

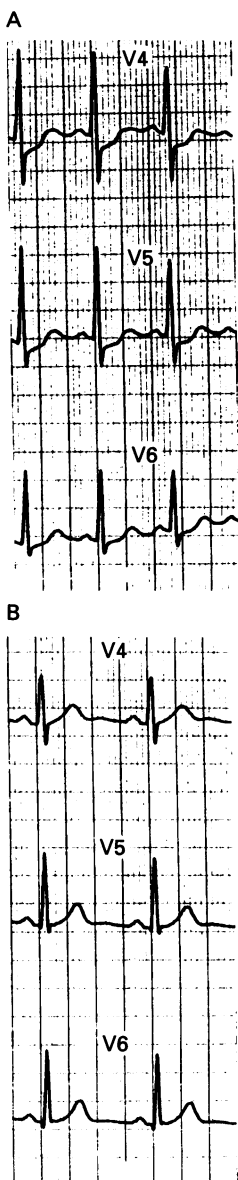
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SHORT CASES IN CARDIOLOGY

Unsuspected coronary artery disease revealed by administration of nebulised salbutamol

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Electrocardiogram (A) during administration of salbutamol 2.5 mg by nebuliser and (B) an hour later.

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A 29 year old man presented with an acute attack of asthma. His regular medications included inhaled ipratropium bromide and salbutamol and he had been taking oral theophylline for three days before presentation. The peak expiratory flow rate was 160 l/min rising to 350 l/min after administration of 5 mg of nebulised salbutamol. Posteroanterior chest radiography, arterial blood gas analysis, and white cell count were unremarkable. There were no identifiable risk factors for coronary artery disease.

He was given a further three doses of 2.5 mg of nebulised salbutamol over the next 7 h and when the last dose was given he complained of mild chest discomfort. An electrocardiogram performed during the period of discomfort showed sinus tachycardia with upsloping ST segment depression in the septolateral leads. The pain ceased promptly when he stopped using the nebuliser, and the electrocardiogram reverted to normal (fig). Similar pains and electrocardiographic changes occurred after repeat nebuliser administration of salbutamol 12 h later. He reverted to aerosol delivery of salbutamol and did not experience further pain.

At the time of the first episode of pain serum creatine kinase was 303 IU/l (normal 30-250 IU/l) lactate dehydrogenase 342 IU/l (normal 100-200 IU/l), and the aspartate transaminase concentration was normal. Repeated estimations over the next 2 days were normal.

A week later a routine electrocardiogram remained normal and transthoracic echocardiography was normal. Treadmill exercise electrocardiography using the standard Bruce protocol showed 2 mm planar ST segment depression in leads V4-6, with a walking time of 12 minutes limited by fatigue. The maximal heart rate was 160 beats/min and no chest pain was reported. The same response was seen 1 h later after the administration of 2.5 mg salbutamol by nebuliser.

Ten days after admission, coronary angiography showed that the left anterior descending artery was occluded at its origin, filling retrogradely from the right coronary artery. Both the left circumflex and right coronary arteries were normal. Left ventricular end

diastolic pressure was 16 mm Hg, and the ventriculogram seemed normal with no significant impairment of anterior wall motion.

Comment

Inhaled β_2 agonists increase systolic blood pressure, reduce diastolic blood pressure, and are positive inotropes

The electrocardiogram may show reduced T wave amplitude and prolongation of the corrected QT interval (QTc).^{1,2} Positive chronotropic effects have been reported to hasten the onset of exercise-induced ischaemia in those with known occlusive coronary artery disease, an effect possibly heightened by steal from subendocardial to the subepicardial vessels.³ A 60 year old hypertensive smoker had a myocardial infarction while using a salbutamol nebuliser,⁴ and severe ischaemia was seen in three other patients with previously diagnosed ischaemic heart disease.

Our case is unusual because the patient's age, lack of risk factors, normal resting electrocardiogram and lack of prior symptoms meant that without the chance use of nebulised salbutamol coronary disease might never have been suspected. Furthermore, abnormal electrocardiographic changes occurred readily with inhaled salbutamol but only late and at much higher heart rates during exercise, suggesting a role for subendocardial steal.³ Patients in whom unusual electrocardiographic changes are seen during treatment with β_2 agonists should be closely examined even if the diagnosis of ischaemic heart disease seems unlikely.

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