycerine, aspirin and clopidogrel were administered immediately. IV Streptokinase 1.5 million units/60 min was commenced. Chest pain subsided within 10 min of starting the thrombolytic therapy. While the patient was being monitored an interesting polymorphic ventricular tachycardia – Torsade de pointes was observed and recorded as he was receiving the thrombolytic agent (Fig. 2). This episode of ventricular arrhythmia lasted for about 3 s. QRS duration was 90 ms during sinus rhythm and Torsade de pointes manifested as a broad complex tachycardia with a QRS duration of 180 ms. The QT-interval was not prolonged. There was no sinus bradycardia prior to the occurrence of the arrhythmia. The patient remained haemodynamically stable during and after the arrhythmia. After 5 min of occurrence of this arrhythmia evidence for reperfusion was observed on the electrocardiogram – a drop of Σ ST by more than 8 mm (Fig. 3). The time taken after starting the thrombolytic agent to the occurrence of the arrhythmia was 30 min.[1] Laboratory reports of serum electrolytes including serum potassium serum magnesium and serum calcium was reported normal [4.8 mEq/L, 2.7 mEq/L and 9.5 mEq/L respectively]. Cardiac biomarker Troponin T 6 h after onset of chest pain was elevated [842 μg/L]. The patient had an uneventful recovery except for a few insignificant Ventricular premature beats noted during the first 3 h. A coronary angiogram after 5 days showed recanalised left anterior descending artery with a residual proximal 20% stenosis. The patient was well and stable when last seen in March 2007.

2. Discussion

Following thrombolytic therapy, reperfusion occurs with a frequency of about 74%.[2] During this process arrhythmias have been noted.[3] These are likely to occur due to (i) Heterogeneity of recovery of excitability resulting in re-entry or enhanced ventricular

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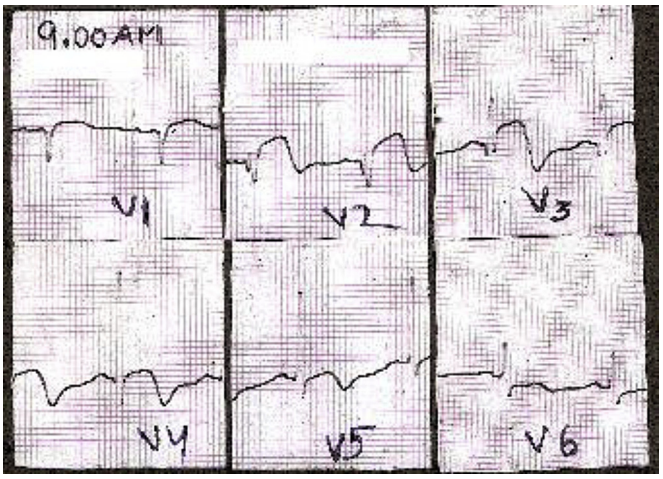

Fig. 1. Pre-streptokinase electrocardiogram showing acute antero-septal infarction with lateral wall ischaemia.

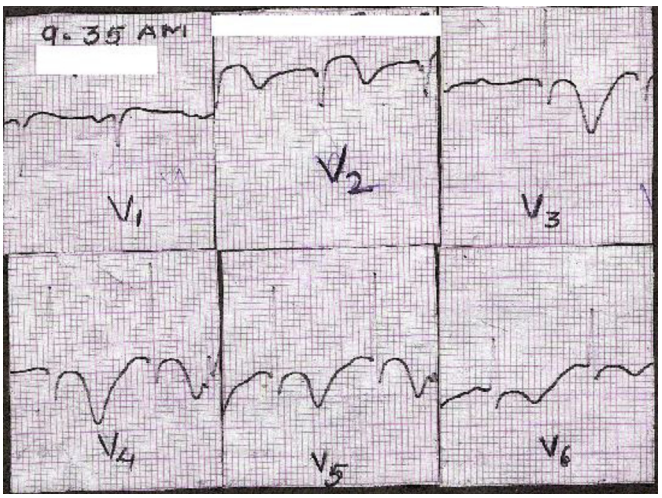

Fig. 3. Electrocardiogram shows ST-segment almost isoelectric.

