Hepatitis tests, six differentiated. Her normal. Treatment prolonged (0 protein
45 nerve 45
iliopsoas
The appeared days,
depth
pronounced
lower limb weakness. On
Pinprick
also the

Epstein-Barr
respectively.

formation,
showed massive loss of

vibration and

(right
slowed
and
conduction velocities were

and lateral nerves,
and
pronounced

of

and

legs,

of

and

slowly

of

and

years of

weeks

and

months

for

and

immunological
abnormality on auxiliary and serological
and

antibodies

and

medications in

and

myelin
fibres
and

with

inflammatory
reaction.

few

his,
clinic

conduction
velocities

later

weakness

progressed.

and

but

of

and

legs,

and

and

infasting plasma glucose

18:1 µU/ml. A fasting test showed hyperglycaemia associated with

insulin analogues in type 2 diabetes.

The

of

local nerve, F wave velocities were decreased except for the left

tibial nerve. Electrophysiological studies showed evidence

deviation was found in the left vastus

medialis. These abnormalities were compatible with

an axonal sensorimotor neuropathy.

Sural nerve biopsy showed axonal degeneration.

Other causes of acquired polyneuropathy

such as carcinomatous polyneuropathy,

paraproteinemia, uremia, alcohol, diabetes mellitus,

cystic fibrosis, familial amyloidosis, and hypothyroidism were excluded but a 4 cm insulinoma was identified

and surgically removed. Hyperglycaemic attacks have not occurred and there has been motor improvement but "paraparesis" has persisted during the next three years.

Electrophysiological studies demonstrated no significant change.

Peripheral neurological symptoms in the course of hyperglycaemic attacks are unusual and are probably due to maintained hyperglycaemia rather than secondary to hyperinsulinemia. After excision of a pancreatic tumour, sensory symptoms resolved but definite improvement in muscle power is uncommon. After three years of follow-up, no objective sensory impairment was evident in our patient. Motor improvement was also found. Electrophysiological studies were carried out in only 10 of the 30 cases previously reported. In one case, there was an analysis of nerve histology and there were two necropsy studies. The neuropathology of this syndrome, however, remains controversial. Muscle histology has been described in three cases, showing nerve fibre atrophy and nerve fibre regeneration.

Pathological studies disclosed degeneration of pyramidal tracts, myelin

Peripheral neuropathy in association with insulinoma: clinical features and neuropathology of a new case

Peripheral neuropathy associated with hyperglycaemia secondary to insulinoma is unusual and just 30 patients have been described. Three years before admission this 60 year old woman started episodes of confusion, self harm, irritability and ataxia. After three months before entry, she noted progressive loss of sensation and weakness below the knees and hands. Neurological examination disclosed a mild paresis with mild impairment of all sensations in a glove and stocking distribution. The deep tendon reflexes were decreased. Laboratory investigations showed a fasting plasma glucose concentration below 50 mg/dl and a 2 hour plasma glucose concentration of 18:1 µU/ml. A fasting test showed hyperglycaemia associated with inappropriately raised insulin levels. Coelic angiography, abdominal echography, and abdominal CT scan showed a 3 cm mass in the head of the pancreas. The motor nerve velocities were slightly decreased and there was an important reduction in muscle action potential amplitude. Sensory nerve action potentials were absent with the exception of the right median nerve; F wave velocities were decreased except for the left tibial nerve. Electrophysiological studies showed evidence of denervation in the right first dorsal interosseous, tibialis anterior, and both extensor digitorum brevis muscles. No denervation was found in the left vastus medialis. These abnormalities were compatible with an axonal sensorimotor neuropathy.
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 patient are characteristic of a pure axonal degeneration, but a coexisting lesion in the anterior horn cell cannot be ruled out.

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

Trigeminal neuroma 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 symptomatic. Lesions 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 fossa with contralateral typical trigeminal neuroma as the main symptom. A 58 year old married woman complained of a right typical trigeminal neuroma 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 hypealgesia over the face and forehead. EEG and otopathological findings such as audiometry, caloric responses, electroneurogrammography, 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 neuroma is the only symptom for a long time. Haddad and Tahar summarised 21 such cases from the medical literature. Six of these patients had a typical trigeminal, 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 neuroma, 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.

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Panhypopituitarism after cavernous sinus thrombosis

Anterior hypopituitarism is a rare late complication of cavernous sinus thrombosis. 1,2 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 headaches, increasing in the week before admission, and was found to have hypopituitarism. 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. 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 marked orbital 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 an involved retina and 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 compression 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 secondarily 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 polymorphonuclear was grown from blood cultures. She improved on June 5, 2022 by guest. Protected by copyright.