after admission, a week before the appearance of the cutaneous eruption.

The pathogenesis of HSV-2 myelitis in this case remains ill defined. Although IgM antibodies were detected in the first serum sample we are not sure that the patient's myelodacilitis reflected true HSV-2 primary infection because reappearance of IgM antibodies can be detected in herpes virus reactivation. All but one previously reported case of HSV-2 myelitis were fatal and occurred in immunocompromised hosts. This patient was neither diabetic nor HIV-infected, but despite normal CT examinations of the thorax and abdomen, a hidden malignancy cannot be excluded.

Reactivation of a latent infection within dorsal root ganglia neurons with a contiguous spread via sacral ganglia to the spinal cord has already been proposed in HSV-2 ascending myelitis. It is of note that despite antiviral treatment, our patient died within six weeks. Thus an immunologically mediated injury triggered by the herpes infection could also be involved here, as in another patient with HSV-1 myelitis whose necropsy examination showed patchy demyelination of the spinal cord.

EMMANUEL ELLE
Service de Neurologie, Hopital du Haut-Leveque, CHU Bordeaux, France

FLORE ROZENBERG
Service de Bacteriologie-Virologie, Hopital Saint-Vincent de Paul, Paris, France

VINCENT DOUTSSET
Service de Neurologie, CHU Bordeaux, France

MARIE BEYLOT-BARRY
Service de Dermatologie, CHU Bordeaux, France

Correspondence to: Dr Emmanuel Elle, INSERM U139, rue Camille-Saints, 33077 Bordeaux Cedex, France.


Peduncular hallucinosis and right hemiparkinsonian causd by left mesencephalic infarction

Since the original description of Lhermitte's several cases of peduncular hallucinosis have been reported, but always in relation to a bilateral mesencephalic lesion. We describe here a patient with prolonged visual hallucinations and right hemiparkinson-

ism. An MRI showed a unilateral infarction involving the left cerebral peduncle.

A previously healthy 70 year old hander woman presented with visual hallucinations two months previously, she began one night, to see objects (motor-bikes), animals (dogs, horses), and people (Japanese) entering and driving silently round her room, across the entire visual field. Although the images were of normal colours and sizes, she was aware that they were not real, and never described “deja vu” or “jamais vu” phenomena, tactile, or auditory hallucinosis. The hallucinatory events became progressively longer and more frequent — up to hours to minutes — during both day and night. Her medical history included mild hypertension, normofunctional multinodular goitre, and surgery for left breast cancer six years earlier, without subsequent evidence of recurrence. There were no other remarkable personal or familial antecedents. On examination, she was alert, oriented, and cooper-

ative. She remembered four of five words after five minutes, and the mini mental state test was 32/35. She remained in a left tilted posture, and showed severe impairment of postural reflexes, mild bradykinesia, cogwheel rigidity, and postural instability (nystagmus) in the right extremities (mainly in the lower limb). Tendon reflexes were brisk and increased on the right side, but there was no clonus and the plantar responses were flexor. There were no other remarkable findings on general or neurological examination. Laboratory investigations, including ESR, routine haematological, biochemical, and immunological studies, thyr-

don function tests, serological tests for syphilis, cerebrospinal fluid examination, EEG, and cranial CT were normal or negative. An MRI showed an abnormal high intensity signal in the left cerebral peduncle on T2-weighted images (figure). Multiple foci of T2-weighted high signal intensity were also seen throughout the periventricu-

lar white matter. Such findings were consist-

ent with ischaemic damage. Despite treatment with haloperidol and phenytoin the patient became more impaired within the next several weeks, showing continuous visual hallucinations, with frequent episodes of agitation and disorientation.

Vascular lesion of the upper brainstem is the most often cause of peduncular hallucini-

nosis. A case of Sudden hallucinosis due to bilateral mesencephalic infarction diagnosed by MRI has been recently reported. The present one is the first report, however, to our knowledge, of peduncular halluci-

nosis due to unilateral lesion. Although the precise anatomical basis for peduncular hallucinosis remains unclear, it seems that the substantia nigra pars reticulata (SNpr) is directly implicated. Little is known about the pathogenesis of peduncular hallucinosis. A relation with the rapid eye movement (REM) phase of sleep has been proposed. In this sense, it is known that the SNpr may play an important part in the regulation of the different phases of sleep through its connections with centromedian/parafascicular-

lar nuclei of the thalamus, superior collicu-

lus, and reticular formation.

Finally, although we cannot rule out the presence of brainstem encephalitis, it is probable that the right hemiparkinsonism found in our patient was related to ischaemic damage of the left substantia nigra pars compacta.

RAUL DE LA FUENTE FERNANDEZ
JOSÉ MARÍA LÓPEZ
PABLO REY DEL CORRAL
neurology

FERNANDO DE LA IGLESIAS MARTINEZ
Department of Internal Medicine,
Hospital Juan Carlos I, La Coruña,
Spain.

Correspondence to: Dr Raul de la Fuente Fernandez, Servicio de Neurología, Hospital Juan Carlos I, Xabieros de Arribas, 84, 15006 La Coruña, Spain.

1 Lhermitte J. Syndrome de la calotte du pédon-


4 Beckstead RM, Frankfurter A. The distribution and some morphological features of substantia nigra that project to the thala-

mus, superior colliculus and pedunculoponti-


Intraneural ganglion of the sciotic nerve detected by ultrasound

Intraneural ganglia are a rare cause of peripheral nerve lesions most often affecting the peroneal nerve. Their origin is unknown. Some 50 cases were reported up to 1979. Sciatric nerve ganglia are very rare. We report a sciatric nerve lesion caused by a giant ganglion situated at the level of the distal thigh and damaging the tibial portion only.

