"Empirical" thrombolysis in catastrophic pulmonary embolism

EDITOR.—We report a case of pulseless electrical activity thought to be caused by catastrophic pulmonary embolism. The early and "empirical" use of thrombolysis, accompanied by prolonged resuscitative efforts, appears to have been lifesaving. We seek to draw a distinction between "catastrophic" pulmonary embolism, which causes pulseless electrical activity and "massive" pulmonary embolism, which is a term used in the literature to describe cases of pulmonary embolism associated with hypotension.

A 69 year old woman attended the accident and emergency department having collapsed in her general practitioner’s surgery. She was extremely anxious, tachypnoeic (33 bpm), blood pressure 70/30 mm Hg, and heart rate 130 bpm. Electrocardiography (ECG) showed a classical right ventricular strain pattern. Anteroposterior radiography of the chest showed no sign of cardiac failure or pneumothorax. Arterial oxygen saturation was 96% on high flow oxygen.

Although the history was not typical and the ECG changes non-diagnostic, a diagnosis was made of massive pulmonary embolism with hypotension. She was given an intravenous bolus of 5000 units of unfractionated heparin. Arterial blood gas showed an oxygen tension of 15 kPa and a carbon dioxide tension of 4.1 kPa on 95% fractional inspiratory oxygen. The alveolar-arterial oxygen gradient was in keeping with pulmonary embolism.

Shortly after the bolus of heparin, cardiopulmonary arrest occurred. Resuscitation, following standard life support protocols, was attempted. In total she received 7 mg of adrenaline and 2 x 200J DC shocks for pulseless ventricular tachycardia with a bolus of 10 mg of recombinant tissue plasminogen activator (rt-PA) followed by an infusion of 90 mg over two hours. In intensive care, a dobutamine infusion of 5 mg/kg/min was started. The rt-PA infusion was stopped after 90 mg when her mouth was found to be filled with blood. Intra-venous heparin was continued. A full recovery ensued.

Subsequent investigations including echocardiography showed only trivial mitral regurgitation. Cardiac ischaemia as a low creatine kinase (muscle and brain) fraction of 2.8%, which is not diagnostic of acute myocardial infarction. Total creatine kinase was raised markedly in keeping with prolonged resuscitative efforts.

The accepted best treatment for "massive" pulmonary embolisms is angiography followed by surgical embolectomy. Several studies have demonstrated that thrombolysis can be followed by restoratation of normal pulmonary circulation.3 There is little written about the management of "catastrophic" pulmonary embolism and we believe that this is the only case reported in which a patient with a pulseless rhythm suspected to have sustained a massive pulmonary embolism on clinical grounds alone, was successfully resuscitated employing rapid bolus thrombolysis. In our opinion, this patient was saved by initial mechanical disruption and peripheralisation of the clot by resuscitation followed by rapid clot lysis with rt-PA.

There may be a role for the use of bolus thrombolysis in cases of pulseless electrical activity due to suspected pulmonary embolism, and would welcome correspondence from other physicians who have used these agents in similar circumstances.

T O N Y K E H O E
Senior House Officer

D I L I P D A C R U Z
Accident and Emergency Consultant, Torbay Hospital, Lansdowne Bridge, Torquay, Devon TQ2 7AA (e-mail: tony chếo@virgin.net)


Spontaneous carotid artery dissection

EDITOR.—I read with interest the case report of Mirza et al on spontaneous carotid artery dissection.1 We wish to highlight a similar case, who presented to our accident and emergency (A&E) department recently.

A 36 year old man presented with a five day history of discomfort in the left side of his neck associated with a gradual onset of a left temporal headache and diminished left temporal vision. The headache was increasing in severity and he had also developed diplopia and was confused. Clinical examination was normal. Past medical history revealed occasional migraine of four to five attacks accompanied by tunnel vision and he had also developed a brief period of head injury in Christmas of 1996 when he was punched and bunted with a brief loss of consciousness but with no obvious neurological deficit.

Computed tomography and electronencephalography were normal. The headache and neck stiffness continued and he then had a two minute episode of numbness in his right arm. Magnetic resonance imaging (MRI) suggested a left internal carotid artery dissection and he was thus started on heparin and warfarin.

He made a complete recovery and remained well until he represented nine months later after his warfarin was stopped with an acha behind his left eye associated with neck stiffness but there was no visual impairment or hemichore disorder. Clinical examination (MRI) suggested a left intracranial carotid artery dissection and was thus started on heparin and warfarin.

This report further supports the view expressed in the article of Mirza et al that a diagnosis of carotid artery dissection should...