Ipsilateral hemi-Parkinsonism secondary to an astrocytoma

Parkinsonism has rarely been described in association with intracranial tumours. In a previous review by Polyzoisids et al in 1985, 49 verified cases with intracranial tumours were collected from the literature; the majority of which were meningiomas.9 10 12 Further review of the current literature adds another four cases.15 17 12 Only six of these 55 cases directly involved the thalamus and or the basal ganglia. All six were infiltrating intracranial gliomas.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 intracranial 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 left arm and head, with slight right sided weakness, 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 cranial nerve 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) 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 recognized and sometimes also produce hemidystonic syndromes.12 Most of these cases have been due to haemorrhages, infarction or tumours. Tumours may produce Parkinsonism either by pressure on the basal ganglia17 18 12 or more rarely by direct infiltration7 as in our case. Extra-axial masses impinging on the basal ganglia appeared to be more common than infiltrating lesions.6 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.12 Direct destruction of the basal ganglia may explain the clinical response to anti-Parkinsonian drugs in our patient and also in other patients described in the literature. Garcia de Yebenes J, et al reported a significant decrease of caudate postsynaptic dopamine receptors which could account 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 contralateral11 12 in contrast with our patient who presented with an ipsilateral hemi-Parkinsonism. Oliver7 and Polyzoisids et al1 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.

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

7 Pall HS. 74 year old lady who developed bilateral parkinsonism secondary to an intrinsic cerebral tumour. J Neurol Neurosurg Psychiatry 1987;50:1360.
nerve, but no evidence of tumour or abnormal vasculature. Postoperatively her visual acuity was unchanged.

Review of the literature reveals that this type of pathology is very unusual. Intraschial haematomata have been recognised,1 some of which are due to chiasmal glioma.1 Bleeding causing chiasmal compression has also been reported secondary to cryptic vascular malformations identified at operation2 and in patients with clinical evidence of vascular anomalies elsewhere.3 Burnbaum et al3 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.4 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 intraneural 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.


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 posterosalateral cervical gutter, where it is then sutured to the dura.5 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.6 The most frequent complications are infections and the development of a false meningocele 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 dural stenosis, and was treated with a Torkildsen shunt. He had a history of alcohol induced peripheral neuropathy.

On admission he was alert, disorientated 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 ventricular shunt 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 quadripleptic. 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 burrying 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 recovers.7 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.


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 quadriaparesis 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 quadriaplegia (MRC grade 0 to 2), palsy 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 bucal 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 technique 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.
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