measurement of CO-Hb, levels may have fallen substantially. Measurement of CO either in the exhaled breath or in the ambient air at the scene can be helpful. The cornerstone of treatment is high-flow oxygen. This reduces the half-life of CO-Hb to 4–6 hours. With hyperbaric oxygen this comes down to 15–30 minutes.³ Whilst hyperbaric oxygen confers theoretical advantage, the absolute indications for its use remain in question. In England, Northern Ireland and Wales, the British Hyperbaric Association runs a 24-hour telephone helpline (01752 209 999) to discuss cases and advise on referral. CO poisoning is avoidable, and household detectors are widely available at modest cost.

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A not-so-simple collapse

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In unexplained collapse, the possibility of carbon monoxide poisoning must not be neglected.

CASE HISTORY

A man of 40 was admitted having collapsed. His only medical history was treatment for depression. Examination was unremarkable except for complete short-term memory loss to such an extent that he was unable to recall his address. Blood tests and electrocardiography were normal. CT and MRI (Figures 1 and 2) revealed symmetrical lesions in the globus pallidus and posterior putamen. There were

also ill-defined lesions in the posterior periventricular white matter. The following day the patient's neighbour reported finding a hosepipe attached to his car's exhaust pipe.

The radiological findings are characteristic of carbon monoxide poisoning. The patient was referred for hyperbaric oxygen therapy. The results of psychometric testing improved but he was left with defects that limited his everyday function.

Figure 1 CT of brain

Figure 2 MRI of brain

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Box 1 Differential diagnosis of the CT and MRI appearances in carbon monoxide poisoning

Vascular

Lacunar infarcts
Late-stage hypertensive haemorrhage
Hypoxic injury

Toxic

Carbon monoxide poisoning
Methanol poisoning
Cyanide poisoning
Manganese poisoning

Metabolic

Mitochondrial cytopathies
Wilson's disease
Hallervorden-Spatz disease

Neurodegenerative

Acquired hepatocerebral degeneration

Physiological

Giant perivascular spaces
Age-related iron deposition

Infective

Acute disseminated encephalomyelitis
Acute bilateral striatal necrosis

Congenital

Neurocutaneous syndrome: neurofibromatosis type 1.

COMMENT

Carbon monoxide poisoning is responsible for about 50 deaths and up to 200 severe injuries a year in England.¹ Most cases are accidental. Signs and symptoms of poisoning depend upon exposure, and range from those of a non-specific viral illness to arrhythmias, coma and death.² The classic cherry-red lips, cyanosis and retinal haemorrhages rarely occur. Diagnosis is by measurement of carboxyhaemoglobin, although there is poor correlation between severity of symptoms and concentration of carboxyhaemoglobin. Patients may present for imaging studies with unexplained loss of consciousness or neurological abnormalities where carbon monoxide is not initially suspected. The differential diagnosis of the CT and MRI appearances is summarized in Box 1.

Treatment requires oxygen to shorten the half-life of carboxyhaemoglobin. A recent double-blind randomized trial demonstrated a reduced incidence of cognitive sequelae at six weeks in patients treated with hyperbaric oxygen,³ though the optimum number of sessions and the maximum time to therapy were not established.

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Superficial femoral vein aneurysm with massive pulmonary embolism

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Aneurysms of the venous system are rare. Possible complications include thrombosis and pulmonary embolism.^{1,2}

CASE HISTORY

A man of 41 consulted his general practitioner having been increasingly short of breath over the past four weeks. Previously he had been fit and healthy, a competitive oarsman. His rowing performance had declined over the same period. On referral to the medical team he was normotensive with an oxygen saturation of 97%. He became dyspnoeic on walking more than 10 m. Arterial blood gases were normal with the exception of a PaO₂ of 9.5 kPa on air. Forced vital capacity and forced expiratory volume exceeded predicted values. An electrocardiogram showed sinus bradycardia with right bundle branch block and right axis deviation. On echocardiography there was moderate dilatation of the right ventricle, good left and right ventricular function but some mitral regurgitation. Spiral CT revealed extensive pulmonary emboli at lobar and segmental levels (Figure 1). On duplex scanning the deep venous system of the lower limbs was normal but the left superficial femoral vein was affected by an aneurysm measuring about 10 × 4 × 4 cm. An area of high density was thought to represent a mobile thrombus less than 1 cm in size within the aneurysm (Figure 2). The patient was then anticoagulated with unfractionated heparin. On screening for thrombophilia, protein C, protein S and antithrombin 3 were all within the normal range. The extrinsic and intrinsic pathways were normal.

In view of his young age, his previous excellent health and the evidence of right ventricular dysfunction, peripheral thrombolysis was then attempted with streptokinase. The thrombus within the aneurysmal vein was not felt to be a contraindication because of its small size. The planned duration of thrombolysis was 72 h, but it had to be stopped after 48 h when a haematoma developed at the site of a

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