LETTERS TO THE EDITOR

Occupational meralgia paraesthetica

Meralgia paraesthetica is a common, and usually unilateral mononeuropathy\(^3\) characterised by a particularly unpleasant pain in the anterior and lateral aspect of the thigh, in the territory of the lateral cutaneous nerve of the thigh. Aetiology of the condition can be identified. We have seen four patients with meralgia paraesthetica that could be attributed to their work, when it was necessary for them to support heavy bundles on the anterior and superior aspect of the right thigh.

The first case was a healthy 44 year old male. His symptoms began seven months earlier with burning pain in the anterior and lateral aspect of the right thigh. From the age of 13 years he had worked as a sidewalk salesman, carrying large heavy trays which he supported on the anterior superior part of the right thigh.

Examination revealed hypoaesthesia in the territory corresponding to the right lateral cutaneous nerve of the thigh. Palpation of the right iliac crest triggered dysaesthesia in this region. Once the patient stopped supporting weight on this thigh his symptoms improved.

The second patient was a 55 year old mason. He complained of a disagreeable "pinprick" numbness of the anterolateral aspect of the thigh, which also developed aching. He had a history of more than 30 years of loading and unloading heavy sacks catching them on the anterior face of the right thigh as they were dropped from the truck.

Physical examination revealed hypoaesthesia with hyperpathia on the anterior and lateral face of the right thigh and Tinel's sign at the level of the anterolateral iliac spine. The condition improved when he discontinued work.

The third case was a 45 year old male. This mason usually lifted 50 kg bags of cement from the floor to the anterior face of the right thigh and then shifted them to their destination at 38 years he experienced dysaesthesia of the anterolateral face of the right thigh. Palpation of the anterolateral iliac spine generated "pinpricks" in the territory innervated by the right femoral cutaneous nerve, which also evidenced hypealgasia. The patient continues his employment and his symptoms persist.

The last case was a 45 year old male. Part of his work consisted of supporting the edge of large sheets of metallic plastic on the right groin. A few weeks after beginning his work he complained of "electrical" pains, which extended to the lateral aspect of the right thigh. Physical examination confirmed painful hypealgasia of the region and Tinel's sign could be elicited at the level of the anterolateral iliac spine. Changing his work alleviated the symptoms.

The quality and distribution of the pain, the presence of Tinel's sign, the absence of motor deficits, sensory abnormalities, and the normal results of the complementary tests characterise our patients as typical cases of meralgia paraesthetica. Our patients did not have diabetes or significant obesity, which have been described as responsible for some cases.\(^3\)

Although our patients could have been categorised as idiopathic, their working conditions suggest a traumatic origin. In all four cases the right lateral cutaneous nerve of the thigh was compressed by repeated blows to the groin, where the nerve leaves the abdominal cavity. Discontinuing this activity can alleviate symptoms. We therefore suggest that patients should be asked about their occupational history.

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**Figure MRI: SE\(_{1000}\). Paramedian high signal intensity lesion (arrowed).**

Vertical gaze palsy due to a resolving midbrain lesion

Vertical gaze palsies usually result from midbrain damage due either to tumours or vascular lesions. Less frequent causes include progressive supranuclear palsy\(^3\) and Niemann-Pick disease, type C\(^4\). We describe a case of vertical gaze palsy associated with a rostral midbrain lesion shown on magnetic resonance imaging (MRI). This clinical and MR imaging abnormalities subsequently resolved completely.

A 21 year old female presented with painless horizontal diplopia which had a stuttering onset over a few hours. She tended to stagger to her right. Eight weeks earlier she had developed varicella. Although typical in all other respects, this was a severe infection keeping her bed-bound for three weeks and away from work for seven. Examination revealed residual scarring over her face and trunk. Visual acuities were 6/4, N4-5 in each eye, and fundoscopy was normal. Her visual fields were full to a 1° arc, while target at 1 metre. Vertical saccades and pursuit eye movements were considerably reduced and on attempted upgaze there was divergence of the left eye. Horizontal saccades and pursuit movements, as well as convergence were normal. Both horizontal and vertical oculocephalic movements were full. Pupillary reflexes were brisk and symmetrical without evidence of light near dissociation. Neither lid retraction nor convergence/retraction nystagmus were present. The remainder of the examination was normal.

