

melanoma by MacKay,<sup>7</sup> even without melanosome, these microtubules suggest the diagnosis of achromatic metastases of a melanoma.

The development of such metastasis could be explained in several ways: the blood arterial tract, the retrovenous route from the cavernous sinus through the superior petrosal sinus,<sup>5</sup> a nerve brain route from the oropharynx to the CPA; such a route could also explain the leptomeningeal carcinomatosis, which is responsible for the dissemination in the CNS via the CSF.

Clinically, the metastases are very different from the other tumours of CPA. Brackmann<sup>1</sup> nevertheless emphasises that the hearing loss is invariable, rapid, associated with headache, and involvement of the last cranial nerves suggests a malignant neoplasm.

The CT scan, however, often suggests an acoustic neuroma. A petrous erosion<sup>14b</sup> or a vascular blush may confirm the diagnosis.<sup>8</sup> In the absence of clinical evidence the nature of the lesion is often discovered at surgery or necropsy.

O DELERUE

A DESTEE

Department of Neurology A, Centre Hospitalier  
Universitaire, H<sub>U</sub>, Lille.

P DEVOS

Department of Neurology, CHG, Boulogne, France

Correspondence to: Professor Destee.

- 1 Brackmann DE, Bartels LJ. Rare tumors of the cerebello pontine angle. *J Oto laryngol* 1982;34:89-103.
- 2 Cornil L, Paillas JE, Vague J. Localisation pontocérébelleuse métastatique d'un épithélioma du cavum. *Rev Neurol (Paris)* 1934;67:106-11.
- 3 Fischer A, Marres E, Thijssen H. A tumor in the cerebellopontine angle region. An unusual case. *Clin Neurol Neurosurg* 1977;80:189-94.
- 4 Nakada T, St John JN, Knight JN. Solitary metastasis of systemic malignant lymphoma to the cerebellopontine angle. *Neuroradiology* 1983;24:225-8.
- 5 Cohen DL, Diengdoh JV, Thomas GT, Hims-worth RL. An intracranial metastasis from a PRL secreting pituitary tumor. *Clinical Endocrinology* 1983;18:259-64.
- 6 Jahrsdoefer RA, Sweet DE, Fitz-Hugh S. Malignant fibrous xanthoma with metastasis to cerebellopontine angle. *Arch Otolaryng* 1976;102:117-20.
- 7 MacKay B, Avala AG. Intracranial tubules in human melanoma cells. *Ultrastruct Pathol* 1980;1(1):1-6.
- 8 Kendall B, Symon L. Investigation of patients presenting with cerebellopontine angle syndromes. *Neuroradiology* 1977;13:65-84.

### Myasthenia gravis aggravated by pyrantel pamoate

Pyrantel pamoate is an antihelmintic agent used worldwide.<sup>1</sup> We report worsening of myasthenia gravis by pyrantel pamoate in one patient.

In October 1989 a 72 year old diabetic man noticed mild intermittent palpebral ptosis when watching television. He was blind in the right eye from diabetic retinopathy. In mid November of the same year, he complained of diarrhoea. A stool specimen was positive for *Ascaris lumbricoides* and a single 1000 mg dose of pyrantel pamoate was taken orally with breakfast on 26 November. Several hours later he became fatigued when chewing and also when walking. The following week he could not chew meat and bread and he noticed dyspnoea. Neurological examination on 4

December showed bilateral palpebral ptosis with fatigability, limitation of abduction of both eyes and limitation of adduction of the right globe, hypophonia, weakness of neck extension and weakness of abduction of both arms.

Administration of 2 mg of edrophonium chloride reversed the ophthalmoparesis. Electrophysiological study revealed a normal area of compound muscle action potential in the right abductor digiti quinti muscle both at rest and after 15 seconds of maximal voluntary effort. Supramaximal repetitive stimulation of the right ulnar nerve at 3 Hz and 30 Hz were within normal limits.

