SHORT COMMUNICATION

The lumbar sedimentation sign: spinal MRI findings in patients with subarachnoid haemorrhage with no demonstrable intracranial aneurysm

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ABSTRACT. We present a new MRI finding within the lumbar spine in a series of six patients admitted with CT proven subarachnoid haemorrhage (SAH) where cerebral angiography demonstrated no aneurysm and who had not had a lumbar puncture. A retrospective audit of 130 patients presenting to a regional neurosciences centre over a 13 month period with a suspected diagnosis of SAH was performed. Seven patients with proven SAH underwent MRI of the lumbar spine during the same admission. In six of these patients layering of haemorrhagic blood products was demonstrated within the lumbosacral spine. The process is analogous to the previously ubiquitous bedside erythrocyte sedimentation rate assay and has been termed the "lumbar sedimentation" sign. This finding has not previously been reported in the literature in relation to SAH. While this is a small unselected group it may provide a useful adjunct in the diagnosis of SAH.

Introduction

Subarachnoid haemorrhage (SAH) can be a difficult diagnosis in patients presenting with a headache. The consequences of a missed diagnosis for both patient and clinician can be severe [1]. Headache is a common symptom and a failure to make the diagnosis of SAH is reported in 23–53% of patients at their first attendance to hospital [2, 3]. Of all patients who present with headache 1 in 100 ultimately have a proven diagnosis of SAH [2], Migraine is 50 times more common than SAH in patients presenting to emergency departments, but conversely 12% of patients presenting with severe acute headaches have SAH [4, 5].

CT is the mainstay imaging modality for the diagnosis of SAH. It is readily available, quick, has a high sensitivity for acute blood and is relatively inexpensive. The sensitivity of CT decreases over time, depending on the volume of haemorrhage and clearance by the cerebrospinal fluid (CSF).

In the immediate aftermath of the haemorrhage the sensitivity of CT in detecting SAH can exceed 95%. The likelihood of detection falls to 85% after 5 days, 50% after 1 week, 30% after 2 weeks and to almost zero after 3 weeks [6].

Patients who have a normal CT scan, but a clinical history suggestive of SAH, should undergo lumbar puncture. In a study by Morgenstern et al [7] of 107 patients with "worst headache" 2 out of 89 CT negative patients had SAH detected by CSF analysis. In Van der Wee et al’s study [8] CT failed to diagnose SAH in 2 out of 119 cases of CSF proven SAH. CSF xanthochromia is detectable by spectrophotometry in all patients up to 2 weeks after haemorrhage and in 70% after 3 weeks [9]. Recently there has been some interest in assaying CSF ferritin levels as a further adjunct to diagnosis [10].

MRI of the head offers some promise in the diagnosis of SAH [11]. However, the use of this modality has traditionally been hampered by limited availability, long scanning time and difficulty in scanning confused or unwell patients. Although Mitchell et al [12] conclude that MRI of the brain had 94% sensitivity in acute SAH and 100% sensitivity in subacute SAH, other authors have published conflicting results. Chakeres and Bryan [13] concluded that high concentration haemorrhage was almost isointense to brain using conventional MRI sequences, giving CT a distinct advantage.

Methods

A retrospective review of SAH patients admitted to the South-West Neurosurgical Centre over 13 months (1 July 2007 to 31 July 2008) identified 130 patients. As part of their investigation or inpatient follow-up, 7 patients underwent MRI of the lumbar spine (Table 1). Four patients had lumbar spine imaging after complaining of back pain during their inpatient stay. The remaining three patients underwent the routine departmental protocol of brain and cervical spine MRI but as a result of protocol error serendipitously had additional lumbar spine imaging at the same time. All patients were imaged on a 1.5 T
magnet (Siemens Avanto, Camberley, UK). Sagittal $T_1$ and $T_2$ turbo spin echo (TSE) and axial $T_2$ TSE sequences were performed (Figure 1-2). The delay between the ictus and the time of lumbar spine imaging ranged from 1–6 days and is further described in Table 1.