A 36 year old male right handed and right footed state officer complained of pain in his right calf for about six years especially when jogging and walking for more than 30 minutes. He was treated for a varicose disc hernia, but a lumbar CT was unremarkable. Simultaneously he noted discomfort in his right toes when wearing shoes; this was relieved by wearing orthopaedic sandals.

On first neurological examination he was found to have Laségue's sign with his right leg at an angle of about 85°. Reflexes and sensation in the lower extremities were nor-

mal. There was moderate tenderness of the toe flexors. Plain radiographs of the lower spine showed six lumbar vertebrae and a fissured vertebral arch of the first sacral segment.

Axial T2-weighted MR image obtained with a 0–T unit shows an area of hyperintensity in the left cerebral peduncle, consistent with infarction.
On electromyographic examination fibrillation potentials and positive sharp waves were recorded from the abductor hallucis and flexor digitorum brevis muscles. Motor conduction velocity of the right tibial nerve was slow on the right (38 m/s) and normal on the left (55 m/s) side; sensory nerve conduction velocity of the right sural nerve and H-reflexes bilaterally were normal.

Eleven months later the pain was localised to the outer aspect of the right calf and foot, and the ankle jerk was diminished on the right side. Weakness of the toe flexors was unchanged, Hoffmann-Tinel's sign was positive over the posterior tarsal tunnel and palpation of the popliteal fossa yielded neither a mass nor tenderness. Sensory nerve conduction velocity of the right medial planar nerve could not be determined. The distal motor latency of the right tibial nerve was prolonged (7.8 ms).

As the findings were not typical of a tarsal tunnel syndrome, surgery was discouraged.

Thirteen months later MRI of the lower leg up to the popliteal fossa showed no abnormalities other than atrophy of the popliteal and posterior tibial muscles. Electromyography yielded equivocal evidence of neurogenic damage to the posterior or tibial muscle prompting examination of the proximal tibial and sciatic nerve by B-scan ultrasound. Here a cystic formation of about 7 x 3 x 3 cm was found at the bifurcation of the sciatic nerve at the level of the distal thigh (figure). A subsequent transaxial CT confirmed this result.

The patient was operated on one week later. A spindle like multiloculated cyst measuring roughly 15 x 3 x 2 cm was found between the biceps and semitendinosus/semitendinosus muscles within the sciatic nerve, producing a gelatious mass on incision. The cyst was excised by a microsurgical technique leaving the fascicles of both the tibial and peroneal compartment intact. Histologically, a benign ganglion with pseudocystic walls was found. After the operation paresis of the short and long toe flexors was more pronounced and the ankle jerk and tibialis posterior reflex were absent, accompanied by mild trophic changes. One year later the pain had disappeared and walking for two hours was possible without complaint. The ankle jerk was still absent, flexion and distention of the toes parietal, the tips of the toes hypesthetic, and the sole of the foot anhidrotic.

Having first sought the location of the lesion too far cephalic (lumbar disc hernia) it subsequently was found too far caudal (tarsal tunnel) because of clinical involvement of the toe flexors only. Sciatic nerve lesions are more prone to involve the peroneal than the tibial fascicles and the cyst was missed initially. It was the ultrasound technique which gave the first clue as to the true level of the sciatic ganglion.

Although the ganglion in this patient was located proximal to the bifurcation of the sciatic nerve into the tibial and peroneal portion, only tibial fibres were affected. The origin of the cyst is not clear. A mucoid degeneration of collagen fibres, metaplasia of irritated connective tissue, ectopia of embryonal synovial tissue, or a mucoid degeneration of a formerly solid tumour for example, a Schwannoma—are possibilities.

After Mahaley's first report of a ganglion of the (posterior) tibial nerve at the popliteal fossa other ganglia or (Baker's) cysts were also found at the tarsal tunnel.1 Only two intraneural synovial cysts of the sciatic nerve were reported between 1980 and 1991.2

However rare, a ganglion such as this must be considered in the differential diagnosis of L5 and S1 root, sciatic, tibial and peroneal nerve lesions, as it is amenable to surgery and results are usually favourable. Like other authors3 we strongly recommend nerve ultrasonography in any case of progressive peripheral nerve damage of unexplained origin.

The MRI was done by Dr V Buchholz and colleagues, Erlangen.

Correspondence to: PD Dr Christoph J G Lang, Neurologische Universitätsklinik, Schwabachanlage 6, D-91054 Erlangen, Germany.


Familial Parkinson’s disease and polymorphism at the CYP2D6 locus

Because 10% of patients with Parkinson's disease have an affected relative1 and kindred with a pattern of disease transmission compatible with autosomal dominant inheritance have been described,2 genetic factors may play a part in the pathogenesis of the disease. Defective 4-hydroxylation of debrisoquine by CYP2D6, a member of the cytochrome P-450 family, has been found in more than 50% of patients with idiopathic Parkinson's disease, but in less than 20% of controls.3 Four alleles of the CYP2D6 gene, containing point mutations or deletions which inactivate the gene, result in a poor metaboliser phenotype.4 The mutant allele CYP2D6B in particular, occurs twice as often in the patients than in the controls, with a relative risk ratio of 2.7.5 The risk of idiopathic Parkinson's disease in those with the CYP2D6 mutant
Intraneural ganglion of the sciatic nerve: detection by ultrasound.

C J Lang, U Neubauer, S Qaiyumi and R Fahlbusch

J Neurol Neurosurg Psychiatry 1994 57: 870-871
doi: 10.1136/jnnp.57.7.870-a

Updated information and services can be found at:
http://jnnp.bmj.com/content/57/7/870.2.citation

These include:
Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/