Cranial computed tomodography (CT) with contrast was normal. MRI was performed and a Picker 0-5 T superconducting machine using axial SE\(_{1000}\) 4 mm slice thickness sequences. A single region of increased signal in the upper midbrain, at the level of the red nuclei, was demonstrated just to the left of the midline and ventral to the aqueduct of Sylvis (fig.). Her signs remained unchanged for a week. The vertical eye movements then slowly improved, with reversion to normal over one month. Repeat MRI 14 months later showed that the midbrain lesion had resolved, and that no new lesions had appeared.

Lesions affecting both upgaze and downgaze are situated in the rostral midbrain and are thought to involve the interstitial nucleus of Cajal, the rostral interstitial nucleus of the medial longitudinal fasciculus and possibly the nucleus of Darkschewitz.\(^5\) There are usually tumours or infarcts. In this case, however, the spontaneous complete resolution of the lesion is more in keeping with a demyelinating lesion. There is no additional evidence for a diagnosis of multiple sclerosis, though it cannot be excluded. An alternative possibility is that she had a localised post-infectious encephalitis following varicella, a sequence well known to occur even in the adult. Resolution of MRI abnormalities has been reported in both multiple sclerosis\(^6\) and post-infectious encephalomyelitis.\(^7\) This is the first report of a remitting vertical gaze palsy in association with an appropriately sited resolving MRI lesion.

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Mumps and Guillain-Barré syndrome

A variety of neurological sequelae to mumps have been reported including meningitis, encephalitis, myelitis and rarely polyneuropathy. By 1981 a total of ten cases fulfilling accepted diagnostic criteria for Guillain-Barré syndrome had been reported and half of these cases were associated with orchitis. We can find no subsequent reports of this association and would like to report a further case of Guillain-Barré syndrome following mumps parotitis and orchitis.

A 48 year old male was admitted on 9 December 1988 with progressive generalised muscle weakness. Three weeks earlier he had developed bilateral painful parotid swelling and within days this was followed by pain and swelling of the left testicle. There was no previous history of mumps infection but in 1963 the patient had suffered a traumatic upper lumbar fracture dislocation at T12/L1 with moderate residual flaccid paraparesis.

On admission the patient gave a five day history of distal paraesthesia and progressive weakness of all limbs and a 24 hour history of increasing bilateral facial weakness. He normally practised intermittent self-catheterisation and had not noticed any change in sphincter function. Examination revealed severe bilateral facial weakness, moderate weakness of all limbs and marked weakness of the trunk. In the limbs there was generalised weakness graded MRC 3 proximally and MRC 2 distally and all deep tendon reflexes were absent. Pinprick sensation was impaired bilaterally to the wrist and there was widespread abnormality of superficial sensation in the legs related to the previous injury.

The clinical diagnosis of Guillain-Barré syndrome was supported by investigations. The cerebrospinal fluid (CSF) was acellular with an elevated protein content of 1.2 G/L and nerve conduction studies revealed normal peripheral conduction and delayed F waves. Other routine investigations were normal, including urinary porphyrin estimation.

Complement fixation tests for mumps antigen were performed on admission and again two weeks later. The S antigen in mumps infected peaks within the first two weeks of infection and declines thereafter. The V antigen appears at the end of the first week after infection and persists, sometimes for years, as a marker of previous infection.1 In our case the S antigen on admission was 128, declining to 64 two weeks later. The V antigen was 64 on admission, and had not changed at the second examination. These titres are compatible with recent mumps infection. Viral cultures were negative, but mumps virus is only detectable in the CSF for four days after infection,1 and our failure to culture the virus is in keeping with the three week period at presentation.

