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.[3] 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.[7]

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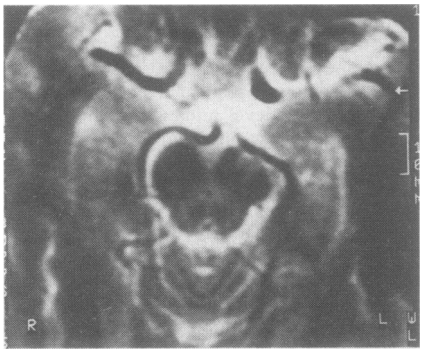
Peduncular hallucinosis and right hemiparkinsonism caused by left mesencephalic infarction

Since the original description of Lhermitte[1] several causes 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 hemiparkinsonism. An MRI showed a unilateral infarction involving the left cerebral peduncle.

A previously healthy 70 year old right handed woman presented with visual hallucinations. Two months previously, she began, one night, to see objects (motorbikes), 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, lasting from minutes to hours, during both day and night. Her medical history included mild hypertension, normofunctional multinodular goitre, and surgery for left breast carcinoma six years earlier, without subsequent evidence of recurrence. There were no other remarkable personal or familial antecedents. On examination, she was alert, oriented, and cooperative. 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 intermittent resting tremor 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, thyroid 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 periventricular white matter. Such findings were consistent 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 hallucinosis. A case of peduncular hallucinosis due to bilateral mesencephalic infarction diagnosed by MRI has been recently reported.[2] The present one is the first report, however, to our knowledge, of peduncular hallucinosis 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.[3] 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 nuclei of the thalamus, superior colliculus, and reticular formation.[4]



Axial T2-weighted MR image obtained with a 0·5-T unit shows an area of hyperintensity in the left cerebral peduncle, consistent with infarction.

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

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Intraneural ganglion of the sciatic nerve: detection 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.[1] Sciatic nerve ganglia are very rare. We report a sciatic 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 lumbar 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 Lasègue's sign with his right leg at an angle of about 85°. Reflexes and sensation in the lower extremities were normal. There was moderate paresis of the toe flexors. Plain radiographs of the lower spine showed six lumbar vertebrae and a fissured vertebral arch of the first sacral segment.