when isoflurane is used at higher concentrations. Isoflurane, unlike enflurane and to a certain extent halothane, has not been found to cause EEG or clinical seizure activation⁴ or to produce adverse effects on cerebral energy stores⁵ and like barbiturates, it is an effective agent for reducing cerebral metabolic rate. Isoflurane, however, can increase cerebral blood flow and intracranial pressure possibly by interfering with cerebral autoregulation.

Volatile or intravenous general anaesthesia should be the last line of treatment for status epilepticus since prolonged anaesthesia is not without risks including hypotension, cardiac depression, hypothermia, venous thrombosis, pressure sores and increased susceptibility to infection.

Inhalational anaesthesia is costly and requires the availability of an anaesthesia machine and experienced medical and technical personnel to monitor the patient and equipment. The isoflurane vaporiser and carbon dioxide absorber need to be replenished periodically. In addition to continuous monitoring of vital signs and ECG, oximetry and capnography are essential in these patients and the ability to monitor end tidal isoflurane is also very useful. Muscle relaxants are not indicated and control of seizures can therefore be judged clinically as well as by intermittent or continuous EEG monitoring. With the proper facilities, isoflurane anaesthesia may be effective for intractable status epilepticus and is relatively safe, easily titratable and rapidly reversible after the seizures have been controlled.

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Intrinsic spinal cord haemorrhage due to streptokinase treatment for myocardial infarction

Haemorrhagic complications of streptokinase therapy for myocardial infarction (MI) occur in approximately 1% of patients.¹ The most commonly reported neurological complication is intracranial haemorrhage.¹ We report a case of spontaneous intrinsic spinal cord haemorrhage associated with streptokinase therapy for MI.

A 50 year old woman was admitted with a

three day history of discomfort in the neck. In the early hours on the day of admission the pain became much worse. An ECG and enzyme studies supported the clinical diagnosis of myocardial infarction, showing elevation of creatine kinase and lactate dehydrogenase, with S-T segment elevation in the anterior and lateral ECG leads. She was given opiate analgesia and streptokinase, 1 500 000 units intravenously.

Late in the afternoon she complained of altered sensation and weakness of the legs. This progressed rapidly, and she was transferred to the regional neurosurgical unit. She deteriorated rapidly, however, and by the time she arrived she was unable to move either leg and there was severe weakness of the arms. The weakness had started in the left arm, then the legs simultaneously, and the right arm. She had developed urinary retention during the afternoon and had been catheterised.

General medical examination was unremarkable. Neurological examination showed a flaccid quadriplegia, 0-3/5 in the arms, 0-5 in the legs. There was a sensory level to pinprick and light touch at C5, though there was some preservation of joint position sense in the arms. She was areflexic with upgoing plantars.

MRI of the cervical cord (figure) showed swelling of the cord from C3 to C7, with a central increase in signal on the T2 weighted sagittal sections. There was no evidence of cord compression, and no hypointensity which might represent a focal haematoma, although it was possible that the appearances might represent oedema around petechial haemorrhage. Nerve conduction studies were normal. Clotting studies on the day of admission showed prolongation of the prothrombin time at 17s, prolongation of the thrombin clotting time at 16s with reduction of fibrinogen below the haemostatic level at 0.7 G/l. She was given 1 unit of cryoprecipitate, and the next morning the thrombin time was only marginally elevated at 10s, and other haemostatic parameters were within normal limits. Subsequent clotting studies were also normal.

At 11 am the day after admission she deteriorated suddenly, developing a complete quadriplegia, complete bulbar palsy, and she required ventilation. Over the next few days she deteriorated further, developing a gaze palsy to the left and a right internuclear ophthalmoplegia, she developed complete heart block, hypotension and a chest infection. On the ninth day of her illness she arrested and died.

Necropsy examination showed haemorrhage centrally in the cord extending from the pons down to T10. For most of its length the haematoma was narrow, between 2-4 mm, but there was a 5 mm haematoma in the lower pons.

Neurological morbidity from thrombolytic therapy in MI occurs in approximately 0.5% of patients and has been largely limited to small intracerebral haematomata with low mortality.² To our knowledge, this is the first report of intrinsic spinal cord haemorrhage.

The administration of thrombolytic therapy to patients who subsequently appeared to have other disorders such as aortic dissection has been reported previously.¹ Guillain Barré syndrome (GBS) may present with chest or (more commonly) back pain, and the occurrence of pain in this circumstance is associated with a tendency for plasma creatine kinase level (CK) to rise.³ For this reason we felt it was necessary to carry out nerve

Figure T2 weighted sagittal image of the cervical and upper thoracic spinal cord, showing swelling of the cord between C3 and C7 (arrows) and increased signal in the central part of the cord.

conduction studies to rule out this disorder. Transverse myelitis may also present with chest pain and abnormal ECG, and this diagnosis was also considered, particularly as the MRI appearances, though atypical, would have been consistent with it. Given that neurological deterioration occurred between the time of the MRI and the necropsy findings, the reason for the atypical (for haematoma) MRI findings remain unclear.

Data regarding the duration of action of single dose streptokinase unaccompanied by heparin are scant,⁴ but do suggest that clotting may remain abnormal for more than 48 h after intravenous injection of 1.5 million units. At the time of her deterioration this patient's clotting remained marginally abnormal despite having been given cryoprecipitate, though well within the usual haemostatic limits. Nonetheless, the acute nature of the deterioration makes it highly likely that it was indeed due to extension of the haemorrhage.

Where symptoms of spinal cord dysfunction following the administration of streptokinase cryoprecipitate should be given without delay, even when the clinical picture is compatible with primary neurological disease.

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