Stimulation single fibre electromyography<sup>2</sup> of the right extensor digitorum comunis muscle showed a mean jitter of 40  $\mu$ s (upper normal limit 25  $\mu$ s<sup>2</sup>): 50% of insertions showing increased jitter and no blockings were found. Haematocopycal and biochemical tests were normal. Antinuclear antibodies were positive at a 1/340 titre with an homogeneous pattern. AntiDNA antibodies were negative. Antismooth muscle and antimitochondrial antibodies were positive at a 1/80 titre. CT of the thorax was normal. Antiacetylcholine receptor antibodies were not determined.

He was treated with 180 mg day of pyridostigmine bromide without alleviation of his symptoms. There was no improvement in spite of increasing the daily dose of pyridostigmine and muscarinic symptoms appeared. Prednisone 60 mg day was started. Two weeks later he could tolerate solid food. Dyspnoea, weakness of neck extension and weakness of the extremities disappeared, but ophthalmoparesis persisted. From February 1990 he is on alternate day prednisone, 70 mg every other day, without neuromuscular symptoms apart from ptosis and bilateral ophthalmoparesis.

We feel that, in this patient, ingestion of pyrantel pamoate aggravated a previously existing myasthenia gravis. Pyrantel exerts its antihelmintic action by blocking the worm's neuromuscular transmission, producing a depolarising-type neuromuscular block.<sup>3</sup> In rabbits, parenteral administration leads to paralysis and death,<sup>3</sup> but toxic neuromuscular effects in humans have not been reported to date to our knowledge.

ENRIQUE BESCANSÁ

Section of Neurology

MANUEL NICOLÁS

Service of Internal Medicine

CRISTINA AGUADO

Family Medicine Unit

MANUEL A TOLEDANO

Service of Clinical Neurophysiology

MONTSERRAT VIÑALS

Section of Neurology, Regional Hospital Reina  
Sofía, Córdoba, Spain

Correspondence to: Dr Bescansa, Section of Neurology, Regional Hospital Reina Sofia, Avda Menendez Pidal s/n, 14004 Córdoba, Spain.

- 1 Katz M. Anthelmintics. Current concepts in the treatment of helminthic infections. *Drugs* 1986;32:358-71.
- 2 Trontelj J, Mihelin M, Stalberg E. Axonal stimulation for end-plate jitter studies. *J Neurol Neurosurg Psychiatry* 1986;49:677-85.
- 3 Rollo IM. Drugs used in the Chemotherapy of helminthiasis. In: Goodman Gilman A, Goodman LS, Gilman A, eds. *The pharmacological basis of therapeutics*, 6th ed. New York: Mac-Millan, 1980:1013-37.

### Myxopapillary ependymomas arising from nerve roots of the spinal cord

Conventional pathogenesis suggests that myxopapillary ependymomas arise from

ependymal cells lying adjacent to fibrous tissue. The large majority of myxopapillary ependymomas arise from the filum terminale where this arrangement applies. I report two cases of myxopapillary ependymomas arising from nerve roots of the cauda equina. This unusual and so far unreported origin, may be due to myxopapillary ependymomas arising directly from ependymal cells lying against the nerve roots.

Ependymomas form 2-6% of all gliomas.<sup>5</sup> Myxopapillary ependymomas belong to a distinctive subgroup of ependymoma. They are virtually restricted to the cauda equina and are thought to originate from the filum terminale and conus medullaris. Myxopapillary ependymomas form (16%) 9/53 to (21%) 8/27 of intraspinal tumours (six and two respectively). Their restrictive origin and fibrous histology has led to the belief that the pathogenesis of myxopapillary ependymoma is related to ependymal cells lying against fibrous tissue mainly the filum terminale.<sup>9,12</sup> Myxopapillary ependymomas also arise, rarely, in subcutaneous sacrococcygeal tissues,<sup>3,4</sup> the lateral ventricle,<sup>11,12</sup> and the cervico-thoracic cord.<sup>12</sup> The pathogenesis for myxopapillary ependymomas arising in such sites is less clear.

