Ependymal cyst of the spinal cord presenting with acute paraplegia

Sir: Ependymal cysts of the spinal cord are rarely encountered and reported. The usual presentation is pain, associated with a slowly progressive neurological deficit, the distribution being dependent on the level of the cyst. By contrast, the case of a patient with acute sensorimotor paraplegia caused by an ependymal cyst of the spinal cord is presented.

On 8 August 1986, a 25 year old black male experienced the acute onset of severe, midline upper thoracic back pain while exercising with weights. Three days later he developed band-like chest pain in a T1–T3 distribution and bilateral lower extremity weakness. The following day he noted that he was dragging both feet when walking. The next day he awoke to find that he could not move either lower extremity and was numb below his nipples. His chest pain and upper thoracic spinal pain persisted. He was then brought by ambulance to the Harbor/UCLA Medical Center.

The patient had a flaccid paraplegia, patulous anal sphincter, an absent bulbocavernosus reflex, absent patellar reflexes, and normal Achilles reflexes bilaterally. The plantar response was absent bilaterally. Pin and touch sensation were absent below T4 on the right and below T6 on the left. Position and vibration sensation were absent in both lower extremities. The upper thoracic spine was markedly tender to palpation over the spinous processes of T1 and T2. No cutaneous abnormalities were noted.

Plain radiographs of the spine were normal. Positive contrast myelography with a water soluble agent showed a complete intradural, extramedullary block of the contrast column corresponding to the T1–T2 disc space on both cervical and lumbar injections of contrast. CT scan of the region of the block showed it to be caused by a right-sided intraspinal mass lesion anterolateral to the spinal cord.

The patient was taken immediately to the operating room where a C7–T2 laminectomy was performed. The exposure was extended to allow visualisation of the anterolateral aspect of the spinal cord by removing the right T1–T2 facet joint and the T2 pedicle with a high speed drill. Upon opening the dura, a tense, 4 cm, extramedullary cyst with an extremely thin, translucent wall was identified anterolateral to the spinal cord. The cyst contained clear, colourless fluid that appeared similar to cerebrospinal fluid. The cyst was easily dissected from the inner surface of the dura, but sharp dissection was required to free it

from the spinal cord. Microscopically, the cyst was lined by a single layer of non-ciliated cuboidal to columnar epithelium with regular ovoid nuclei and a moderate amount of eosinophilic cytoplasm. Immunoperoxidase method for cytokeratin was strongly positive, whereas those for S-100 and glial fibrillary acidic protein were negative. Mucicarmine stain for mucin was negative. The final pathologic diagnosis was neuroepithelial lined cyst, consistent with ependymal cyst.

On the first postoperative day the patient had recovered partial sensation below the nipples, and was able to move his toes bilaterally. By one week he had anti-gravity strength in the left lower extremity, and some return of bowel and bladder function. At 6 months postoperatively he was walking with braces, had a nearly full return of all sensory modalities, and control of urine and faeces.

An unusual feature of our case is the acute mode of presentation, although one previously reported case also presented with acute paraplegia.¹ We hypothesise that the cyst acutely increased in size to produce this syndrome. A mechanism of the increase may have been a one-way valve effect on the wall of the cyst wherein an acute increase in the subarachnoid pressure filled the cyst. Upon normalisation of pressure the fluid could not escape. Our patient was lifting weights just prior to the onset of his symptoms; a Valsalva manoeuvre during this exercise could have been the source of the increased subarachnoid pressure. Interestingly, in the other reported case of acute paraplegia from an ependymal cyst the symptoms began shortly after exercise.¹

The excellent postoperative return of neurological function from a preoperative paraplegia is also of note in this case. Although there have been both clinical and experimental demonstrations of functional recovery from a complete motor and sensory paraplegia caused by intraspinal space occupying lesions,² this unusual circumstance occurs more often with the removal of benign extramedullary, intradural masses. There has been one reported case of recovery from a complete sensorimotor paralysis caused by an ependymal cyst.³ Our case and the previously reported cases^{2,3} demonstrate that paraplegia caused by a favourably located and benign intraspinal mass lesion is not a hopeless situation, and requires prompt operation.

PHILLIP ASHLEY WACKYM
TONY FEUERMAN
GEORGE F GADE
TERRY J DUBROW

The Division of Neurosurgery,
Department of Surgery,
Harbor/UCLA Medical Center,
Torrance, California, USA

Address for correspondence: Phillip Ashley Wackym, M.D., A7-122 CHS, UCLA Hospital, Los Angeles, CA 90024, USA.

References

- 1 Dharkar SR, Kanhere S, Dharker RS. Intramedullary epithelial cyst of the spinal cord. *Surg Neurol* 1979;12:443–4.
- 2 Tarlov IM, Herz E. Spinal cord compression studies IV. Outlook with complete paralysis in man. *Arch Neurol Psych* 1954;72:43–59.
- 3 Hoffman GT. Cervical arachnoid cyst. Report of a 6 year old negro male with recovery from quadriplegia. *J Neurosurg* 1960;17:327–30.

Accepted 27 February 1988

Abdominal muscle paralysis from herpes zoster

Sir: Segmentally distributed cutaneous eruptions along with sensory changes and pain are frequently seen in herpes zoster. Motor loss is infrequent¹ and involvement of the thoracic musculature is rare.² We describe a case of thoracic motor zoster presenting as an abdominal swelling 3 weeks after a typical cutaneous zoster.

