Lesson of the Week

Development of neurological problems after lumbar puncture

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Introduction

Relative and absolute contraindications to lumbar puncture, such as suspected cerebral tumour, abscess, and subdural haematoma, are well known.¹ In the absence of clinical evidence of raised intracranial pressure correctly performed lumbar puncture is generally assumed to be safe, with "benign" low pressure headache the only likely complication.² Nevertheless, symptoms after lumbar puncture should never be dismissed as routine or trivial. Here we describe three patients who developed serious disorders after lumbar puncture, which were in two cases heralded by headache.

Case histories

Case 1—A 32 year old woman, admitted for investigation of chronic severe bilateral D7/8 dermatomal pain, had no other symptoms, and physical examination was normal. An ascending myelogram carried out through a lumbar puncture gave unremarkable results, with normal cerebrospinal fluid. Ten minutes after the procedure she suddenly developed severe occipital headache, neck stiffness, nausea, vomiting, and photophobia. Her symptoms improved over 24 hours, and she was discharged home. Three months later a second severe occipital headache, identical with the first, suddenly occurred. Lumbar puncture carried out in the local general hospital showed xanthochromia, and subsequent computed tomography confirmed subarachnoid haemorrhage. Four vessel angiography showed a single aneurysm at the junction of the right posterior communicating artery and the carotid siphon. This was clipped, and postoperative recovery was uneventful.

Case 2—A 32 year old man with a five week history of severe weakness of rapid onset after a diarrhoeal illness was transferred for further investigation. He had generalised muscle weakness, depressed tendon reflexes, and hyperaesthesia of the soles of both feet. Neurophysiological studies identified severe axonal peripheral neuropathy. He complained of persisting headache after lumbar puncture, which had been carried out seven days before transfer. Spinal fluid opening pressure had been 12 cm cerebrospinal fluid, and the results of laboratory analysis were normal. Guillain-Barré syndrome was diagnosed, and his headache was controlled by simple analgesics. He was discharged. Two months later he was readmitted still complaining of headache, which had continued since his discharge and latterly been accompanied by nausea, vomiting, and drowsiness. There was no history of head injury. Magnetic resonance imaging showed bilateral subdural haematomas (fig 1), causing compression and twisting of the lateral ventricles and early tentorial herniation with loss of the ambient cistern (fig

Symptoms developing after lumbar puncture, especially headache, require careful assessment as they may be the first indication of unsuspected potentially fatal lesions

2). The haematomas were evacuated, and postoperative recovery was uneventful.

Case 3—A 15 year old girl with pneumococcal meningitis was transferred for further investigation when her condition rapidly deteriorated after

FIG 1—Magnetic resonance image T₂ weighted (spin echo 2200/80) axial slice showing bilateral subdural haematomas.

lumbar puncture and she had a prolonged apnoeic episode. She was found to have flaccid quadriplegia and areflexia with fully preserved voluntary eye movements and nystagmus. Before lumbar puncture there had been no clinical evidence of raised intracranial pressure. Computed tomograms were normal with no evidence of an abscess but did not exclude tonsillar herniation. The "locked in syndrome" due to medullary infarction was diagnosed. Three months later, with no improvement in her neurological condition, she developed bronchopneumonia and died. Necropsy showed

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infarction of the rostral medulla and inferior aspects of the cerebellar hemispheres, consistent with basilar artery occlusion. No cause for this was identified other than tonsillar herniation consequent to lumbar puncture.

FIG 2—Magnetic resonance image T₁ weighted (spin echo 500/40) coronal slice showing compressive effects of bilateral haematomas.

Discussion

The precipitation of aneurysmal rupture by lumbar puncture, as in case 1, has been proposed in several case reports,^{3,4} and patients who have already had a subarachnoid haemorrhage from an intracranial aneurysm occasionally bleed again immediately after lumbar puncture.⁵ Probably removing cerebrospinal fluid at lumbar puncture leads to traction on the basal blood vessels and results in aneurysmal bleeding. Hence in a previously asymptomatic patient who develops severe headache after lumbar puncture subarachnoid haemorrhage should be looked for. Nevertheless, the characteristics of "benign" headache after lumbar puncture may be similar to those of subarachnoid haemorrhage: both may present with meningism, photophobia, nausea, and vomiting, with pain confined to the occipital and cervical areas,⁶ and with no localising neurological signs. The most important differentiating factor is that posture affects headache after lumbar puncture, the headache being aggravated by the upright position.⁷ In patients who present with suspected subarachnoid haemorrhage lumbar puncture is best avoided if computed tomography shows subarachnoid blood.⁵ Nevertheless, if the scan is normal, or not immediately available, lumbar puncture should be performed as the only method available to confirm the diagnosis, or to exclude bacterial meningitis.

In case 2 lumbar puncture was followed by protracted headache. Reports of intracranial subdural haematoma related to lumbar puncture⁸⁻¹¹ have emphasised that diagnostic confusion may arise as headache after lumbar puncture may last for several weeks¹² and in some cases months.¹³ Nevertheless, such persisting headache cannot be considered benign until subdural haematoma has been actively excluded. Possibly a sudden decrease in intracranial pressure from a loss of cerebrospinal fluid leads to a shift in the brain tissue and traction on cortical blood vessels, which may rupture. Rupture of presumably normal intracranial vessels causing subdural haematoma has been reported after the sudden lowering of intracranial pressure by intravenous infusion of urea for treating hypertension.¹⁴

Lumbar puncture is mandatory in cases of suspected bacterial meningitis.¹⁵ Tonsillar herniation is a recognised rare complication,^{16,17} though the benefits of lumbar puncture outweigh this risk, and case 3 emphasises that even without clinically raised intracranial pressure tonsillar herniation may ensue with fatal consequences. The absence of papilloedema does not exclude the diagnosis,¹⁸ the important features being progressive headache, a deteriorating level of consciousness, and the development of focal neurological signs.

Lumbar puncture is still a valuable procedure in a wide range of conditions,² and in the vast majority of cases is uncomplicated. Nevertheless, our three case histories show that all patients undergoing lumbar puncture should be closely observed and that all symptoms warrant careful assessment.

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If a patient receiving long term warfarin treatment has transient ischaemic attacks should 75 mg of aspirin daily be prescribed in addition to the warfarin?

The evidence from clinical trials does not support the use of oral anticoagulants to prevent strokes in patients with transient ischaemic attacks. If there is any reduction in the risk of stroke it is probably small and the benefit is outweighed by the increased risk of major bleeding.¹ Aspirin, in doses of 1 g or more daily, has been shown to be useful in preventing stroke in patients with transient ischaemic attacks.² Although smaller doses are likely to be equally effective, this has not been demonstrated by clinical trial.

Aspirin increases the risk of bleeding in patients taking warfarin in three ways.³ Large doses (more than 2 g daily) may have an additive hypoprothrombinaemic effect. Smaller doses (1 g daily) have been shown to produce a transient increase in prothrombin time in occasional patients. This disappears within 72-96 hours and has been attributed to displacement of warfarin from protein binding. It is probably dose related and is unlikely to be important with a daily dose of 75 mg of aspirin. Thirdly, aspirin prolongs the bleeding time. This is probably the most important cause of the increased risk of bleeding. Platelet function is impaired by 75 mg of aspirin so the risk of bleeding is probably also increased with this dose.—LINDA BEELEY, consultant clinical pharmacologist, Birmingham.

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