LETTERS TO THE EDITOR

Ipsilateral hemi-Parkinsonism secondary to an astrocytoma

Parkinsonism has rarely been described in association with intracranial tumours. In a previous review by Polyzoidis *et al* in 1985, 49 verified cases with intracranial tumours were collected from the literature,⁸ the majority of which were meningiomas.^{8,10,12} Further review of the current literature adds another four cases.^{3,5,7,12} Only six of these 55 cases directly involved the thalamus and/or the basal ganglia.⁸ All six were infiltrating intra-axial gliomas.^{8,12} When symptoms of Parkinsonism occur in a patient with intracranial tumour, it is not uncommon to make an incorrect diagnosis of Parkinson's disease.¹¹

We report an additional case of an intra-axial glioma located in the left thalamic area with ipsilateral hemi-Parkinsonian features, but without any clinical signs of increased intracranial pressure.

A 62 year old man presented with a one year history of increasing tremor of the head and left limbs, impairment of memory, slowness in his actions and unusual fatigue. His complaints failed to respond to anti-Parkinsonian drug therapy. There was no other previous medical history of note.

At the time of admission on 28 August 1989, physical and neurological examination revealed facial impassivity, a marked resting tremor involving the left limbs and head in combination with mild cog-wheel rigidity. There was no sign of pyramidal involvement and no sensory deficits. Plantar responses were flexor. There was no other evidence of the intracranial involvement except for a right homonymous hemianopia. There was no papilloedema. His attention span was brief. He had great difficulty buttoning clothes and his walking was slow with a tendency to fall frequently.

A cranial enhanced CT scan showed an extensive area of mixed attenuation in the left thalamic region with some mass effect (fig)

Figure CT scan with contrast showing a tumour mass with surrounding oedema causing destruction in the left thalamic region.

which turned out to be an astrocytoma Grade II after the biopsy on 20 September 1989 was taken. He was then transferred to receive a course of radiation therapy. At the time of transfer the clinical picture was unchanged.

Structural lesions of the basal ganglia are well recognised and sometimes also produce hemidystonic syndromes.⁴ Most of these cases have been due to haemorrhages, infarction or tumours. Tumours may produce Parkinsonism either by pressure on the basal ganglia^{3,5,7,8,10,12} or more rarely by direct infiltration^{8,9} as in our case. Extra-axial masses impinging on the basal ganglia appeared to be more common than infiltrating lesions.⁸ Several authors have shown the reversibility of clinical findings after resection of the tumour which caused compression and displacement of the basal ganglia structures rather than infiltration or destruction.^{8,12} Direct destruction of the basal ganglia may explain the poor clinical response to anti-Parkinsonian drugs in our patient and also in other patients described in the literature. Garcia de Yébenes *J*, *et al* reported a significant decrease of caudate postsynaptic dopamine receptors which accounted for their patient's Parkinsonism secondary to a craniopharyngioma.²

Intracranial tumour can mimic many neurological conditions including Parkinsonism. When Parkinsonian signs have been present in the intracranial tumour patient, resting tremor and rigidity were reported to be mostly contralateral^{1,8,9} in contrast with our patient who presented with an ipsilateral hemi-Parkinsonism. Oliver⁶ and Polyzoidis *et al*⁸ reported two other patients who presented with an ipsilateral resting tremor and rigidity due to a parieto-occipital and a frontoparietal glioma respectively. They both argued that the signs in these cases were not due to direct pressure on or invasion of the basal ganglia but were due to midbrain compression from upward or downward herniations displacing the midbrain against the tentorial edge. We prefer this explanation because in our case the Parkinsonism was ipsilateral to the lesion.

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- 1 Fahn S. Secondary Parkinsonism. In: Goldensohn ES, Appel SH, eds. *Scientific approaches to clinical neurology*. Philadelphia: Lea and Febiger, 1977:1159-89.
- 2 Garcia de Yébenes J, Gervas JJ, Iglesias J, Mena MA, Martín del Río R, Somoza E. Biomechanical findings in a case of Parkinsonism secondary to brain tumor. *Ann Neurol* 1982;11:313-6.
- 3 Kumar S. Parkinsonism as a feature of temporal lobe glioma. *J Indian Med Assoc* 1988;86(7):180-1.
- 4 Marsden CD, Obeso JA, Zarranz JJ, Lang AE. The anatomical basis of symptomatic hemidystonia. *Brain* 1985;108:463-83.
- 5 Navarro JA, Ezquerro JJR, Albuquerque TL, Gutierrez JC. Parkinsonism due to corpus callosum astrocytoma. *J Neurol Neurosurg Psychiatry* 1986;49(12):1457-8.
- 6 Oliver L. Parkinsonism due to midbrain compression. *Lancet* 1959;ii:817-9.
- 7 Pall HS. 74 year-old lady who developed bilateral parkinsonism secondary to an intrinsic cerebral tumour. *J Neurol Neurosurg Psychiatry* 1987;50:1386-7.
- 8 Polyzoidis KS, McQueen JD, Rajput AH, MacFadyen DJ. Parkinsonism as a manifestation of brain tumor. *Surg Neurol* 1985;23:59-63.
- 9 Sciarra D, Sprockin BE. Symptoms and signs referable to the basal ganglia in brain tumor. *Arch Neurol Psychiatry* 1953;69:450-61.
- 10 Siegfried J. *Die Parkinsonische Krankheit und ihre Behandlung*. Wien-New York: Springer-Verlag, 1968:262.
- 11 van Eck JHM. Parkinsonism as a misleading brain tumour syndrome. *Psychiat Neurol Neurochir* 1961;64:109-12.
- 12 Wakai S, Nakamura K, Niizaki K, *et al*. Meningioma of the anterior third ventricle presenting with Parkinsonism. *Surg Neurol* 1984;21:88-92.

