sheaths, and anterior horn cells but no abnormalities in dorsal and ventral roots or in dorsal root ganglia.[3] A sural nerve biopsy study indicated axonal neuropathy.[1] A necropsy study showed destruction of anterior horn cells but no abnormalities of dorsal root ganglia and peripheral nerve.[4] Therefore the damage in this neuropathy could be located in the axon or in the nerve cell body. The studies carried out on our patient are characteristic of a pure axonal degeneration, but a coexisting lesion in the anterior horn cell cannot be ruled out.

M TINTORÉ
J MONTALBÁN
C CERVERA
A CODINA
Department of Neurology
I ROYO
J CRESPO
Department of Neurophysiology
C NAVARRO
Department of Neuropathology, Hospital General Universitari, Vall d'Hebron, Barcelona, Spain

Correspondence to: M Tintoré, Servei de Neurologia (3ª Planta anexes), Hospital General Universitari Vall d'Hebron, Psg Vall D'Hebron 119-129, 08035 Barcelona, Spain.

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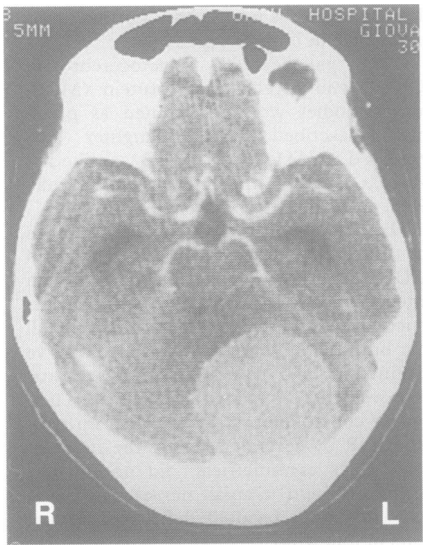
Trigeminal neuralgia due to contralateral meningioma of the posterior cranial fossa

Posterior fossa meningiomas are uncommon intracranial tumours, accounting for about 10% of all intracranial meningiomas.[1] They are now easily diagnosed by CT, but sometimes they grow very large before becoming clinically evident.[2] Impairment 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.[3] Infrequently the tumour may cause contralateral cranial nerve involvement, which confuses the diagnosis.[4 5]

We report a case of meningioma of the posterior fossa, presenting with contralateral typical trigeminal neuralgia as the main symptom. A 58 year old married woman complained of a right typical trigeminal neuralgia 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 present and there was no hypesthesia or hypalgesia over the face and forehead. EEG and otological findings such as audiometry, caloric responses, electronystagmography, and brainstem auditory evoked response were normal.



CT scan of the head showing a contralateral (left sided) high density lesion of the posterior fossa.

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 × 4 × 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.[1 5] 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 neuralgia is the only symptom for a long time. Haddad and Taha[4] summarised 21 such cases from the medical literature. Six of these patients had a typical neuralgia, 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.[4]

In conclusion, an isolated trigeminal neuralgia, especially if carbamazepine resistant, 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.

G MASE
M ZORZON
L CAPUS
E BIASUTTI
B VITRANI
G CAZZATO
Department of Neurology, University of Trieste, Trieste, Italy

Correspondence to: Dr M Zorzon, Department of Neurology, University of Trieste, Cattinara Hospital, Strada di Fiume 447, 34149 Trieste, Italy.

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Panhypopituitarism after cavernous sinus thrombosis

Anterior hypopituitarism is a rare late complication of cavernous sinus thrombosis.[1–5] Posterior hypopituitarism 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/mm.[3] 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 bilateral periorbital swelling with a right proptosis. 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 a fixed and dilated 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 congestion 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

Table 1 Water deprivation test after treatment with hydrocortisone, thyroxine, and carbamazepine

	Fluid balance (l)	Urine osmolality (mmol)	Plasma osmolality (mmol)
0 hours	0	114	288
8 hours	−1·8	231	299
After DDAV (2·0 g im)		587	290

DDAV = diamine-D-arginine-vasopressin; im = intramuscularly.

but three months later at follow up still had no perception of light in her right eye, with a pale optic disc. She had normal vision in her left eye, and full ocular movements.

One month later she re-presented with a two day history of headache, nausea, vomiting, and urinary and faecal incontinence. She had a fever of 39·2°C, a blood pressure of 92/48 mmHg, and a pulse of 98 beats/min. General examination showed mild diffuse lower abdominal tenderness. She was drowsy, but obeyed first order commands, and was localising pain, with mild nuchal rigidity, photophobia, and a pale right disc with a right afferent pupillary defect. Eye movements were normal other than a partial right sixth nerve palsy. She showed left sided hyperreflexia and a left extensor plantar response. CT of the brain showed a small anterior collection in the sphenoid sinus and a small area of low attenuation in the right medial frontal region unchanged from the previous scan. The pituitary was normal in size and position. Her CSF had a protein concentration of 47 mg/l, a glucose concentration of 3·5 mM (blood sugar 3·8 mM), and 10 lymphocytes/mm.[3] Serum electrolytes were mildly abnormal (sodium 142 mM, potassium 3·8 mM, chloride 113 mM, bicarbonate 22 mM, urea 6·3 mM, and creatinine 134 μM). She was treated empirically with cephotaxime, metronidazole, and benzylpenicillin and improved over 24 hours although no organism was identified. She then gave a history of amenorrhoea from the time of her original admission. Electrolytes were now abnormal (sodium 151 mM, potassium 3·5 mM, chloride 116 mM, bicarbonate 25 mM, urea 2·3 mM, and creatinine 85 μM). A diagnosis of Addisonian crisis was made and she was treated with hydrocortisone (100 mg six hourly) and over the next 36 hours improved considerably with her blood pressure rising to 120/70 mmHg. Cortisol concentration measured before starting treatment with hydrocortisone was abnormally low at 71 nM. Her serum osmolality was then 296 mmol with a urinary osmolality of 105 mmol indicating diabetes insipidus, later confirmed with a water deprivation test (table 1). She was also biochemically hypothyroid, with a TRH test showing a subnormal TSH and prolactin response to TRH (table 2). Growth hormone was undetectable and LH (2·0 mU/l) and FSH (4·0 mU/l) both subnormal. These results were interpreted as indicating panhypopituitarism. She was therefore treated with a combination of hydrocortisone, thyroxine, and desmopressin in replacement dosages. Oestradiol valerate was later added. Her blood pressure returned to 102/60 mmHg with no postural drop. Cranial MRI performed on recovery failed to show any abnormality in the region of the hypothalamus.