A 60 year old diabetic female was referred because of swelling on the left side of her abdomen. This swelling had started about one week previously, 3 weeks after the start of a cutaneous vesicular eruption involving the area of the T8–T12 dermatomes on the left. Investigation for intra-abdominal tumour had begun. Examination revealed healing eruptions in the distribution of the left T8–T12 dermatomes (fig 1) and an obvious asymmetry of the abdomen, with the left side bulging remarkably (fig 2). An attempt to inspire and maintain inspiration would move the umbilicus to the right side. Touch and pin prick were reduced in the left T8–T12 dermatomes. EMG one week later revealed obvious denervation on the left side in the T9 through L1 paraspinal segments. Examination of the contralateral side at the T11 level did not reveal abnormality. Physical examination of the patient 9 months later revealed that the swelling had disappeared and the weakness had disappeared.

Fig 1 Left lateral view showing healing eruptions from cutaneous zoster in the T8-T12 distribution.

The thoracic segments are often affected by cutaneous zoster, but this area is relatively free of motor involvement. However, weakness of intercostal and abdominal muscles may pass unnoticed and, therefore, motor deficits at these levels may be more

Fig 2 Frontal view showing prominent left-sided abdominal bulging. A scar from a previous cholecystectomy is present on the right side.

frequent than reports in the literature would suggest.² This case illustrates a severe degree of weakness of the abdominal musculature, such that an abdominal tumour was suspected.

Diabetic thoraco-abdominal neuropathy has been reported to present with a localised abdominal swelling³ but we do not believe that the diabetes was the cause because the paraspinal EMG findings were not generalised as would be expected in this condition and the excellent recovery is less in keeping with a diabetic radiculopathy.

The clinical course of this case with good resolution of the motor paralysis is consistent with other reported cases of motor zoster where the majority of patients recover² and only 15% have significant residual deficit.

The authors thank Mary Dampier, MD for referring the patient.

RUSSELL H GLANTZ
RUZICA K RISTANOVIC
Rush-Presbyterian-St Luke's Medical Center,
1653 West Congress Parkway,
Chicago, Illinois 60612-3864, USA

References

- 1 Ragazzino MW, Melton LJ III, Kurland LT, et al. Population-based study of herpes zoster and its sequelae. *Medicine (Baltimore)* 1982;61:310-61.
- 2 Thomas JE, Howard SM Jr. Segmental zoster paralysis—a disease profile. *Neurology* 1972;22:459-66.
- 3 Boulton AMJ, Angus E, Ayyar DR, et al. Diabetic thoracic polyradiculopathy presenting as abdominal swelling. *Br Med J* 1984;289:798-9.

Accepted 10 February 1988

Matters arising

St Paul and temporal lobe epilepsy

Sir: There have been many efforts to explain the dramatic spiritual conversion of the Apostle Paul (Saul of Tarsus). It has been postulated that his conversion experience was a manifestation of temporal lobe epilepsy.¹ However, we would argue that this diagnosis can only be speculative. It requires the simultaneous occurrence of rare phenomena, selective reading of the biblical texts ignoring several important statements, and still it constructs a scenario marred by inconsistencies.

Paul's conversion (Acts 9:3-9, Acts 22:4-11, and Acts 26:9-18) took place while he was journeying to Damascus to arrest Christians. On the road he was blinded by a bright light, he fell to the ground, and he heard a voice saying "Saul, why do you persecute me?" He replied "who are you, Lord?" He was answered "I am Jesus whom you persecute," and instructed to go to

Damascus, where he regained his vision after three days of blindness.

Dr Landsborough postulates that Paul's experience can be ascribed to an "attack of (temporal lobe epilepsy), perhaps ending in a convulsion, which was startling and dramatic. The blindness which followed may have been post-ictal."¹ This explanation requires the coincidence of an intense emotional aura recalled as being pleasurable, with a prolonged post-ictal cortical blindness. Both of these phenomena, as pointed out by Dr Landsborough's own review, are rare as manifestations of epilepsy.

An analysis of this event can only be made from the descriptions preserved in the biblical text; Dr Landsborough's review carefully examines portions of the text but ignores some other key points. A fact omitted is that the people travelling with Paul are also described as hearing the voice (Acts 9:7) or seeing the light (Acts 22:9). In addition, all of the people travelling with him are said to have fallen at the event (Acts 26:14). These descriptions are inconsistent with an epi-

leptic explanation for Paul's experience.

There are other problems with this explanation for Paul's conversion. When cortical blindness has been reported as a post-ictal phenomenon it has followed generalised motor ("grand-mal") seizures, with gradual return of vision over hours to days.² In five described cases of ictal blindness accompanying occipital status epilepticus, the two patients who had bilateral blindness persisting interictally both manifested depressed mental states. In addition, the patients described showed the lack of awareness of their deficits which is characteristic of cortical blindness.³

In contrast, no mention of a convulsion is made in any of the descriptions of Paul's conversion, nor elsewhere in reference to his life; furthermore, Paul was honoured as a spiritual leader in a culture which may have interpreted a seizure as a sign of demonic influence. There is no indication of any confusion or stupor during the events of Paul's conversion. Paul's distinct memory of his experiences on the Damascus road, including