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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Figure 1 Spiral CT of thorax. Arrows show emboli in right and left pulmonary arteries

Figure 2 Duplex scan showing thrombus in aneurysmal superficial femoral vein (arrow)

previous radial artery puncture. Over the next two days the dyspnoea improved and on duplex scanning the thrombus within the superficial femoral vein aneurysm had disappeared. On further echocardiography, the right ventricle was no longer enlarged and there was no valvular incompetence.

Seventeen days after admission, while he remained fully anticoagulated with intravenous heparin, the left groin was explored under general anaesthesia. A large lipomatous lesion was found to be completely enveloping the proximal superficial femoral vein. The profunda artery was mobilized away from the lesion and the superficial femoral vein was ligated distally, then divided at its confluence with the

profunda vein. Postoperatively the patient was prescribed below-knee TED stockings and advised to wear these for three months; he was also anticoagulated with warfarin for the same proposed time. Histological examination of the operative specimen confirmed an aneurysm of the superficial femoral vein. The lipomatous lesion was normal fat. At one-month follow-up, the patient had resumed competitive rowing.

COMMENT

Thromboembolism in athletes is rare. When it does occur it is usually due to recent lower limb fracture or lower limb surgery under general anaesthesia, prolonged immobility in cramped conditions or a prothrombotic disorder. When these have been excluded, anatomical anomalies should be sought.

Venous aneurysms have been reported in the deep and superficial vein systems of both the lower and the upper limbs. It is not the same condition as venomegaly,³ a generalized dilatation of veins systemically; like arterial aneurysms, venous aneurysms are focal, usually due to a congenital weakness in the vein wall or associated with an arteriovenous fistula. Patients presenting with thromboembolism should be treated surgically since the incidence of recurrent pulmonary embolism exceeds 80%.⁴

Pulmonary embolism of the magnitude experienced by this patient would in many cases be fatal. The insidious presentation was probably attributable to the high cardiorespiratory reserve associated with his athletic lifestyle. The point is of clinical importance. Athletes tolerate more than usual blood loss or respiratory embarrassment before showing symptoms or signs.

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