The first case was a 48 year old male with a one month history of low back ache and left leg sciatica with dragging of his left foot. There was weakness of the left ankle dorsiflexion, eversion of the left ankle and weakness of the knee on the same side. Myelogram showed complete block at spinal level lumbar 2. The second case was a 44 year old male who had experienced low back pain for three years. There were no neurological signs. Myelogram revealed a block at spinal level thoracic 12.

Macroscopically these were large encapsulated tumours. The myxopapillary ependymoma in case 1 arose from the first sacral root and in case 2 it arose from the second lumbar root. Treatment for both cases was excision of tumour and resection of the nerve root. Microscopically these were discrete highly vascular tumours. There was a compact arrangement of ependymal cells and fibrous tissue around cores of hyaline acellular connective tissue rich in blood vessels.

The presentation of these two tumours is similar in many ways to myxopapillary tumours arising from the filum terminale or the conus medullaris, namely low back pain with myelographic block.<sup>12</sup> The orthodox pathogenesis for myxopapillary ependymoma is from ependymal cells lying against fibrous tissue for example the filum terminale. Other proposed theories include myxopapillary ependymoma arising from ependymal rests in extra dural locations such as the sacrococcygeal region.<sup>10</sup> It has been observed<sup>7</sup> that ependymal rests occur in normal children, and occur where myxopapillary ependymomas arise in the sacrococcygeal region, namely the dermis-subcutaneous junction. It may be that myxopapillary ependymomas arise in abnormal sites as a result of heterotopia, but heterotopia of tissue of the central nervous system in sites other than nasal is open to doubt.<sup>13</sup>

Others<sup>11</sup> have suggested that the presence of ependymal cells against fibrous tissue is not essential for the formation of the myxoid change seen in myxopapillary ependymomas. They suggest that this myxoid change is the result of anoxia due to vascular changes. Anoxia, however, is unlikely to be the primary cause of myxopapillary ependymomas or the changes seen within them,

otherwise they would occur more frequently. Furthermore, it has been shown<sup>8</sup> in organ cultures of myxopapillary ependymomas originating in the filum terminale, that progressive perivascular sclerosis and hyalinisation is a feature of cultures over twenty days old.

The possibility of myxopapillary ependymomas arising from ependymal cells alone and not requiring contact with fibrous tissue becomes more feasible with the report of myxopapillary ependymomas arising from extra dural sacrococcygeal regions, the cervico-thoracic cord, the lateral ventricle and the nerve roots, as in this report.

I am grateful to Mr DG Hardy, Consultant Neurosurgeon, Addenbrookes Hospital and the Neuropathology and Neuroradiology departments of Addenbrookes Hospital.

ANANT SHARMA  
Addenbrooke's Hospital, Cambridge

Correspondence to: Dr Sharma, Royal Eye Unit, Kingston Hospital, Wolverson Avenue, Kingston upon Thames KT2 7QB, UK.

