Multiple crossed false localizing signs in a posterior fossa tumour

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SYNOPSIS Involvement of the lower cranial nerves as false localizing signs of intracranial tumour is very rare. A laterally placed posterior fossa meningioma gave rise to contralateral cerebellar ataxia and palsies of the fifth, ninth, and tenth cranial nerves.

With the earlier diagnosis of cerebral tumours in Western countries it is becoming increasingly rare to see striking false localizing signs with an intracranial mass. The following case is reported as a remarkable and unprecedented example in which a laterally placed posterior fossa meningioma gave rise to a cerebellar ataxia and fifth, ninth, and tenth nerve palsies on the *opposite* side. This is believed to be only the second case reported of the lower cranial nerves being involved in false localizing signs.

CASE REPORT

(SBH 554407) A 54 year old housewife had a two and a half year's history of morning retching, sometimes accompanied by twinges of occipital pain. Previously she had been well and there was no family history of neurological disease.

She was first referred to a general surgeon, several months after the onset of symptoms. A vagotomy, cholecystectomy, and hiatus hernia repair were accomplished at a single operation with only transient relief of her symptoms, to which was now added diarrhoea. Her occipital pain became more prominent and was now accompanied by neck stiffness, which failed to respond to cervical traction. It was now two years since her illness had begun and over the next five months she developed in succession, and progressively, unsteadiness of gait, numbness of the right side of the face and palate, transient diplopia with horizontal separation of the images, choking when swallowing fluids, and blurring of vision.

She was referred to a neurologist and was admitted to the neurosurgical unit in December 1974.

On examination her intellectual status was found to be normal and general examination was unremarkable, other than for a blood pressure of 190/120 mmHg. She had gross bilateral papilloedema. Corrected visual acuity was J4 on the right and J14 on the left. There was impairment of both pinprick and light touch sensation in all three trigeminal divisions on the right and the corneal reflex and corneal sensation were absent on the right. The palate was weak on the right side, and the palatal and pharyngeal reflexes and sensation were reduced on the right. No abnormality was found of the other cranial nerves. In particular, there was no evident deficit of the seventh or eighth nerves and the external ocular movements were full, without diplopia or nystagmus. The functions of the 12th and motor fifth nerve were intact. In the limbs there was incoordination of the right leg of a cerebellar type. No weakness, sensory loss, or pyramidal tract signs could be found. Her gait was slightly unsteady, tending to veer towards the right.

A clinical diagnosis was made of an extrinsic tumour on the *right* side of the posterior fossa, probably in the cerebellopontine angle. Chest and skull radiographs and tomograms of the internal auditory meati were normal. Audiometry and caloric testing were normal. A brain scan showed increased uptake of isotope on the *left* side of the posterior fossa (Figure). It was assumed that the scan had been incorrectly labelled for side, especially as a vertebral angiogram (Dr I. Wylie) was thought to show a mass on the right of the posterior fossa. The basilar artery was displaced forwards against the clivus and there was marked right to left displacement of the vermian branches of the left posterior inferior cerebellar artery and also of the inferior vermian vein.

At operation, the posterior fossa contents were

FIGURE Lateral and AP brain scans showing increased isotope uptake on the left of the posterior fossa.

found to be extremely tense with bilateral tonsillar herniation, which was more marked on the left side. On the left side of the posterior fossa was a 7.5 cm diameter vascular meningioma displacing the left hemisphere of the cerebellum almost to the midline. A total piecemeal removal of the tumour was carried out. It proved to be arising from the inferior leaf of the most lateral part of the tentorium, just above the most outer part of the petrous temporal bone.

Postoperatively, she made a recovery which was uncomplicated other than by several grand mal fits, thought to be related to her hydrocephalus. These were easily controlled and electroencephalography did not suggest any supratentorial focus, but only bilateral abnormalities consistent with previous ventricular dilatation.

Six weeks postoperatively, examination of the cranial nerves revealed no abnormality other than a slight reduction of the right corneal reflex and a slight dulling of right oropharyngeal sensation. Swallowing was now completely normal. There was no incoordination of the limbs, tested individually, and her gait was steady.

DISCUSSION

An intracranial tumour which has not metastasized may give rise to focal signs of disordered nervous function at a distance from itself in a number of ways. Ehni (1950) isolates the main mechanisms as (1) hydrocephalus, (2) meningeal reactions, (3) shifts of cerebral tissue distorting distant nerves and blood vessels, and (4) entrap-

ments of neural tissue by distant brain herniations between dural compartments of the skull.

Prominent false localizing signs are less common today than when James Collier (1904) found an incidence of 12.5% among 161 consecutive intracranial tumours; even he observed that they had become markedly less frequent in recent years. This he attributed to earlier diagnosis, as false localizing signs due to cerebral tumour tend to appear late in its course, when severe distortion and shifting of the intracranial contents are occurring. Indeed, he commented that the appearance of focal signs for the first time in the later stages of a cerebral tumour, as happened here, is often an indication that they may not indicate its true situation.

In a more recent review of the occurrence of false localizing signs among 250 intracranial meningiomas, Gassel (1961) points out that they are more common in patients with signs of raised intracranial pressure and that they are most misleading in cases where a large mass is situated in a 'silent' area. Of 20 subtentorial meningiomas in Gassel's series, only two showed false localizing signs (in each case dementia due to hydrocephalus), though according to Northfield (1973), a cerebellar convexity meningioma may give rise to a falsely localizing fifth nerve palsy.

In the present case two possible mechanisms

suggest themselves by which the left-sided tumour produced right posterior fossa signs. One is that the right cerebellar hemisphere might have been differentially impacted in the foramen magnum, and from reactionary oedema itself behaved as a mass lesion, compressing the lower cranial nerves and the spinal trigeminal tract. A similar mechanism was invoked by Batten and Collier (1899) to explain the dorsal column signs which occur with some intracranial tumours. However, this possibility is ruled out by the fact that, at operation, the left cerebellar tonsil was found to be more herniated into the spinal canal than the right one. The other possibility, which is more likely, is that the marked displacement of the left cerebellar hemisphere, almost into the right side of the posterior fossa, forced the brain-stem across to the left, thus exerting traction on various right-sided cranial nerves and compression of the right cerebellar structures. This supposition accords with the finding on vertebral angiography that the anterior vermian vessels were displaced to the left, a finding that preoperatively strengthened the diagnosis of a right-sided mass.

Of the cranial nerves, the fifth is seldom involved as a false localizing sign. Gassel (1961) had eight instances, the motor component being involved in only two of these. Jefferson (1938) thought that where a supratentorial tumour gives rise to a trigeminal palsy, the usual mechanism is herniation of brain into the middle fossa exit foramina of the various divisions. Ehni (1950) reported a case in which a cerebellar convexity meningioma produced a contralateral sensory and motor fifth nerve palsy. He postulated that the fifth nerve had become compressed between a blood-vessel crossing it and the outer bony wall of the posterior fossa.

There appears to be only one previous report in the English literature of the lower cranial nerves being involved as false localizing signs. In this case (Dodge *et al.*, 1955), a left parietal astrocytoma gave rise to palsies of the fifth to eighth right cranial nerves and also a right palatal palsy, all of which recovered after removal of the tumour. A case reported by Bramwell (1899) and mentioned by Gassel (1961) where a left frontal tumour led to difficulty in chewing and swallowing was almost certainly an example of the dyspraxia of swallowing and articulation which is sometimes seen with posterior frontal lesions in the dominant hemisphere (Meadows, 1973).

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