Spontaneous intraneural haematoma of the optic nerve

Acute unilateral optic neuropathy is most commonly caused by retrobulbar neuritis. We describe a patient with visual loss caused by a spontaneous intraneural haematoma of the optic nerve. This has not previously been described.

A healthy 37 year old Greek housewife presented with a three week history of decreased visual acuity in the right eye. She awoke with poor vision in association with a generalised headache lasting 24 hours. A diagnosis of probable retrobulbar neuritis was made by the referring hospital and she was given oral prednisolone for five days. Her vision did not improve. Vision in the left eye was unaffected. There was no previous visual or neurological history of note.

On examination she could read J16 with the right eye and J1 with the left. There was a central scotoma with colour desaturation. Fundoscopy showed right optic atrophy. General examination was unremarkable and she was normotensive. A CT brain scan showed a minimally enhancing lesion in the region of the right optic nerve which could have been either a meningioma or an optic nerve glioma (fig). Angiography was normal. In view of the possibility of vision in the left eye becoming affected, surgery was recommended.

At craniotomy the right optic nerve was found to be haemorrhagic and measured 3 cm in diameter compared with the normal left nerve measuring 0.5 cm. Careful dissection revealed no evidence of a vascular abnormality. An incision was made in the swollen nerve and the haematoma evacuated. Again no evidence of abnormal vessels was identified. Histology showed blood clot, haemosiderin, fibrin, and disrupted optic

Figure CT brain scan with contrast showing a minimally enhancing lesion in the region of the right optic nerve.

nerve, but no evidence of tumour or abnormal vasculature. Post-operatively her visual acuity was unchanged.

Review of the literature reveals that this type of pathology is very unusual. Intrachiasmal haematomata have been recognised,¹ some of which are due to chiasmatic gliomas.^{1,2} Bleeding causing chiasmatic compression has also been reported secondary to cryptic vascular malformations identified at operation^{3,4} and in patients with clinical evidence of vascular anomalies elsewhere.⁵ Burnbaum *et al*⁶ described a patient with a blue-domed haemorrhagic cyst arising extrinsically to the optic nerve and compressing it at its junction with the chiasm. Holt recorded a similar case.⁷ The likely cause of these lesions is haemorrhage into a pre-existing cyst.

The aetiology of our patient's intraneural haematoma is not clear. Maitland *et al* proposed that most idiopathic intrachiasmatic haematomata are caused by small venous or cryptic vascular malformations, and this would seem to be the most likely explanation with our case. The finding of optic atrophy after only three weeks of symptoms would support this possibility.

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- 1 Maitland CG, Abiko S, Hoyt WF, Wilson CB, Okamura T. Chiasmal apoplexy: report of four cases. *J Neurosurg* 1982;56:118-22.
- 2 Schneider RC, Kriss FC, Falls HF. Prechiasmal infarctions associated with intrachiasmal and suprasellar tumours. *J Neurosurg* 1970;32:197-208.
- 3 Roski RA, Gardner JH, Spetzler RF. Intrachiasmatic arteriovenous malformation. Case report. *J Neurosurg* 1981;54:540-1.
- 4 Fermaglich J, Kattah J, Manz H. Venous angioma of the optic chiasm. *Ann Neurol* 1978;4:470-1.
- 5 Rishede J, Seedorff HH. Spontaneous haematoma of the optic chiasma. Report of a case. *Acta Ophthalmol* 1974;52:317-22.
- 6 Burnbaum MD, Harbison JW, Selhorst JB, Young HF. Blue-domed cyst with optic nerve compression. *J Neurol Neurosurg Psychiatry* 1978;41:987-91.
- 7 Holt H. Cysts of the intracranial portion of the optic nerve. *Am J Ophthalmol* 1966;61:1166-70.