- Bale PM. Ependymal rests and subcutaneous sacrococcygeal ependymoma. *Pathology* 1980;12:237-43.
- Barone BM, Elvidge AR. Ependymomas. A clinical survey. *J Neurosurg* 1970;33:428-38.
- Helwig EB, Stern JB. Subcutaneous sacrococcygeal myxopapillary ependymoma. *Am J Clin Pathol* 1984;81:156-61.
- Matsuo K, Kumagai K, Kawai K, Tsuchiyama H. Subcutaneous sacrococcygeal myxopapillary ependymoma. A case report and review of the literature. *Acta Pathol Jpn* 1985;35(4):925-31.
- McKeran RO, Thomas DGT. The clinical study of gliomas. In: Thomas DGT, Graham DI eds. *Brain Tumours. Scientific basis, clinical investigation and current therapy*. London: Butterworths, 1980.
- Mork SJ, Loken AC. Ependymoma. A follow up study of 101 cases. *Cancer* 1977;40:907.
- Pulitzer DR, Martin PC, Collins PC, Ralph DR. Subcutaneous sacrococcygeal ('Myxopapillary') ependymal rests. *Am J Surg Pathol* 1988;12(9):672-7.
- Rawlinson DG, Rubinstein LJ, Herman MM. In vitro characteristics of a myxopapillary ependymoma of the filum terminale maintained in tissue and organ culture systems. *Acta Neuropath* 1974;27:185-200.
- Russell DS, Rubinstein LT. *Tumours of central neuroepithelial origin*. London: Butterworth, 1989.
- Rutherford GS, Adam AE, O'Rourke SO. Subcutaneous myxopapillary ependymoma. *Histopathology* 1987;11:218-20.
- Sato H, Ohmura K, Mizushima M, Ito J, Kuyama H. Myxopapillary ependymoma of the lateral ventricle. A study on the mechanism of its stromal change. *Acta Pathol Jpn* 1983;33(5):1017-25.
- Sonneland PRL, Scheithauer BW, Onofrio BM. A clinicopathologic and immunocytochemical study of 77 cases. *Cancer* 1985;56:883-93.
- Willis RA. *The borderland of embryology and pathology*. London: Butterworth, 1958.

### Spinal extradural venous haemorrhage controlled by a drawing pin: a new technique in neurosurgery

Massive bleeding from venous plexuses can be a life threatening complication of surgery. This is well recognised from the cranial dural sinuses and is also common during rectal and prostatic surgery.

We describe a case of venous haemorrhage during the removal of a cervical meningioma which was controlled by an unusual technique.

A 64 year old man presented with a one year history of progressive paraesthesiae and numbness affecting both hands associated with clumsiness of fine hand movements and

