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 Three hours later she became withdrawn and complained of nausea for which she was given prochlorperazine 12.5 mg intramuscularly.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.

DJM REYNOLDS

C FREER

Addenbrooke's Hospital,
Hills Road,
Cambridge CB2 2QQ, UK

Reference

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

Accepted 5 November 1988

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.

wound healed well, her symptoms rapidly resolved, and she went home 2 weeks after her operation.

Iatrogenic meningocoeles have been reported following spinal surgery, usually for disc disease.¹⁻³ The majority are due to failure of recognition of a surgical injury, or failure of a repair attempt. It is possible that the puncture wound for myelography may be responsible for a few cases.⁴ They have been reported after intradural operations⁵ but at higher levels when the problem has been cord compression.

The recurrent symptoms may be caused by adhesion of a nerve root to the fistulous track or prolapse through it with resultant traction on the root. This did not apply in the case reported here. It is possible that the distension of the sac excited nerve endings in the muscles and periosteum of the laminae upon which it pressed, the radiation being due to common innervation. These meningocoeles are found when patients are investigated for recurrence of symptoms: I have been unable to find reports of any not thus discovered, although this does not mean they do not occur.

One factor known to be associated with failure of a dural repair is the presence of a clip upon the dural edge.⁵ None were used in this case. However temporary sutures were used to retract the dural edges. It is possible that the small hole left by one of these gradually enlarged and led to the defect found at operation, for it did not appear to be related to the suture line. Care must therefore be used in the employment of retraction sutures, and perhaps all dural repairs ought to be covered with gelfoam or a similar preparation to encourage fibrinous reaction. Such materials alone are not recommended for dural repair.¹

PETER J WARD,

Department of Neurological Surgery,
St Bartholomew's Hospital,
London EC1A 7BE, UK.

Current address for correspondence: Hurstwood Park Neurological Centre, Colwell Road, Haywards Heath, West Sussex RH17 7GJ, UK.

References

- 1 Rinaldi I, Hodges OT. Iatrogenic lumbar meningocoele: report of three cases. *J Neurol Neurosurg Psychiatry* 1970;33:484-92.
- 2 Schumacher HW, Wassman H, Podlinski C. Pseudomeningocoele of the lumbar spine. *Surg Neurol* 1988;29:77-8.
- 3 Swanson HS, Fincher EF. Extradural arachnoid cysts of traumatic origin. *J Neurosurg* 1947;4:530-8.

4 Kar AM, Pradhan S, Mittal P. Cerebrospinal fluid fistula after lumbar puncture. *Br Med J* 1987;295:528.

5 Hyndman OR, Gerber WF. Spinal extradural cysts, congenital and acquired: report of cases. *J Neurosurg* 1946;3:474-86.

Accepted 5 November 1988.

Recurrent localised myositis

Sir: Polymyositis is typically a diffuse disease throughout its course, but rarely may begin as a localised process. We describe a case with recurrent localised myositis with fluctuating elevation of serum creatine kinase (CK) levels, but who clinically merely had relapsing focal muscle lesions, without diffuse progression. All other cases of localised myositis previously published have progressed rapidly to the typical generalised pattern.

A 66 year old shepherd presented in August 1985 with a 5 week history of tender swellings in both calf muscles, and progressive disability in walking. Normally, he walked 20 miles a day on his farm, but at the time of presentation could only walk with the aid of a stick. He had no relevant past medical history. General examination revealed no evidence of systemic disease, in particular there were no heart murmurs, his heart size was normal and his peripheral pulses were normal, and he was normotensive. Abnormal physical findings were confined to the calf muscles. Both were tender, and very indurated with nodular swellings. Movement of the affected muscles was

limited by pain. There was no obvious loss of power, wasting or fasciculation, tendon reflexes were retained, and there was no sensory deficit.

Creatine kinase was elevated at 1190 (normal 40-150 IU/l). Sedimentation rate, haemoglobin and white cell count, radiographs of legs and chest, complement levels, toxoplasma serology and autoantibody screen were all normal. There was no eosinophilia. No antibodies to coxsackie virus were detected. Stools contained no cysts or ova. Sputum cytology was negative. Electromyograph (EMG) of gastrocnemius showed no spontaneous activity, and polyphasia with good recruitment and abundant small motor units. Needle muscle biopsy of uninvolved muscle (left quadriceps) was normal. Open muscle biopsy of left gastrocnemius revealed well marked changes of polymyositis. There were foci of active muscle necrosis, with collections of lymphocytes and an increase in interstitial connective tissue. There were also lymphocytic infiltrates around blood vessels. Direct immunofluorescence showed C₃ and IgM within blood vessels.

While being investigated, his clinical condition spontaneously improved. The induration receded, he was able to walk unaided and the CK level fell to 370 IU/l. In view of this improvement, it was decided to withhold steroid therapy.

Following discharge, with regular review he has been able to continue his work, walking 8-10 miles per day. His only complaint normally is of stiff calves in the morning. He has had two further clinical episodes of focal myopathy. In December 1985, he redeveloped tender induration in both calves, but again spontaneously settled

Table Change in CK value (normal 40-150 IU/l) during relapses and remissions of recurrent localised myositis

Months from onset	CK	Site of nodules	Number
1-125	1404	gastrocnemius/soleus	2
1-50	1190		2
1-75	370		1
4-50	1374		—
5-00	1521		2
5-50	1100	both hamstring & right calf	1
6-00	1106		—
7-50	1350		—
10-00	2281		—
12-00	953		—
13-00	4084		3
13-125	891		1
16-00	106		—
17-00	722		—
20-00	2454		—
23-00	6985		—
36-00	62		—