automaticity. (ii) Rapid changes in potassium, pco2 and intracellular calcium, (iii) Alterations in the regional concentration or tissue compartment localisation of amphiphilic lipid metabolites such as lysophosphoglycerides and long chain acyl-carnitines, or, (iv) Alpha 1 adrenergic stimulation.[4] The mechanism[s] underlying Torsade de pointes remains uncertain, although work with a canine model suggested that the characteristic "spindles" which link morphologically distinct QRS complexes during the arrhythmia represent fusion of two colliding cycles of epicardial depolarisation.[5] Pause-dependent Torsade de pointes has been reported following MI. The maximal QT prolongation and QT-related arrhythmias occurred 3 days–11 days after the onset of MI and in one study, none of the patients had ventricular arrhythmias during the hyperacute phase. Also, the sequence of events, with numerous arrhythmic episodes documented three days after hospitalization, suggested that myocardial infarction played the main role in triggering the arrhythmias.[6] This interesting ventricular tachycardia Torsade de pointes either pause-dependent or pause independent has not been described so far as a reperfusion arrhythmia in a setting of an acute myocardial infarction during ongoing treatment with thrombolytic therapy. The patient had complete relief from chest pain within 10 min after starting thrombolytic therapy and developed the arrhythmia about 30 min after starting streptokinase. This was almost immediately (5 min) followed by electrocardiographic evidence of reperfusion even as Streptokinase infusion was about to finish. Thus, this interesting arrhythmia is most likely to have been triggered by the ongoing process of reperfusion rather than ischaemia, given the time interval between the complete relief of pain and the occurrence of the arrhythmia. Kato et al reported Torsade de pointes during reperfusion in acute myocardial infarction and all patients received mechanical revascularization therapy within 6 h of onset of symptoms. A subset of these patients were treated with intracoronary verapamil (0.25–1.0 mg) to terminate the reperfusion-induced tachyarrhythmia.[7] The difference between the two was that no mechanical revascularization was attempted during the acute phase and intravenous thrombolysis was instituted in our patient. Also, no further therapeutic measures with antiarrhythmic drugs were required as the arrhythmia showed spontaneous termination [3 s] without any haemodynamic compromise.

Polymorphic ventricular arrhythmias (polymorphic ventricular tachycardia and ventricular fibrillation) related to an acute myocardial infarction can generally strike during the hyperacute phase, and are clearly related to ischaemia and are not associated with a long QT interval.[6,8,9] But, in this patient, a ongoing ischaemia triggering the arrhythmia was unlikely as chest pain had completely subsided within 10 min after starting thrombolytic therapy. Soon after the polymorphic ventricular tachycardia occurred, the electrocardiogram showed ST-segment to be almost isoelectric, thus strongly indicating that the Torsade de pointes was a most likely a reperfusion arrhythmia. A coronary angiogram after 5 days showed recanalised left anterior descending artery with a residual proximal 20% stenosis Irrespective of TIMI-flow in the epicardial infarct-related artery, ST-segment resolution has the

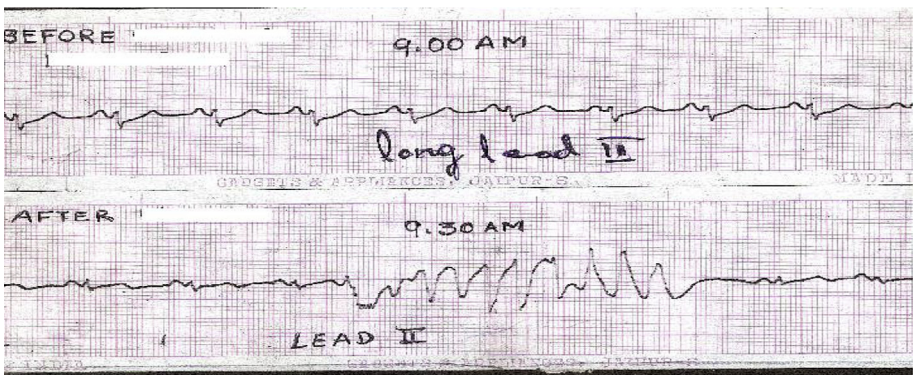

Fig. 2. Torsade de pointes recorded while on streptokinase infusion.

advantage of measuring successful reperfusion at the myocardial tissue level.[10]

The clinical significance of this rare event is to underline the need for constant monitoring for ventricular arrhythmias including Torsade de pointes during intravenous thrombolytic therapy. It also shows that apart from ischaemia, Torsade de pointes can occur as a reperfusion arrhythmia during intravenous thrombolysis for acute myocardial infarction.

Continuous monitoring during intravenous thrombolytic therapy helped to elucidate the process of myocardial reperfusion, Torsade de pointes and ultimate recovery in this patient.

Conflicts of interest

The author has none to declare.

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