

T-wave alternans: a harbinger for malignant ventricular arrhythmias

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DESCRIPTION

A 50-year-old male chronic alcoholic presented to our emergency department with seizures and loss of consciousness after an alcoholic binge. He had no other significant medical history and was not on any regular medications. On examination, his pulse was 90 beats/min and his blood pressure was 80/60 mm Hg. His resting ECG showed sinus rhythm with QT prolongation. His serum sodium was 133 mmol/dL, potassium was 3.5 mmol/dL, magnesium was 1.4 mmol/dL and calcium was 6.6 mg/dL. The ECG demonstrated wide QRS tachycardia with a heart rate of approximately 200 beats/min and mild irregularity and morphology suggestive of polymorphic ventricular tachycardia initiated by a ventricular premature contraction falling on the terminal part of the T wave of the preceding sinus beat (figure 1A, black arrows). The postelectroversion tracing showed sinus rhythm at a rate of 55 beats/min, with alternating small, narrow (figure 1B, red arrows) and broad deep T-wave inversions (figure 1B, black arrows) with a measured QT interval of 800 ms and QTc of 769.80 ms suggestive of T-wave alternans (TWA). An emergency two-dimensional echocardiogram showed no regional wall motion abnormality and a normal ejection fraction of 64%. Corrective measures were instituted to restore electrolyte imbalance on an urgent basis, but he subsequently developed an episode of sudden-onset unresponsiveness. Intravenous magnesium sulfate 2 g bolus dose was administered followed by infusion at 10 mg/min via central venous catheter and norepinephrine infusion at 15 mcg/min was started in view of hypotension. Inj Calcium Gluconate 10% 100 mL was given in

100 mL normal saline over 30 min. Attempts at correction of the electrolyte disturbances were unsuccessful, and he developed a ventricular fibrillation and expired despite attempts at resuscitation.

TWA refers to beat-to-beat variability in the timing or shape of T waves on the surface ECG. It is distinct from electrical alternans totalis, as TWA does not involve the QRS complex. TWA is well known to be a harbinger of malignant ventricular arrhythmias.¹ The most common form is a microvolt TWA which is undetectable in standard 12-lead ECG and requires advanced signal processing techniques like spectral and modified moving average methods for demonstration.² Rarely as in our case, a macrovolt TWA is demonstrable in the ECG tracing. The presence of TWA might serve as a potential guide for deciding the need for prophylactic implantable cardioverter-defibrillator in patients at risk for sudden cardiac death, namely those with cardiomyopathies, hereditary channelopathies and postmyocardial infarction. This finding is indicative of temporal differences in ventricular repolarisation, an important mechanism underlying re-entrant arrhythmias in TWA.³ Patients with acquired long QT syndrome usually develop torsade during periods of bradycardia. The most common causes of acquired long QT syndrome are medications and electrolyte disorders (eg, hypokalaemia, hypomagnesaemia). Several epidemiological studies have identified chronic excessive alcoholism as a risk factor for QT prolongation.⁴ Our patient was a chronic alcoholic and had hypomagnesaemia and hypokalaemia. Hypomagnesaemia in alcoholics is due to inappropriate magnesuria, possibly due to hypophosphataemia, metabolic acidosis or a direct

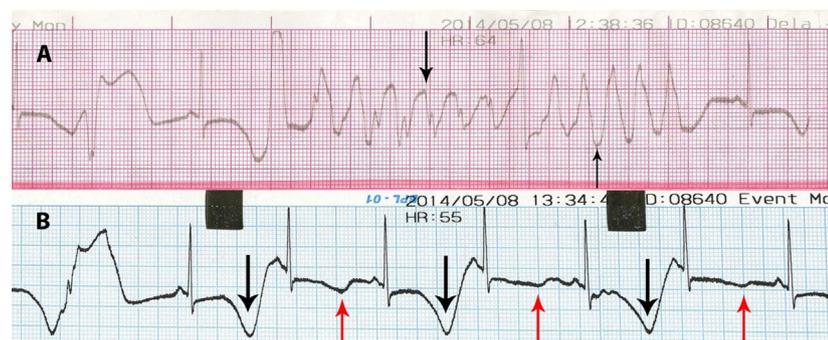


Figure 1 ECG demonstrating wide QRS tachycardia with a heart rate of approximately 200 beats/min and mild irregularity and morphology suggestive of polymorphic ventricular tachycardia initiated by a ventricular premature contraction falling on the terminal part of the T wave of the preceding sinus beat (A, black arrows). The postelectroversion tracing showed sinus rhythm at a rate of 55 beats/min, with alternating small, narrow (B, red arrows) and broad deep T-wave inversions (B, black arrows).



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Learning points

- ▶ Abnormal depolarisation of the myocyte is the causative mechanism of T-wave alternans (TWA).
- ▶ All the conditions producing long QT syndrome, such as medications and electrolyte disorders like hypokalaemia and hypomagnesaemia, pose a risk of sudden cardiac death.
- ▶ Vulnerability to ventricular fibrillation, susceptibility to malignant arrhythmias and the risk for sudden cardiac death can be assessed by observing TWA when present.
- ▶ It must be borne in mind that TWA has been found to be one of the strongest predictors of sudden cardiac death.

magnesiuric effect of acute alcohol consumption in excess. Alcoholics are prone to hypomagnesaemia, which can predispose them to torsade, as was the case in our patient.

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