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Trunkal myoclonous with spontaneous priapism and seminal ejaculation in Wilson's disease

We wish to record an uncommon clinical phenomenon seen in a patient with Wilson's disease which to our knowledge has not been previously reported.

A 25 year old unmarried male was admitted in December 1988 for the treatment of tremor and frequent jerkings of the body. He became symptomatic at the age of 14 years with postural tremor of the right hand. Frequent myoclonic jerks of the trunk appeared at the age of 20 years. During the past two years, he experienced priapism with seminal ejaculation up to three and four times per day, associated with some of the myoclonic episodes of the trunk. There were no other symptoms.

Examination revealed bilateral Kayser-Fleischer rings with postural tremors of the upper limbs, myoclonic jerkings of the limbs and trunk and a dystonic gait. No long tract signs were seen and no abnormality was detected in any other system. Examination of the genitalia did not reveal any abnormality.

The diagnosis of Wilson's disease was established by the bilateral Kayser-Fleischer rings and the low serum ceruloplasmin. The ejaculate during priapism was confirmed to be semen by the detection of spermatozoa. The patient was put on d-penicillamine which caused almost total amelioration of his symptoms over a period of six months.

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Increased amplitude of F-response in Lambert-Eaton myasthenic syndrome

Increased amplitude of the F response may be due to synchronisation of different motor units activated in this response, as in spasticity, or to reinnervated large amplitude single motor units, as in neurogenic disorders.¹ We describe one case of Lambert-Eaton myasthenic syndrome (LEMS) with F amplitude exceeding M amplitude.

A patient with a recurrence of bronchial neoplasia after surgery was admitted to our unit because of diffuse weakness which he had experienced for some weeks. Muscle weakness without amyotrophy was evident in

Figure Recording: extensor digitorum brevis. Upper traces: 4 selected traces; stimulation of peroneal nerve at the ankle. Lower trace: stimulation at the peroneal head.

the arms and legs. Facial and ocular motricity were normal and the Babinski sign absent. Sensation was not impaired. Needle electromyography (tibialis anterior, rectus femoris) was performed with concentric needle electrodes. No fibrillations were seen but trigger and delay-line techniques showed fluctuation of amplitude and morphology of individual motor units. Conduction velocities were normal for the sural nerve (47 m/s-15 μ V) and the peroneal motor conduction velocity was also normal (62 m/s). However, compound muscle action potential amplitude (CMAP) was reduced (100 μ V) and morphology fluctuated from one stimuli to another in spite of supramaximal stimuli. F waves were recorded at the extensor digitorum brevis by subcutaneous needle electrodes by stimulating the peroneal nerve at the ankle. Stimulation rate was 2/s and more than 20F responses were recorded. Most of them had an amplitude up to 25 μ V and some were greater than M amplitude (135 μ V). Minimal F latency was normal for the height (50.9 ms). Repeated stimulation (20 Hz) of the median nerve at the wrist, with recording at the abductor pollicis brevis, was consistent with the diagnosis of LEMS (increment: 532%).

F waves are produced by centrifugal discharges from motoneurons initiated by artificially produced antidromic impulses in the axon by electric stimulation. F-waves studies have proved to be useful in detecting peripheral neuropathies, especially proximal lesions.¹ Some parameters of F waves were studied, including F maximal amplitude, often expressed in per cent of M response (F%). In normal subjects, F is usually lower than 5%.²⁻³ Significant increase in the percentage of F response exists in neuropathies of various origin. In chronic spasticity, F amplitude is said to be larger¹ but some studies showed that there was no significant variation but an increased occurrence of F responses.³

Nevertheless, in all these cases, F amplitude never exceeded the value of M response, the latter representing the electrical activity of all motor units, the former only part of them. LEMS is a condition in which antibodies directed against calcium channels in the presynaptic nerve terminal membrane are responsive for a decrease in ACh release. At low rates of stimulation, CMAP is reduced in amplitude and shows a decrement in successive responses. At higher rates, usually above 20 Hz, the response becomes strongly in-

cremental.⁴ In our patient the interval between the direct stimulation (at the ankle for the peroneal nerve) and the reactivation by the F response was between 45 and 31 ms, estimated by the interval between M and F responses. These intervals correspond to shocks delivered at a frequency between 22 and 32 Hz, which corresponded to the facilitating rate and thus explains the unusually high amplitude of F response.

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Subarachnoid haemorrhage related to a lumbosacral fusion: a case report

Subarachnoid haemorrhage is a common disorder which is usually caused by the rupture of an aneurysm or arteriovenous malformation. We report an unusual case where the subarachnoid haemorrhage was caused by bleeding into a lumbar pseudomeningocele which developed after lumbosacral fusion.

A 43 year old woman had an L5/S1 discectomy and fusion with a stainless steel (Hartshill) rectangle two years previously. This was complicated by a small dural tear which was apparently repaired at operation with one suture. She made a good post operative recovery and her previous symptoms disappeared. Fourteen months later, while flyfishing, she twisted and immediately developed low back pain and a progressively more severe bilateral sciatica. On examination she had marked neck stiffness, mild pyrexia and bilateral extensor plantars with no other signs. Cerebrospinal fluid (CSF) obtained at lumbar puncture for myelography was blood stained and xanthochromic. Her clotting screen and cranial CT scan were normal. The first myelogram which was done at the referring hospital showed only a hint of a lumbosacral pseudomeningocele. The examination, however, was incomplete. A second myelogram a week later showed a more readily filling lumbosacral pseudomeningocele closely related to the Hartshill rectangle. There was a filling defect on the left side in the pseudomeningocele due to a clot (fig 1b). There were no other abnormalities.