Displaced Torkildsen's shunt: an unusual cause of cervical myelopathy

The Torkildsen's shunt procedure involves placing the proximal end of a rubber catheter through a burr hole into the occipital horn of the lateral ventricle, and the distal end through the cisterna magna into the posterolateral cervical gutter, where it is then sutured to the dura.¹¹ This procedure is nowadays seldom employed due to the high failure rate but complications are rare after the shunt has been functioning for a number of years.² The most frequent complications are infections and the development of a false meningocoele due to leakage of CSF through the cervical dura mater.²

A 75 year old male presented after six months of progressive gait unsteadiness and urinary incontinence. In 1964 he developed obstructive hydrocephalus due to a post-traumatic aqueductal stenosis, and was treated with a Torkildsen shunt. He had a

Figure Axial CT through cervical C2 level showing the Torkildsen's shunt tube imbedded into the cord causing a false syrinx.

history of alcohol induced peripheral neuropathy.

On admission he was alert, disoriented to time, and his attention and short term memory were very impaired. The only abnormality noted on his general physical examination was the right parieto-occipital burr hole for the shunt; vital signs were normal. Cranial nerve examination was unremarkable; there was no papilloedema. He had spasticity in all four extremities, marked hyperreflexia in both arms, areflexia in the legs, and bilaterally extensor plantar responses. Sensory examination showed marked vibratory and proprioceptive losses in the feet. He had dysmetria and intention tremor of all extremities and his gait was wide based and unsteady.

A cranial CT demonstrated dilatation of the lateral and third ventricles and a right parietal shunt tube terminating in the mid right ventricular body. A temporary ventriculostomy was made and the intracranial pressure readings were never higher than 8 cm H₂O over a 48 hour period. A CT myelogram revealed that the distal tip of the shunt tube was within the parenchyma of the upper cervical spinal cord (fig).

The patient had a cervical C1-C3 posterior laminectomy. The shunt tube was found imbedded in the spinal cord; consistent with the CT image. The tube was withdrawn from the cord cavity without difficulty and shown to be draining CSF. It was then shortened, repositioned in the subarachnoid space, and sutured to the dura at C2 level. No intraoperative complications occurred but on the third postoperative day the patient became lethargic and quadriparetic. An emergent CT of the head and cervical spine revealed increased ventricular size and a posterior cervical fluid collection from C2 to C4 that was displacing the cord. Despite the placement of a ventriculo-peritoneal shunt the patient failed to improve and died of a necrotising pneumonitis one month later.

The reported cases of migration of the Torkildsen's catheter describe the tube breaking loose from the dura and burying itself into an extra-arachnoidal position. The CSF then drains into the soft tissue and causes a blind sac in the upper cervical region and the intracranial hypertension reoccurs.³ This is probably what occurred in our patient on the third post operative day.

Our case is unusual since the catheter migrated into the dorsal aspect of the cervical cord causing a false syrinx. To our knowledge this late complication has not been reported before, but it should be noted for the patients who still have the ventriculocisternostomy catheter in place.

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- 1 Scarff JE. Treatment of hydrocephalus: an historical and critical review of methods and results. *J Neurol Neurosurg Psychiatry* 1963;26:1-26.
- 2 Torkildsen A. A new palliative operation in cases of inoperable occlusion of the Sylvian aqueduct. *Acta Chir Scand* 1939;82:117-24.
- 3 Paine KW, McKissok W. Aqueduct stenosis. Clinical aspects, and results of treatment by ventriculocisternostomy (Torkildsen's operation). *J Neurosurg* 1955;12:127-45.

Guillain-Barré syndrome associated with idiopathic thrombocytopenic purpura

There is mounting evidence that nerve injury in Guillain-Barré syndrome (GBS) is immunologically mediated, but the roles of cellular and humoral immune mechanisms are uncertain. The association of autoimmune diseases with GBS adds further support to the hypothesis of an autoimmune basis for this disorder. We report a patient who simultaneously experienced GBS and idiopathic thrombocytopenic purpura (ITP) following an upper respiratory tract infection.

This 75 year old woman developed acute progressive quadriplegia two weeks after the start of influenza-like symptoms consisting of fever, sore throat and coryza, which were treated with aspirin and amoxicillin. On examination there was flaccid areflexic quadriplegia (MRC grade 0 to 2), palsies of the right seventh and ninth cranial nerves, and distally diminished sensation in all extremities. Numerous petechiae were observed on the hands, wrists, legs and buccal mucosa. The spleen was not enlarged. Haemoglobin was 13.2 g/dl and haematocrit 38.8%. The white cell count was 12000/mm³, with a normal differential count. Platelet count was 6000/mm³. The prothrombin time and partial thromboplastin time were normal. Urinalysis was normal except for haematuria. Liver function studies, serum protein electrophoresis, antinuclear antibodies, rheumatoid factor, complement levels and LE test were all negative or normal. An increased number of megakaryocytes was found in an otherwise normal bone marrow examination. Platelet-associated and plasma autoantibodies were investigated by immunofluorescence techniques but failed to demonstrate any immunoreactivity. Prednisone, 60 mg daily, was administered and the platelet count gradually increased to 250000/mm³ on the seventh hospital day.