In the days following admission the limb weakness progressed and the patient became bed-bound with no useful upper limb function. Respiratory function was carefully monitored throughout admission and assisted ventilation was not required. Plasmoapheresis was started on the second day and the patient had five exchanges. An improvement in muscle power was noted on the second day and he subsequently made a rapid recovery. By the time of discharge two weeks after admission there had been a complete functional recovery.

The clinical and investigative features in this patient agree with accepted diagnostic criteria for Guillain-Barré syndrome. The temporal relationship to serologically proven mumps infection suggests a causative relationship as in other viral infections including mononucleosis, acute encephalitis and cytomegalovirus. Muscle weakness associated with a lymphocytic pleocytosis, thus mimicking poliomyelitis, may occur in mumps but this usually accompanies the acute illness.2 Polyneuropathy develops one to three weeks following the viral infection and in the majority of cases is preceded by mumps orchitis. This is not likely to reflect the severity of infection and is unexplained. Nonetheless, it is important to recognise Guillain-Barré syndrome as a potentially serious complication of mumps infection and this could be of particular relevance in view of the recent widespread introduction of mumps vaccination.

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The role of combined valve prolapses in the prognosis of cerebro-vascular ischaemic attacks associated with mitral valve prolapse

The role of Mitral Valve Prolapse (MVP) as a cause of ischaemic stroke (IS) remains controversial. MVP has been associated with cerebral ischaemic events, especially in young adults, though the incidence of ischaemic stroke in subjects with MVP is quite rare. This paradox may be explained by the existence of a subgroup of patients with MVP with specific characteristics that may lead them to carry a high risk for suffering IS.

In a recent paper Barletta et al.1 suggest that ischaemic stroke associated with MVP could be related to the presence of myxomatous degeneration of several valves. These authors found that such patients were more frequent in patients with IS and MVP than in a control group with MVP and suggested that the group of patients with such characteristics would have a higher risk of cerebral embolism.2

In a prospective study carried out from January 1982 to December 1986 in the Hospital "Valle de Hebrón" in Barcelona, 386 patients under 50 years old had presented with a cerebral ischaemic event. The mean (SD) age was 42.5 (7.6) years old (range: 22-93). Baseline studies included ECG, laboratory determinations, chest and skull radiology, continuous wave Doppler ultrasonography, M-mode and bidimensional echocardiography, and computerised tomography scan. Angiography studies were performed in 166 patients. The criteria for MVP were defined as those proposed by Alpert et al.4 Aortic valve prolapse was identified using the criteria proposed by Morganroth et al. and tricuspid valve prolapse was diagnosed using analogous criteria as used for MVP.

Out of all 386 patients studied, 17 had MVP. One had atrial fibrillation and was excluded from the study. The mean (SD) age of the 16 remaining patients was 41 (9.2). Seven were male and nine female. Seven patients presented with TIA and four with RIND. The other five had established stroke. Only one had previous ischaemic events and none of them was hypertensive or diabetetic. ECG was normal in 13 patients, but one had an ischaemic pattern and two showed branch block. The non-invasive study of extracranial arteries was performed in all the patients. Two had aortic valve prolapse and another had tricuspid valve prolapse in the echocardiographic study.

During a mean (SD) follow up period of 25 (16) months on treatment with antiplatelet drugs, only three patients had new ischaemic events: one had TIA, another RIND, and the third six TIs. Such patients coincided with those who presented with multiple valvular prolapses.

The evaluation of our series suggest that the incidence of MVP is not so great in the group of young adults with ischaemic stroke as has been stated, especially not in the youngest patients. However, our data reinforce the existence of high risk groups in those patients with multiple valvular prolapses.

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J Neural Neurosurg Psychiatry 1990 53: 708-709
doi: 10.1136/jnnp.53.8.708-a