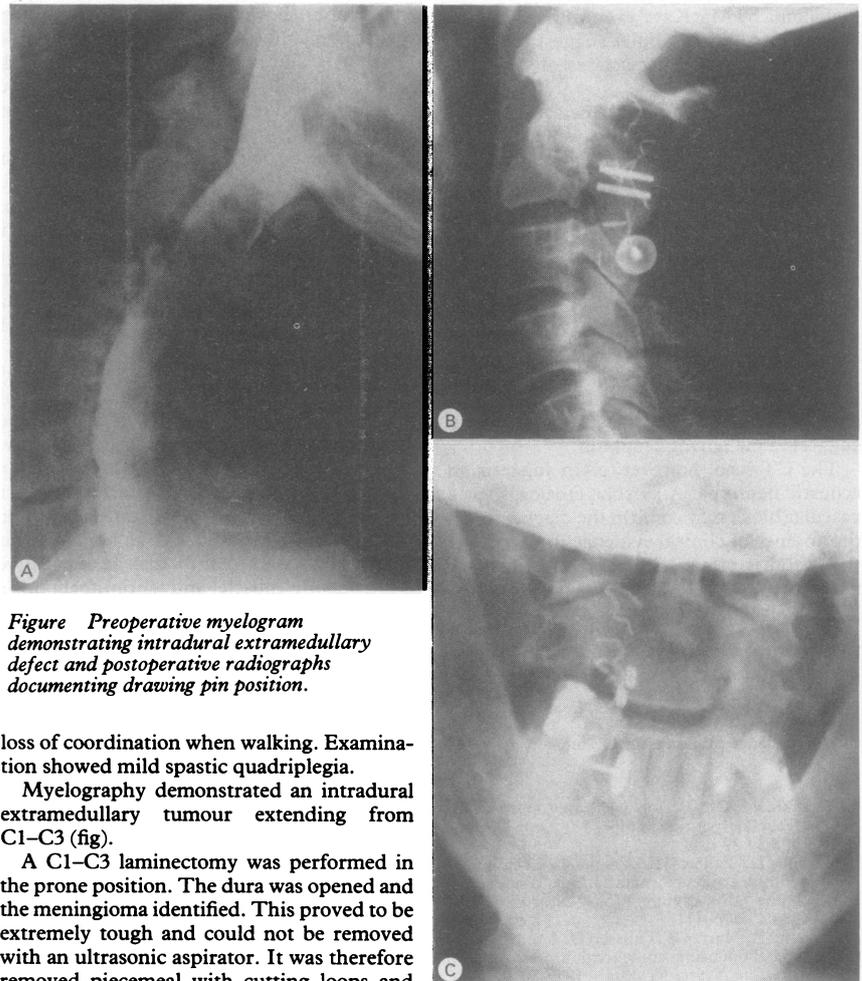


Figure Preoperative myelogram demonstrating intradural extramedullary defect and postoperative radiographs documenting drawing pin position.

loss of coordination when walking. Examination showed mild spastic quadriplegia.

Myelography demonstrated an intradural extramedullary tumour extending from C1-C3 (fig).

A C1-C3 laminectomy was performed in the prone position. The dura was opened and the meningioma identified. This proved to be extremely tough and could not be removed with an ultrasonic aspirator. It was therefore removed piecemeal with cutting loops and rongeurs.

There was considerable haemorrhage from arterialised veins in the tumour bed and from the extradural venous plexus. The tumour had an en-plaque origin and haemorrhage was controlled by diathermy, packing, local pressure and suction. These manoeuvres allowed 75% of the tumour to be removed resulting in decompression of the cervical cord.

At this stage it became increasingly difficult to stop the haemorrhage from the extradural venous plexus despite using all conventional haemostatic methods. The bleeding was staunch by continuous pressure exerted on patties and bone wax but the close proximity of the cervical cord prevented permanent maintenance of pressure despite the use of suture buttresses. The patient had received a 30 unit transfusion becoming hypotensive for only a brief period.

The remaining haemorrhage was immediately and completely controlled by a drawing pin passed through the dura transfixing an extradural pattie to the wall of the vertebral canal (fig). This provided permanent tamponade of the extradural venous plexus.

The wound was closed and the patient made an uneventful post operative recovery with significant improvement in his neurological condition and no wound infection.

Excluding the cranial dural sinuses there are three sites in the body with thin walled venous plexuses which are prone to bleed during surgery: the pre-sacral plexus, the prostatic plexus, and the spinal extradural venous plexus.

Once bleeding has started attempts at haemostasis often seem to provoke more haemorrhage elsewhere.

The use of drawing pins to tamponade venous plexuses to control bleeding is not a new idea. The method was first used in rural China to control life threatening haemorrhage during rectal operations.<sup>1</sup> Khan *et al*<sup>2</sup> and Nivatvongs *et al*<sup>3</sup> discuss four such cases. They describe the use of specially constructed titanium pins and add the caveats of possible reaction to the metal used or superadded infection.

In our case an autoclaved stationer's brass drawing pin was used to tamponade the extradural plexus. The risk of death or morbidity from continuing haemorrhage was felt to outweigh any possible complications either from the metal constituents of the pin or from subsequent infection. Prophylactic antibiotics were used.

We describe this technique in the hope that other neurosurgeons may find it useful for the temporary or permanent control of haemorrhage or in situations where temporary dural fixation is impractical using standard methods.

KM MORRIS  
GFG FINDLAY  
Mersey Regional Department of Medical  
and Surgical Neurology,  
Walton Hospital,  
Liverpool, UK

Correspondence to: Mr Morris.

- Qinyao W, Weijin S, Youren Z, Wenqing Z, Zhengrui H. New concepts in severe presacral haemorrhage during proctectomy. *Arch Surg* 1988;120:1013-20.
- Khan FA, Fang DT, Nivatvongs S. Management of presacral bleeding during rectal resection. *Surg Gynaecol Obstet* 1987;165:275-6.
- Nivatvongs S, Fang DT. The use of thumbtacks to stop massive presacral haemorrhage. *Dis Colon Rectum* 1986;29:589-90.