CASE REPORT

Sotalol associated polymorphic ventricular tachycardia and coronary spasm

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Abstract

Sotalol may induce torsade de pointes through cardiac action potential prolongation, but a proarrhythmic effect secondary to its β blocking action has not been reported. A 54 year old man presented with symptoms of angina and presyncope, subsequently demonstrated to be associated with coronary spasm. Treatment with sotalol exacerbated his symptoms and resulted in recurrent polymorphic ventricular tachycardia with a pattern indistinguishable from that caused by a class III action. Following sotalol withdrawal polymorphic ventricular tachycardia resolved completely. Polymorphic ventricular tachycardia in patients treated with sotalol may therefore not always be the result of delayed repolarisation, but may be caused by β adrenoceptor blockade.

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Keywords: sotalol; coronary spasm; polymorphic ventricular tachycardia; torsade de pointes; arrhythmias

D,L sotalol is proposed to act as an antiarrhythmic as a function of both β adrenoceptor blocking action and a class III effect.^{1,2} QT interval prolongation and resultant torsade de

pointes caused by its class III action are well known,³ but a proarrhythmic effect has not been associated with the β blocking action of sotalol. We describe a patient treated with sotalol in whom β blocking activity is strongly implicated in the induction of polymorphic ventricular tachycardia with appearances indistinguishable from those seen in conjunction with action potential prolongation.

Case report

A 54 year old man was admitted to hospital following a presyncopal episode preceded by chest discomfort. Myocardial infarction was excluded by standard criteria and he was discharged on sotalol 40 mg twice daily, and aspirin. His chest pains increased and he was admitted for cardiac catheterisation, which revealed irregularities in the left coronary artery and a 90% proximal right coronary artery stenosis. Coronary angioplasty to the right coronary artery lesion was successful with minimal (10%) residual stenosis. He was discharged following the procedure and was better for three weeks; subsequently he developed severe symptoms with episodes of chest pain preceding palpitations, presyncope, and syncope.

Repeat cardiac catheterisation showed no significant restenosis; however, 24 hour ambulatory electrocardiography showed episodes of

Figure 1 Continuous trace taken from a typical ambulatory electrocardiogram recorded during symptoms. There is 2 mm ST segment elevation associated with extrasystoles in a bigeminal pattern, followed by self terminating polymorphic ventricular tachycardia.

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Figure 2 Effect of ergonovine administered into the right coronary artery (shown in left anterior oblique projection). (A) Before ergonovine there is a mild (20%) stenosis at the site of the previous angioplasty. (B) Following 5 µg ergonovine severe spasm is induced with almost total coronary artery occlusion.

ST segment elevation coincident with his chest discomfort, associated with extrasystoles and very frequent runs of polymorphic ventricular tachycardia (fig 1). Sotalol was stopped as the appearances were consistent with torsade de pointes, albeit in the absence of QT interval prolongation.³ Without sotalol the episodes of tachycardia ceased entirely, but even after 96 hours and allowing for an adequate elimination of the drug, the patient remained symptomatic with transient chest discomfort and palpitations. Serial 24 hour electrocardiograms at this time showed no evidence of polymorphic ventricular tachycardia but extrasystoles and couplets were again associated with episodes of ST segment shift.

An electrophysiological study five days after stopping sotalol demonstrated no inducible ventricular arrhythmia using up to three extrastimuli with intravenous isoprenaline at two right ventricular sites. Ergonovine (1–5 µg) was injected into the left coronary artery with no spasm. Dramatic results, however, were seen following the injection of ergonovine (1–5 µg) into the right coronary artery with focal spasm and a 95% constriction at the previous angioplasty site (fig 2), and reproduction of the chest discomfort typical of the patient's previous spontaneous episodes. No arrhythmias were observed in the short time before reversal of the spasm was obtained with intracoronary glyceryl trinitrate. Following the demonstration of coronary spasm the patient was started on diltiazem 120 mg three times daily and his symptoms fully resolved, with no further episodes of ST segment shift or

arrhythmias on 24 hour ambulatory electrocardiography. He was fully active and well at 12 months' follow up.

Discussion

D,L sotalol is used increasingly as an anti-arrhythmic drug in view of its demonstrated efficacy compared with other agents.⁴ Because of its class III action, a major disadvantage is the potential for proarrhythmia and polymorphic ventricular tachycardia, seen in up to 3.5% of patients.^{3,4} The occurrence and features of torsade de pointes in this context are well described and associated with dose dependent QT interval prolongation, not usually appearing with doses less than 80 mg twice daily.⁵ Polymorphic ventricular tachycardia in our patient was unlikely to be caused by a class III action as the QT interval was normal at all times and the dose of sotalol never exceeded 40 mg three times a day. The presenting symptoms of chest pain and presyncope were not characterised in detail and sotalol was started empirically. Following this, his symptoms worsened considerably, consistent with a proarrhythmic effect, and despite some temporary relief following successful angioplasty to the right coronary artery lesion his chest discomfort and syncopal episodes continued. When sotalol was stopped, however, symptoms attributable to arrhythmia decreased and the Holter appearances improved significantly with no further episodes of polymorphic ventricular tachycardia. The observations of ergonovine induced coronary spasm and complete resolution of symptoms with diltiazem are all strongly

suggestive that coronary spasm was the underlying cause of the tachyarrhythmia.

In patients with variant angina β adrenoceptor antagonists may increase coronary artery spasm,^{6,7} and induce ventricular arrhythmias,⁷ an effect abolished by calcium antagonists.⁶ As coronary vasorelaxation is mediated by both β_1 and β_2 adrenoceptor stimulation,⁸ non-selective β blockers can increase coronary vascular resistance further in patients with variant angina prolonging the duration of ischaemic attacks.^{6,7} Sotalol has not been reported previously to precipitate vasospasm, but as it is a non-selective β blocker devoid of intrinsic sympathomimetic activity⁵ it has those features that would be predicted to exacerbate coronary spasm in susceptible patients.

Ventricular arrhythmias precipitated by coronary spasm tend to be rapid and polymorphic,^{9,10} are usually non-inducible during programmed stimulation, but may be provoked following intravenous or intracoronary ergonovine.⁹ The precise mechanisms underlying such arrhythmias are unclear, but in animal models the production of an injury current and re-excitation of cells close to the ischaemic border zone, or re-entrant mechanisms, have been associated with their initiation.¹¹ In some patients, reperfusion rather than acute ischaemia is associated with the onset of arrhythmias.^{9,11} What determines whether arrhythmias occur during ischaemia or on reperfusion is unclear, and in our patient evidence consistent with both patterns was seen.

In conclusion, neither the exacerbation of coronary spasm nor the proarrhythmic effects of sotalol through its β blocking action have been reported previously. It has been established that β blockers, while usually decreasing

arrhythmia risk, may provoke ventricular arrhythmias in susceptible patients through induction of coronary spasm.⁷ This diagnostic possibility should be considered especially in those with polymorphic ventricular tachycardia treated with these agents. In addition it must be considered that polymorphic ventricular tachycardia in patients treated with D,L sotalol may not always be the result of class III activity.

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