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Severe myocardial ischaemia induced by intravenous adrenaline

The dose of adrenaline usually recommended for the treatment of anaphylactic shock is 0·3 mg administered subcutaneously: an additional 0·1 mg may be given intravenously, but intravenous doses in excess of 0·1 mg are normally reserved for cardiac emergencies.[1] We describe a case of severe myocardial ischaemia caused by 0·3 mg adrenaline given intravenously.

Case report

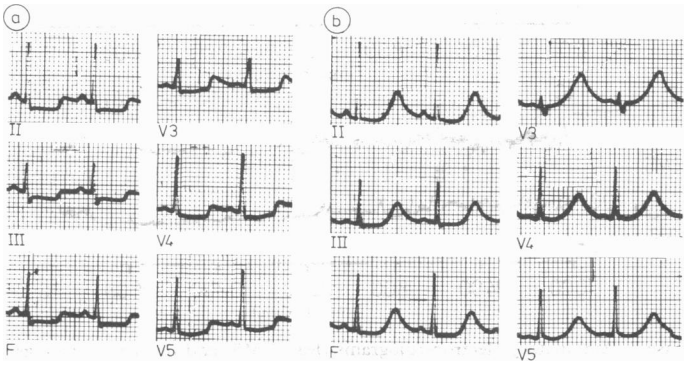
A 23 year old Asiatic woman presented to her doctor with acute pharyngitis. After an intramuscular injection of 600 000 units procaine penicillin she became pale and felt dizzy. Her pulse rate was 120 beats/min and blood pressure 110/70 mm Hg. Suspecting an anaphylactic reaction, the doctor administered betamethasone 4 mg intravenously, promethazine 25 mg intramuscularly, and adrenaline 0·3 mg intravenously. Immediately thereafter she developed severe retrosternal chest pain associated with sweating and dizziness. Sublingual nitroglycerine afforded no relief.

She presented to the emergency unit some five hours later, still suffering from chest pain; pulse rate was 105 beats/min and blood pressure 140/80 mm Hg. Jugular venous pressure was not raised, and her chest was clear. The apical impulse was normal in position and contour, and normal heart sounds with a grade 3/6 late systolic murmur maximal at the fourth left intercostal space were heard at auscultation. Electrocardiography (figure a) showed sinus tachycardia, a normal PR interval and axis, and extensive 1·5 mm planar ST-segment depression while the QT interval corrected for rate was prolonged (0·62 s): this reaction has been reported after administration of adrenaline.[2] Total plasma calcium and protein concentrations measured at the same time were within the normal range.

She was admitted to the coronary care unit, and the chest pain continued to be severe despite treatment with sublingual nitroglycerine (1·5 mg) and oral nifedipine (60 mg). The pain was relieved by an intravenous infusion of nitroglycerine (50 μg/min), with a simultaneous decrease in the degree of ST-segment depression. Subsequent electrocardiograms showed progressive normalisation of the ST segments and the development of tall, peaked T waves in the chest leads (figure b).

Her condition remained stable after her pain had been relieved, and the nitroglycerine infusion was subsequently stopped. Serial estimations of the creatinine phosphokinase MB fraction were all within the normal range.

Results of a submaximal effort stress test incorporating a multigated equilibrium blood pool scan were normal. M mode and two dimensional echocardiography disclosed normal left ventricular wall motion and size with evidence of mild prolapse of the mitral valve. No risk factors for ischaemic heart disease could be found. She remained asymptomatic with a normal electrocardiogram six months later.



Electrocardiogram at time of admission (a) and after administration of intravenous nitroglycerine (b).

Comment

Typical ischaemic chest pain with appropriate electrocardiographic changes in a young woman with no risk factors for ischaemic heart disease after intravenous injection of adrenaline indicates that the adrenaline was almost certainly the cause of the subsequent myocardial ischaemia. This supposition was further substantiated by the results of subsequent investigations, which largely excluded the presence of preexisting ischaemic heart disease. The mechanism of myocardial ischaemia lasting for more than five hours after a single bolus of adrenaline is unclear.

The ability of catecholamines to cause myocardial damage is well established, and these hormones have also been implicated in the myocardial necrosis occurring in patients with phaeochromocytoma.[3] The mechanism is thought to be either a direct effect on the myocardial cell or myocardial damage resulting from ischaemia caused by, among other things, constriction of the coronary artery. An additional mechanism whereby adrenaline may cause myocardial ischaemia is localised coronary artery spasm, as suggested by evidence showing that adrenaline added to strips of large coronary arteries increases the tension through a mechanism susceptible to alpha-antagonistic agents,[4] which reverse coronary artery spasm in some patients.[5]

This report emphasises the potential hazards of injudicious use of adrenaline and suggests that intravenous administration of adrenaline should be confined to cardiac emergencies.

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Late development of incisional hernia: an unrecognised problem

A long term follow up study was carried out to determine the incidence of incisional hernia after major abdominal operations; we report the results.

Patients, methods, and results

A total of 831 patients undergoing major abdominal operations at four centres in south Wales in 1972-3 were entered into a long term follow up study. Patients were examined by a single observer at one, three, and five years for the development of incisional hernia. The details and composition of the original cohort have been described elsewhere.[1] Altogether 564 surviving patients were willing to enter the study at one year. Loss of patients to follow up after one year was negligible and due mainly to death and to a few patients moving out of Wales. Information was obtained from the case notes to compare the prevalence of possible aetiological factors in patients in whom hernias developed early and those in whom they developed late. The table shows the total number of new hernias.

Full details of nine possible aetiological factors were available for 38 of the patients (21 with a hernia at one year, 14 with a hernia at three years, and three with a hernia at five years). Postoperative wound infection was the only