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Spinal arteriovenous malformation unmasked during intravenous urography

Spinal myelonus 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 myelonus 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 have 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 plantar responses were flexor. Sensory examination was normal apart from impaired vibration sense in both feet. After walking up and down

Figure 1a (Left) Sagittal MRI scan of lumbosacral spine (T2 weighted image, spineco) 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 recognised complication.3,4 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 subarachnoidal haemorrhage caused by bleeding into these lesions.

The back pain was probably caused by blood which was initially retained within the pseudomeningocele.5 Blood then escaped into the subarachnoidal space through the small dural fistula, causing a subarachnoidal haemorrhage. The symptoms and signs were indicative of a subarachnoidal 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 subarachnoidal 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.
costal and first lumbar arteries. The AVM drained through a large ascending vein which passed to the posterior aspect of the perimedullary venous plexus (figure). At operation this vein was ligated and divided. Postoperatively he improved considerably and was able to play a full 18 holes of golf without symptoms. The physical signs at rest were unaltered and no longer changed following vigorous exercise.

Administration of intravenous contrast media is associated with a variety of neurological sequelae. The most common of these are seizures, usually occurring in patients with pre-existing structural brain lesions whereas focal neurological deficits without seizures are uncommon. Two reports of spinal myoclonus in patients with AVMs, in one case followed by weakness have been described. In the present case weakness occurred without involuntary movements which therefore appears to be unusual. The mechanism by which the weakness occurred in this case is speculative. Several pathophysiological studies have demonstrated toxic effects of contrast media due to osmotic oedema, alterations in the blood-brain barrier and alterations in regional blood flow either as a result of local or systemic factors. In this patient, the possibility that prolonged supine posture on an X-ray table contributed to an alteration in spinal perfusion, particularly the venous drainage of the AVM, needs to be considered since postural factors are a well recognised precipitant of symptoms in spinal AVMs. However, the usual precipitating posture is forward bending, sitting or standing rather than lying, which leads us to believe that this is an unlikely explanation in this case. Irrespective of the mechanism, the temporal relationship between the procedure and the symptoms was very striking and should raise the diagnostic suspicion of an AVM in future cases of focal neurological deficits following intravenous contrast studies.

Figure (Left) Right subcostal angiogram. (Right) Digital subtraction image of the same frame of the angiogram. There is an angiomatous malformation (arrow) on the dura in the right D12-L1 intervertebral foramen. It is supplied by the radiculo-meningeal branch and it drains through an ascending intradural vein (arrowheads) to the perimedullary venous plexus.

10 flights of stairs the left ankle jerk was lost and the vibration sense impairment extended to the left knee.

Bilateral femoral angiography and magnetic resonance imaging of the brain and spinal cord were normal. Myelography showed no evidence of canal stenosis but revealed an enlarged tortuous vessel at the T12 level. Spinal angiography demonstrated a dural AVM in the right T12/L1 intervertebral foramen supplied from the right sub-

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