Anterior hypopituitarism after cavernous sinus thrombosis is thought to follow ischaemia related to venous thrombosis extending from the cavernous sinus to the hypophysial portal vessels, the sole blood supply of the anterior lobe.[4] Inflammatory involvement of the carotid artery may underlie the seizures and focal areas of cerebral infarction sometimes seen, as in our case, and may have also produced the posterior hypopituitarism by causing hypothalamic infarction, although this could not be detected on cranial MR images.[5] Our experience and that of others[3-5] is that hormonal deficiency may present catastrophically and that hypopituitarism should be actively searched for in all patients and may be an additional factor in the high mortality from cavernous sinus thrombosis.

R K H PETTY
J WARDLAW
P G E KENNEDY
B O'REILLY
R McFADZEAN
R A JOHNSON
Institute of Neurological Sciences, Southern General Hospital, Glasgow G51 4TF, UK

D L DAVIES
Western Infirmary, Glasgow G11 6NT, UK

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Table 2 TRH test

Time after TRH	Prolactin (mU/l)	TSH (mU/l)	T4 (nM)	T3 (nM)
0 min	<20	0·35	30	0·3
20 min	<20	0·45		
60 min	<20	0·53		
Reference ranges	350	0·4–5·0	50–144	0·9–2·7

Olfactory disturbances as the initial or most prominent symptom of multiple sclerosis

Although common in the general population, symptomatic olfactory disturbances are rare in multiple sclerosis.[1 2] When present, they are typically found in longstanding disease with other neurological manifestations. We report two patients with olfactory disturbances as the initial or most prominent symptom of multiple sclerosis.

Patient 1, a previously healthy, 36 year old woman acutely developed parosmias six months before evaluation. She perceived many items, particularly food, as smelling of gasoline. As a result, she developed an aversion to food and a 20 lb weight loss. Three months later, she noted persistent fatigue. Her doctor suspected depression, but a psychiatric evaluation and neuropsychological testing showed no evidence of an affective disorder or cognitive dysfunction. Subsequently, she developed left sided weakness and difficulty walking, prompting evaluation at the University of Pennsylvania. Her medical history was unremarkable apart from her smoking 20 cigarettes daily for 10 years. Neurological examination showed Lhermitte's phenomenon, bilateral optic disc pallor with normal visual acuity, mild spastic left hemiparesis, diffuse hyperreflexia, and bilateral Babinski signs. Cranial MRI showed multiple lesions consistent with demyelinating foci in the hemispheric white matter, brainstem, and cerebellum. There was no sinus disease. The cerebrospinal fluid contained 7 white cells/mm^3 (all lymphocytes), 0·5 g/l protein 68 mg/100 ml glucose, raised IgG, and oligoclonal bands. Blood tests was negative for collagen vascular disease including Sjogren's syndrome, syphilis, HIV, and HTLV-I infection, B12 deficiency, sarcoidosis, and thyroid disease. Olfactory testing was performed with the University of Pennsylvania smell identification test (UPSIT), a widely used, standardised, microencapsulated, 40 odour, 4 item forced choice test.[1] On an abbreviated study, she identified 11 of 15 odours, equivalent to scoring below the 10th percentile for age matched women who smoked. The patient requested that testing be terminated because the test items smelled like gasoline. Methylprednisolone treatment (1 g intravenously each day for five days), produced rapid improvement in her left hemiparesis and olfactory symptoms. On a full length UPSIT two weeks later, she identified 35 of 40 items, including 14 of the 15 previously tested, corresponding to the 70th percentile.

Patient 2, a 48 year old woman was referred to the University of Pennsylvania for evaluation of possible multiple sclerosis. Twenty years earlier, she had developed urinary frequency, which resolved spontaneously over two months. After an uneventful pregnancy two years later, she developed bilateral, sequential optic neuritis. At age 48, she noted an acute loss of smell, prompting her to seek medical attention. She denied other neurological symptoms apart from decreased vision. Medical history was significant for adult onset diabetes mellitus, well controlled with insulin. Examination showed bilateral optic disc pallor with visual acuities of 20/100 OD and 20/40 OS, mild ocular ataxia, and bilateral Babinski signs. There was no evidence of retinopathy or peripheral neuropathy.