Sheaths, and anterior horn cells but no abnormalities in dorsal and ventral roots or in dorsal root ganglia. A sural nerve biopsy study indicated axonal neuropathy. A necropsy study showed destruction of anterior horn cells but no abnormalities of dorsal root ganglia and peripheral nerve.

Therefore the damage in this neuropathy could be located in the axon or in the nerve cell body. The studies carried out on our patients were characteristic of a pure axonal degeneration, but a coexisting lesion in the anterior horn cell cannot be ruled out.

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Trigeminal neuritis due to contralateral meningioma of the posterior cranial fossa

Posterior fossa meningiomas are uncommon intracranial tumours, accounting for about 10% of all intracranial meningiomas. They are now easily diagnosed by CT, but sometimes they grow very large before becoming evident. Impingement of ipsilateral cranial nerves (most often the 5th, 7th, 8th, and 9th nerves), cerebellar ataxia, and signs of increased intracranial pressure are the symptoms presenting most often. Infrequently the tumour may cause contralateral cranial nerve involvement, which confuses the diagnosis.

We report a case of meningioma of the posterior clivus with contralateral typical trigeminal neuritis as the main symptom. A 58 year old married woman complained of a right typical trigeminal neuritis of about one year’s duration, involving all three divisions of the nerve and described as “electric-like bursts”, triggered by eating. She had received 600 mg carbamazepine daily without effect.

Neurological examination showed only a minimal unsteadiness of gait and a fine horizontal nystagmus. The rest of the neurological examination was normal, in particular the corneal reflexes were preserved and there was no hypophagia or hypalgesia over the face and forehead. EEG and otological findings such as audiometry, caloric responses, electroneystagmography, and brainstem auditory evoked response were normal.

CT of the head showed a contralateral (left sided) high density lesion of the posterior fossa enhancing uniformly with intravenous contrast medium. The tumour, measuring 4 x 4 x 3 cm, distorted the brainstem and caused an initial triventricular hydrocephalus (figure). At surgery, the mass, removed in its entirety, was located in the posterior portion of the tentorium, close to the left transverse sinus and the confluence of the sinuses. Histological examination showed a meningioma of transitional type. The patient’s early postoperative course was favourable. The facial pain disappeared completely and a neurological examination after four months only showed a slight unsteadiness of gait. She did not complain of facial pain but six years later she was operated on for a recurrence of the tumour in the same region. The patient is still free of symptoms.

Contralateral involvement of the trigeminal nerve due to a mass occupying the posterior fossa space (such as meningioma) has been reported. In these cases, however, the involvement of the 5th nerve was generally manifest by early impairment of facial sensation and decreased corneal reflex. Very rarely, a contralateral trigeminal neuritis is the only symptom for a long time. Haddad and Tabat summarised 21 such cases from the medical literature. Six of these patients had a typical neuritis, 12 atypical, and in four cases the information available was not complete. There was a predominance of females and meningioma was the most frequent tumour.

In our case, the most plausible mechanism to explain the pathogenesis of the contralateral trigeminal involvement is that the tumour, situated in the back portion of the posterior fossa, pushed the brainstem and caused compression of the nerve root at its point of entry into the tentorial foramen. In conclusion, an isolated trigeminal neuritis, especially if carbamazepine resistance may be due to a contralateral tumour of the posterior fossa, such as a meningioma, and requires CT of the head. Surgical treatment relieves pain in almost all cases.

Panhypopituitarism after cavernous sinus thrombosis

Anterior panhypopituitarism is a rare late complication of cavernous sinus thrombosis. Posterior panhypopituitarism has not as yet been described. We report a 27 year old woman who presented eight months after septic cavernous sinus thrombosis with an Addisonian crisis and diabetes insipidus in association with a urinary tract infection.

The previously well woman gave a six month history of headache, increasing in the week before admission, and was found to have meningism. Her menses had been normal up to the time of admission. Measurements in CSF obtained by lumbar puncture gave a protein concentration of 43 mg/l, a normal glucose concentration (5.2 mM with a blood glucose of 7.5 mM), and a white cell count of less than 5 cells/mm³. No organisms were found. The next day she became drowsy (but obeyed commands) and pyrexial (38°C). She had a pulse of 80 beats/min and a blood pressure of 130/80 mmHg and was transferred for further assessment. There was left sided swelling with a right ptosis. She had no perception of light on the right but normal acuity (4/5) and pupillary responses on the left. The right fundus showed retinal venous congestion, with dilated left pupil. There was a complete right external ophthalmoplegia, with limitation on the left of elevation and depression with absent abduction, but normal adduction. The remainder of the examination was normal. A cranial CT showed opacification of the sphenoid sinus and part of the ethmoid sinuses, filling defects in the cavernous sinuses after intravenous contrast with dilatation of the orbital veins and proptosis. The pituitary appeared normal. There was a small low density area in the right frontal lobe consistent with ischaemia. A diagnosis of septic cavernous sinus thrombosis secondary to sphenoid sinusitis was made. Her electrolytes were normal. She was started on penicillin, chloramphenicol, and metronidazole. Exploration of the right sphenoid sinus yielded pus and a pneumococcus was grown from blood cultures. She improved...