Sotalol-induced torsade de pointes

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Summary
The authors report a case of torsade de pointes atypical ventricular tachycardia in a patient with chronic renal failure and hypertension treated with sotalol hydrochloride which they believe induced the arrhythmia.

Introduction
Differences in the pharmacological properties of the various β-blocking agents affect both their therapeutic action and their adverse effects (Petrie et al., 1976). Manifestations of an overdose of β-blocking drugs include bradycardia, hypotension, low cardiac output failure and cardiogenic shock (Khan and Muscat-Baron, 1977). Most β-blockers are classified as class I and II according to their antiarrhythmic properties. Contrary to other β-blocking drugs, sotalol has been found, experimentally, to possess class III antiarrhythmic action (Singh and Vaughan Williams, 1970).

A case is reported of sotalol-induced recurrent atypical ventricular tachycardia, 'torsade de pointes' (Krikler and Curry, 1976).

Case report
A 21-year-old man who was suffering from severe hypertension and chronic renal failure due to glomerulonephritis was admitted in December 1978 for control of his hypertension.

While on regular haemodialysis and taking propranolol 160 mg thrice/day, hydralazine 50 mg 4 times/day and clonidine 0.15 mg thrice/day his blood pressure remained uncontrolled. At that time he was advised to undergo bilateral nephrectomy but refused.

To provide fewer tablets, sotalol 160 mg 4 times/day was substituted for propranolol which brought his BP down to 180/110 mmHg. Three days later he developed episodes of Stokes-Adams attacks due to torsade de pointes (Fig. 1). Between the attacks, his ECG showed sinus bradycardia at a rate of 45/min with multifocal late ventricular beats, prolonged QTc interval 0.59 s and giant U waves (Fig. 2). Before the correct diagnosis was reached, i.e. lignocaine was given which increased the frequency of the episodes of tachycardia and Stokes-Adams attacks. Isoprenaline infusion 2–8 µg/min increased the tachycardia-free intervals but did not abolish them. He had to be defibrillated 10 times before a pacing wire was placed in the right ventricle. Finally, his tachycardia was brought under control with pacing, which became effective only at rates higher than 110/min. His serum sodium potassium, calcium and magnesium on daily estimations were all normal. Sotalol plasma concentrations could not be measured. On the third day after sotalol was withdrawn and with continuous pacing his ECG returned to normal.

Following this episode he underwent bilateral nephrectomy and his hypertension is now (1980) well controlled by maintenance haemodialysis alone.

Discussion
Although the precise mechanism for torsade de pointes is unclear, an underlying aetiological condition can very often be found. Postulated causes are bradycardias, high grade atrioventricular or sino-atrial block, electrolyte disturbances, myocardial ischaemia, congenital QT prolongation syndromes and myocarditis (Krikler and Curry, 1976). In recent years, various drugs have been blamed as an important cause of the torsade de pointes syndrome. Quinidine and anti-arrhythmic drugs with quinidine-like action, psychotropic and anti-anginal drugs have all been reported from various centres as causing atypical ventricular tachycardia (Krikler and Curry, 1976; Sclarovsky et al., 1979). In almost all the reported cases of torsade de pointes, a prolonged QT interval was found before the occurrence of the tachycardia.

Although β-blocking drugs are widely used in
**Case reports**

**FIG. 1.** Tracings showing bursts of atypical ventricular tachycardia, initiated by ventricular ectopics with long coupling interval and spontaneous termination.

**FIG. 2.** Electrocardiogram showing QTc prolongation (0.59 s) and giant U waves, before the Stokes–Adams episodes.

In clinical practice they have not yet been reported as a cause of torsade de pointes, despite having quinidine-like action. This is perhaps due to the fact that their membrane-stabilizing properties have little or no effect at therapeutic concentrations (Coltart, Gibson, and Shand, 1971). Sotalol hydrochloride, a non-selective β-blocking drug without intrinsic sympathomimetic and membrane stabilizing activity has been...
found experimentally to prolong the duration of the action potential and the QT interval (Singh and Vaughan Williams, 1970).

Although the authors were unable to measure sotalol plasma levels they believe that their patient represents a case of torsade de pointes induced by sotalol, owing to the high dosage scheme and renal failure. The prolongation of the QT interval, the late ventricular extrasystoles, the giant U waves and the configuration of the tachycardia itself further establish the identity of the tachycardia as torsade de pointes. Moreover, the aggravation of the episodes of tachycardia following the administration of i.v. lignocaine, the improvement by i.v. isoprenaline, and the abolition of the tachycardia by right ventricular pacing at high rates confirm this. The duration for 3 days of the episodes can be explained by the renal failure and the fact that sotalol is mainly excreted by the kidneys. Recently there has been a report of 2 cases of sotalol taken as an overdose with QT prolongation and tachyarrhythmias (Elonen et al., 1979). It is possible that they represent cases of torsade de pointes, but the published electrocardiograms are not typical.

It is concluded from this case alone that sotalol should be regarded, at least at high dosage, as a potential cause of the torsade de pointes type of ventricular tachycardia.

References


