Letters

Meningoencephalopathy following iopamidol myelography.

Sir: Iohexol myelography has been reported to cause encephalopathy and probable inappropriate antidiuretic hormone secretion.¹ We have seen a similar complication with iopamidol following a cervical myelogram.

A 55 year old lady with a cervical cord lesion underwent cervical myelography via a lateral C1/C2 puncture. 10 ml of iopamidol, 200 mg/ml were introduced; the procedure was uncomplicated and the usual precautions were taken to prevent excessive intracranial spread of contrast. No lesion was identified and her subsequent clinical course suggests she has multiple sclerosis.

On her return to the ward she was encouraged to drink and she was observed to do so freely. Three hours later she became withdrawn and complained of nausea for which she was given prochlorperazine 12.5 mg intramuscularly. After a further 30 minutes she became very agitated and aggressive and was given 50 mg of chlorpromazine intramuscularly. Shortly after this she had a generalised convulsion. She was pyrexial at 38°C for over 8 hours, had marked neck stiffness and responded only to painful stimuli. A diagnosis of chemical meningoencephalitis was made and she was given dexamethasone 8 mg intramuscularly followed by 4 mg 6 hourly. Before the myelogram her electrolytes were normal. After the convulsion her plasma sodium was 115 mmol/l, potassium 3.7 mmol/l, glucose 7.1 mmol/l, urea 2.7 mmol/l, and the plasma osmolality was 241 mmol/kg. The urinary electrolytes were sodium 115 mmol/l, potassium 47 mmol/l, urea 112 mmol/l, and the osmolality was 439 mmol/kg.

Over the next 24 hours she gradually improved with water restriction and steroids. Three days later she had returned to normal and her plasma sodium had risen to 130 mmol/l.

We conclude that she developed a chemical meningoencephalitis due to iopamidol contrast medium with secondary inappropriate antidiuretic hormone secretion. The phenothiazine may have complicated the picture but was given after the onset of symptoms and clearly did not cause them. As a precautionary note it is worth bearing in mind this unusual complication in

any patient who becomes confused or agitated following myelography.

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Reference

- 1 Donaghy M, Fletcher NA, Schott GD. Encephalopathy after iohexol myelography. *Lancet* 1985;ii:887.

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Iatrogenic lumbar meningocele after excision of a neurofibroma

Sir: Lumbar neurofibromata may produce symptoms similar to those of protruded disc, owing to their location among the roots of the cauda equina. It is usually possible to remove such tumours totally. Permanent resolution of symptoms would therefore be expected. A case is reported where symptoms did recur, and were found to be due to the formation of a "pseudo meningocele".

A 46 year old lady first developed pain in the anterior aspect of both knees in 1981. A more sciatic distribution became apparent as it gradually worsened. A variety of treatments had little effect. By 1985 the pain was waking her at night and she had to walk about the house to obtain relief. During the day such activity would cause exacerbation, as would coughing and sneezing. There was minor constipation, urgency of micturition, a feeling of incomplete bladder emptying and numbness of the lower anterior thighs. She had a little wasting of the left thigh and calf with moderate weakness at hip and knee. There was minimal weakness at the ankle. Pin-prick sensation was impaired over the anterior thigh and shin. Reflexes were normal. Myelography revealed a total obstruction to contrast flow by a lobulated intrathecal mass whose lower border was at the L4/5 level.

At operation a full laminectomy was performed at L4 and L5 and the dura opened to reveal a discrete lobulated 2.5 cm × 1.5 cm tumour lying ventral to the nerve roots on the right side. The arachnoid was opened and the tumour rolled backwards to display a small vascular pedicle connecting to a root. This was divided, there being no other attachments, and the tumour was removed. The dura was closed completely with interrupted

non-absorbable sutures, and the wound closed in standard layered fashion without drainage. Histology showed the typical appearances of a neurilemmoma.

The patient made an uneventful recovery and was discharged home 12 days after surgery. Her symptoms had resolved completely. She remained well for several months, but then low central backache gradually developed to be followed by both buttock and thigh pain. The pain was not as sharp as before although in a similar distribution and with a feeling of numbness in the thigh. There was no sphincter disturbance.

Examination revealed a well healed lumbar wound with no masses or tenderness. She had good spinal movements and normal straight leg raising. There was an area of subjective impairment of pinprick sensation over the outer left calf, but no motor loss or reflex abnormality. Radiology demonstrated the extent of the previous bone removal: myelography showed a large abnormal sac lying posterior to, and communicating with, the lumbar theca at the level of the laminectomies. There was no evidence of tumour recurrence. CT scan confirmed this. At operation the sac, which was lying deep within the muscles, was found to have a thin fibrous wall with a smooth lining. It was 7 cm × 4 cm × 3 cm and was filled with CSF. There was a 5 mm diameter communication between the sac and the theca at the level of the L4/5 disc space to the right of the midline. The suture material of the previous dural closure could be seen and it did not appear that the communication involved the suture line. At the bony edges and over the posterior aspect of the theca the membrane was densely adherent. However a small flap of it could be mobilised and sutured across the defect. The rest of the sac was mobilised easily off the muscles and plicated across the dura. The muscles were partially mobilised to allow approximation and hence obliteration of the space occupied by the sac. The

Fig Axial CT scan of lower lumbar region following intrathecal metrizamide injection.