In six out of seven patients a fluid level was noted at the lumbosacral junction indicating layering of blood products secondary to SAH. None of these patients had undergone a lumbar puncture. The time course of the MRI scan varied from 1 to 5 days after ictus.

Entrez-Pubmed, Cochrane database and OVID Medline literature searches were performed using the keywords: blood, subarachnoid haemorrhage, spinal canal, MRI and layering. No pre-existing reference to this observation as a marker of SAH was demonstrated.

Discussion

The circulation of CSF is complex [14]. Although there is communication between the spinal and cerebral CSF spaces, the mechanism is not completely understood. Two co-existent circulation patterns have been described: a fast flow component reliant on systolic expansion of the major arteries causing CSF to enter the upper cervical spine CSF space; and a much slower bulk flow component in which CSF ascends over the cerebral surface. Many patients experiencing a SAH will describe the initial ictus followed by stiffness migrating to the neck over 24 h. The spinal CSF acts as a capacitance vessel by absorbing these changes in pressure. The duration of recirculation of CSF within the lumbosacral region is therefore an unknown quantity.

Layering of altered blood products within the CSF at the lumbosacral junction demonstrated on MRI is, at present, only an observation in a small number of selected patients but it does raise a number of possibilities for further study. It may provide confirmation of haemorrhage in those patients who have failed to demonstrate

Table 1. Details for patients who underwent MRI of the lumbar spine

<table>
<thead>
<tr>
<th>Patient</th>
<th>Gender</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Fisher grade on first CT</th>
<th>Day of MRI post ictus</th>
<th>Lumbar SAH sign</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>41</td>
<td>Angio –ve SAH</td>
<td>3</td>
<td>4</td>
<td>–ve</td>
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<td>Angio –ve SAH</td>
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<td>+ve</td>
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<tr>
<td>4</td>
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<td>+ve</td>
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<tr>
<td>7</td>
<td>M</td>
<td>49</td>
<td>Angio –ve SAH</td>
<td>3</td>
<td>1</td>
<td>+ve</td>
</tr>
</tbody>
</table>

SAH, subarachnoid haemorrhage; –ve, negative.

Figure 1. Mid-sagittal $T_2$ weighted section (patient 7) demonstrates layering of hypointense material within the sacral canal.

Figure 2. (a) $T_1$ weighted and (b) $T_2$ weighted mid-sagittal sections (patient 2).
blood on CT, and obviate the requirement for lumbar puncture (LP). LP is performed no earlier than 12 h after ictus to allow red cell lysis and the formation of the pigments, which form xanthochromia, detectable on spectrophotometry. A prospective study would be required to determine the duration of the sign and in what proportion of CT negative, but LP positive, patients it manifests.

Some patients with a history of thunderclap headache may present beyond the 3 week diagnostic window. In this group the diagnosis of SAH is impossible to verify by CT, MRI of the brain or by the presence of CSF xanthochromia. Increasingly, in these cases CT angiography is being used to investigate for the presence or absence of an aneurysm. However, this investigation does not allow the distinction to be made between a ruptured and an unruptured aneurysm. The two are completely different entities; a recently ruptured aneurysm has a high chance of early rebleed. In the prospective cooperative aneurysm study [15] rebleeding was maximal (4%) on the first day after SAH, and then constant at a rate of 1% to 2% per day over the subsequent 4 weeks. The risk of rebleeding with conservative therapy is estimated at between 20% and 30% 1 month after haemorrhage, 30–50% 6 months after and then stabilises at a rate of approximately 3% per year. There is a 70% mortality rate for patients who rebleed [16]. An unruptured aneurysm according to the cooperative aneurysm study data [15] has a low risk of rupture that varies according to size and location. The risk of treatment of an unruptured aneurysm by either coil or surgical clip can therefore exceed the risk associated with the natural history of the disease. Persistence of the sign beyond 3 weeks may provide support for advising for or against treatment.

The absence of layering in one of the seven patients indicates the potential for false negatives. The use of alternative sequences such as gradient echo or fluid-attenuated inversion-recovery may increase the sensitivity of the test.

References