The symptoms persisted for three weeks and the patient had surgical exploration. The cavity of the pseudomeningocele was opened and found to be filled with a blood clot on the left and heavily bloodstained CSF. The anchoring wires of the Hartshill rectangle were engaged into the posterior wall of the

Figure 1a (Left) Sagittal MRI scan of lumbosacral spine (T2 weighted image, spinecho) showing the remnant pseudomeningocele cavity with an air-fluid level in it (arrowed).

Figure 1b (Right) Lateral view of lumbar myelogram showing the Hartshill rectangle and the pseudomeningocele filling with contrast. The arrow points to the small filling defect in the upper part of the pseudomeningocele.

pseudomeningocele and the rectangle was loose. The wires were cut, removed and the rectangle was removed. A 3 mm hole was found in the posterolateral dura which was repaired with a silk suture and fascial patch. The walls of the cavity were drawn together and sutured with absorbable sutures. She made a good post operative recovery.

Two weeks later the back pain recurred. Clinically, the patient was tender over the scar but showed no other abnormal signs. An MRI scan showed that the pseudomeningocele was not ablated but contained an air fluid level (fig 1a). This raised the possibility of an infection. A needle was inserted and bloodstained fluid was obtained which did not contain white cells or bacteria. In addition the screen alpha glycoprotein level and systemic white cell count were normal.

A repeat MRI scan two weeks later showed no change. A second exploration was performed. The dural repair was found to be sound and the sac contained only lightly blood-stained fluid with no evidence of infection. The walls of the pseudomeningocele were sutured together again. Post operative recovery was uneventful. Three months later she developed further symptoms related to the original lumbosacral disc for which she had a further exploration. At operation a complete obliteration of the pseudomeningocele was evident.

The formation of a pseudomeningocele following lumbar disc surgery is a well recog-

nised complication.^{1,3} It results from a tear in the dura and arachnoid through which cerebrospinal fluid leaks into the paravertebral space. To our knowledge there have been no documented reports of a subarachnoid haemorrhage caused by bleeding into these lesions.

The back pain was probably caused by blood which was initially retained within the pseudomeningocele.² Blood then escaped into the subarachnoid space through the small dural fistula, causing a subarachnoid haemorrhage. The symptoms and signs were indicative of a subarachnoid haemorrhage which might be of spinal origin. There were no abnormalities on myelography to suggest an alternative pathology such as an arteriovenous malformation. It was felt that spinal or cranial angiography were unnecessary.

We conclude that wire sutures around a pseudomeningocele can cause a haemorrhage into it. The blood leaks through the dural tear causing a clinical subarachnoid haemorrhage. Dural tears are apt to occur during the insertion of the stainless steel wires used for anchoring a Hartshill rectangle for the purposes of fusion. It is clear from this case that careful attention should be given to repairing the leak if this occurs. MRI scans showed the lesion clearly. Even in the absence of symptoms, when a leak of this kind has been produced and repaired, an MRI scan is advisable to ensure that no further leakage has occurred.

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Spinal arteriovenous malformation unmasked during intravenous urography

Spinal myoclonus may be provoked by intravenous contrast media in patients with spinal arteriovenous malformations (AVM).^{1,2} We report a patient who developed a transient paraparesis without myoclonus following contrast injection during an intravenous urogram (IVU) who was subsequently shown to have a spinal AVM. Awareness of this unusual clinical association may alert physicians to the possibility of underlying vascular malformations in patients who present with weakness following contrast studies.

A 68 year old retired headmaster who was previously healthy developed urinary urgency and hesitancy in November 1987. These symptoms were attributed to prostatism and he attended his local hospital for an intravenous urogram. Several minutes after the intravenous injection of contrast (50 mls of Urografin 310) while lying supine on the X-ray table, his legs felt prickly and shortly after became numb below the knees. He had no involuntary movements. As he got up from the table 45 minutes later his legs collapsed beneath him and he experienced some mild low thoracic back pain. During the next 30 minutes the numbness gradually resolved and his legs became sufficiently strong to allow him to get up off the floor, reach his car and be driven home. His legs never fully returned to normal. Most mornings on rising his legs would feel weak and unsteady and he would have to sit on a stool to wash and shave. After walking 400 yards his legs would weaken, his knees would buckle and he would be forced to rest before continuing further. He became unable to play more than two holes of golf. His urinary hesitancy and urgency worsened and in addition he became constipated and impotent.

On examination the cranial nerves and upper limbs were normal. Only the left upper abdominal reflex was present. In the legs there was no muscle wasting and tone was normal. There was mild weakness (MRC grade 4+) of hip flexion and ankle dorsiflexion, more marked on the right. The knee jerks were brisk, the ankle jerk brisk on the left and depressed on the right. The plantar responses were flexor. Sensory examination was normal apart from impaired vibration sense in both feet. After walking up and down