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 myeloradiculitis 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 have been involved, as in another patient with HSV-1 myelitis whose necropsy examination showed patchy demyelination of the spinal cord.1

EMMANUEL ELLIE
Service de Neurologie, Hôpital du Haut-Lévêque, CHU Bordeaux, France

FLORE ROZENBERG
Service de Bactériologie-Virologie, Hôpital Saint-Vincent de Paul, Paris, France

VINCENT DOUSSET
Service de Neuroanatomie, CHU Bordeaux, France

Correspondence to: Dr Emmanuel Ellie, INSERM U394, rue Camille-Saints, 33077 Bordeaux Cedex, France.


Peduncular hallucinosis and right hemiparkinsonism caused by left meningeal infarction

Since the original description of LHermite1 several causes of peduncular hallucinosis have been reported, but always in relation to a bilateral meningeal lesion. We describe here a patient with prolonged visual hallucinations and right hemiparkinsonosis 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.2 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 centromedial/parafascicular nuclei of the thalamus, superior colliculus, and reticular formation.4

Finally, although we cannot rule out the presence of brainstem etiologies, it is probable that the right hemiparkinsonism found in our patient was related to ischaemic damage of the left substantia nigra pars compacta.5

Raul de la Fuente Fernandez
Pablo Rey del Corral
Pablo Reymundo
Fernando de la Iglesía Martínez
Department of Internal Medicine, Hospital Juan Canalejo, La Coruña, Spain.

Correspondence to: Dr Raul de la Fuente Fernandez, Servicio de Neurologia, Hospital Juan Canalejo, Xabier Arribas 84, 15006 La Coruña, Spain.


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Electromyography yielded equivocal or ultrasound. Here proximal tibial and posterior tibial muscles. other tarsal nerve mined. The neither was positive slow on the the on localised potentials the on the left (55 -8 m/s). As 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 plantar 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 this 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 gelatinous 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 reflexes 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 parietic, 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 sought 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 situated 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, ektropia 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. Only two intraneural synovial cysts of the sciatic nerve were reported between 1980 and 1991.

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 authors 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 relative and kindred with a pattern of disease transmission compatible with autosomal dominant inheritance have been described, 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. 1 Four alleles of the CYP2D6 gene, containing point mutations or deletions which inactivate the gene, result in a poor metaboliser phenotype. 2 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. 3 The risk of idiopathic Parkinson's disease in those with the CYP2D6 mutant

(A) Longitudinal (left) and transaxial (right) section of the distal thigh by B-scan ultrasound (Siemens Sonoline SL-2). Septated echogenic space occupying mass (black and white solid bold arrows) measuring 2 cm in diameter distending and displacing the sciatic nerve (+ and +), which measures 7-0 x 4-9 x 9-2 mm. (B) Transaxial CT through the distal thigh (Siemens Somatom DR) confirmed a septated cystic space occupying hypodense mass of 2-8 cm in diameter (slim black arrows) less dense (11 to 12 Hounsfield units) than the surrounding muscles (moderately hypodense, semimembranosus, and biceps femoris).
Intraneural ganglion of the sciatic nerve: detection by ultrasound.

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

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