Letters to the Editor

Ischaemic heart disease in a young man

Sir,

Coronary atherosclerosis is a cause of sudden cardiac death with a surprisingly high frequency in those aged 35 or less.¹ In the Chinese, the prevalence of ischaemic heart disease is low when compared with Caucasians and the occurrence of the disease in young people is unusual.²

Case report

A 29-year-old Chinese man, a nonsmoker and a nondrinker, was rushed to hospital because of sudden onset of retrosternal chest pain, shortness of breath and sweating while having dinner at home. The patient had an unremarkable medical history except for febrile convulsion in his childhood, and had not been on any long-term medications. He was the only son in the family, with no known history of cardiac disease. After admission, the patient lapsed into generalised tonic-clonic convulsion with 10 episodes within five minutes and developed cardiorespiratory arrest requiring cardiopulmonary resuscitation. Electrocardiogram showed ventricular tachycardia refractory to intravenous lignocaine, bretylium and procainamide treatment as well as defibrillation. He died three hours after admission.

At autopsy, the heart weighed 383 g and all the three main branches of the coronary arteries were markedly atherosclerotic with the left anterior descending, left circumflex and right coronary 95%, 75% and 50% occluded by atheroma, respectively. A white thrombus was present in the lumen of the left anterior descending artery 3 cm distal to its origin from the aorta. There was no gross or microscopic evidence of acute myocardial infarction. The cause of death was ischaemic heart disease. Hypercholesterolaemia was diagnosed based on the blood sample taken at autopsy (cholesterol level: 7.1 mmol/l; HDL-cholesterol: 0.55 mmol/l; apolipoprotein AI: 0.94 g/l; apolipoprotein B: 1.88 g/l; triglyceride 4.98 mmol/l).

Comment

Blood cholesterol levels have been shown to predict not only myocardial infarction but also the extent of coronary artery disease assessed by coronary arteriography.³ Retrospectively, our patient was found to have hypercholesterolaemia with a low HDL level. If intravenous fluids are not administered before death, *post-mortem* levels of cholesterol levels are fairly representative of levels during life.⁴ The use of *post-mortem* cholesterol levels as surrogates for *ante-mortem* levels in this case confirmed the relationship between risk factors and the coronary heart disease.

The present case had 'silent' coronary atherosclerotic disease, with no preceding history of angina pectoris or myocardial infarction, which manifest itself as sudden death during sedentary activity. He is probably the youngest man ever reported to die suddenly from 'silent' ischaemic heart disease in the English language literature. Thus, any attempt to reduce cardiac mortality due to hypercholesterolaemia should begin early in life.

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Etomidate-induced hypoglycaemia

Sir,

A 17-year-old woman was admitted with dizziness and palpitations due to atrial tachycardia with 2:1 block. In the past she had had a right ventricular disarticulation for right ventricular dysplasia to control ventricular tachycardia originating from the right ventricle. Subsequently, she suffered from chronic right ventricular failure and had recurrent atrial tachyarrhythmias requiring frequent DC cardioversion. She was taking warfarin, digoxin, amiodarone and frumil.

Her full blood count, serum biochemistry, glucose, and International normalised ratio (INR) levels were normal on admission. She continued to be symptomatic from the tachycardia, her blood pressure dropped, and she became cold and clammy. Synchronised DC cardioversion was performed under general anaesthesia, which was induced by midazolam (2+1+1 mg) and etomidate (6+2+2+2+2 mg).

After three synchronised shocks she reverted to sinus rhythm. Subsequently she was found to have shallow infrequent respiratory effort with reduced level of consciousness.

There was no improvement with intravenous naloxone and flumazenil. She had a grand mal seizure which responded to intravenous diazepam 2.5 mg. As blood glucose was unrecordable on BM testing (Accutest), further haematological and biochemical investigations were performed. Blood glucose was <0.5 mmol/l, sodium 143 mmol/l, potassium 6.5 mmol/l, bicarbonate 12 mmol/l, creatinine 169 μ mol/l, and urea 8.6 mmol/l (sample slightly haemolysed); when repeated 1 hour later, potassium was normal. Haemoglobin was 13.9 g/dl, white blood cell count 12.7×10^9 /l, and platelets 68×10^9 /l. Coagulation profile showed a prothrombin time of 77 s, INR >10 , fibrogen 1.41 g/l, D-dimer 2 μ g/ml. Hypoglycaemia responded to 50 ml of 50% dextrose followed by 5% dextrose infusions and has not recurred.

A random cortisol level at the time of the fall was 259 nmol/l, inappropriately low for someone so acutely ill. Blood cultures were normal. She was put on intravenous dexamethasone. A short synacthen test showed an unsatisfactory response to adrenocorticotropin. Two weeks later, a long synacthen test showed no evidence of abnormal suppression. Since then, steroid replacement has been withdrawn with no further metabolic problems.

The precipitating cause of her hypoglycaemia was adrenal suppression. Septicaemia was excluded as a cause by the normal blood cultures. Addison's disease was excluded by normal long synacthen test.

We conclude that the bolus doses of etomidate suppressed the adrenal glands, leading to profuse hypoglycaemia. It is possible that her poor cardiac reserve contributed to the hypoglycaemia but not the adrenal suppression. Long term use of etomidate has been associated with adrenal suppression.¹ To our knowledge this is the only reported case of bolus doses of etomidate leading to hypoglycaemia. We recommend frequent use of BM sticks after etomidate if patients do not recover from anaesthesia rapidly.

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