Xanthochromia revisited: a re-evaluation of lumbar puncture and CT scanning in the diagnosis of subarachnoid haemorrhage

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SUMMARY The CT and cerebrospinal fluid (CSF) findings of 100 patients with ruptured intracranial aneurysms were reviewed. Forty six percent of the 68 patients who had a lumbar puncture had blood stained CSF but with no xanthochromia. There was no blood visible on the CT scan in 20 patients: seven of these 20 had blood in their CSF, but no xanthochromia. It is concluded that it is blood stained CSF that is important in the diagnosis of subarachnoid haemorrhage (SAH), and not xanthochromia, and that a normal CT scan (EMI 1010) and the absence of xanthochromia in the CSF do not exclude a ruptured intracranial aneurysm. To diagnose SAH, it may be necessary to perform both investigations; the CT scan as the primary investigation in those patients in whom lumbar puncture is judged to be hazardous; the lumbar puncture as the secondary investigation in those patients with a normal CT scan.

Subarachnoid haemorrhage (SAH) resulting from a ruptured intracranial aneurysm is a common emergency presenting to District General Hospitals. Early diagnosis and subsequent referral for neurosurgical management are essential if mortality and morbidity are to be reduced. With the advent of CT the role of lumbar puncture as a first-line investigation in suspected SAH has been questioned. Complications of lumbar puncture are well documented but in the absence of contraindications, lumbar puncture is safe and useful. It is likely to remain the primary investigation for many patients with suspected SAH. Delayed CT scanning can lead to a false negative result; 14-6% of patients who are orientated with SAH will have a negative CT scan.

The aims of this study were to re-examine the place of lumbar puncture in the investigation of suspected SAH and to assess the significance of detection of xanthochromia in the CSF of patients with ruptured intracranial aneurysms.

Methods

We examined the case records of 100 patients who were referred to the Institute of Neurological Sciences in Glasgow, and who then had angiographically confirmed cerebral aneurysms.

The relative contributions of lumbar puncture and CT scanning towards making the diagnosis of subarachnoid haemorrhage were determined and the time interval between onset of symptoms and investigation by one or both of these two methods was recorded. In those patients who had lumbar puncture performed we also noted the laboratory report on the presence or absence of xanthochromia. This was determined in all cases by spinning down the sample in a centrifuge and examining the supernatant under direct vision. The same technique was used in all the bacteriology departments in the region. If a bacteriology report was not available, or if the sample was not sent to the laboratory, we recorded this as "no comment on xanthochromia". CT scans were performed on the EMI 1010 Scanner.

Results

Twenty patients gave a history of similar headaches occurring within the year prior to the onset of their presenting symptoms. Fourteen patients had a coma-producing subarachnoid haemorrhage; focal neurological signs were demonstrated in 29 patients. Angiography was carried out in each patient and showed the following aneurysms:
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antior communicating artery 28
posterior communicating artery 14
middle cerebral artery 11
other aneurysms 11
multiple aneurysms 36

CT scan results
CT was carried out in 99 patients with the following results:

subarachnoid blood 57
intracranial haematoma > 2 cm 6
intracranial haematoma < 2 cm 16
no haemorrhage seen 20

Lumbar puncture results
Lumbar puncture was carried out on 68 patients with the following CSF analysis reports:

normal 2
xanthochromia only 3
blood only (no xanthochromia) 31
blood and xanthochromia 25
no comment on xanthochromia (blood stained CSF) 7

Of the 31 patients with no xanthochromia but with blood in the CSF, 13 were punctured on the day of the ictus, 10 the following day, 2 on each of days 2, 3 and 4, 1 on day 5 and 1 on day 11. The absence of xanthochromia was therefore not related to the time after the haemorrhage. None of these patients showed clinical deterioration within 12 hours of lumbar puncture.

Patients with CT scan and no lumbar puncture
Thirty-two patients had a CT scan, but in view of the CT and clinical findings, did not have a lumbar puncture carried out. Their CT scans yielded the following information:

subarachnoid haemorrhage 17
intracranial haematoma > 2 cm 2
intracranial haematoma < 2 cm 9
no haemorrhage seen 4

Patients with normal CT scans
Twenty patients did not have evidence of subarachnoid haemorrhage on CT scanning; 19 of these were neurologically normal at the time of their CT scan, the 20th was mildly confused. Lumbar puncture was performed on 16 of these patients, the results being:

normal 1
xanthochromia 0
blood only (no xanthochromia) 7
blood and xanthochromia 7
no comment on xanthochromia (blood stained CSF) 1

The four patients with normal CT scans in whom lumbar puncture was not performed presented with localised 3rd nerve palsies and gave histories of acute onset of retro-orbital headaches up to 3 weeks previously. All had posterior communicating artery aneurysms.

One patient with a normal lumbar puncture and a posterior communicating artery aneurysm had a severe occipital headache 3 days prior to this investigation. She was discharged home but returned the following day with an isolated left 3rd nerve palsy. Angiography confirmed a left posterior communicating artery aneurysm.

Discussion
It has been estimated that the diagnosis of subarachnoid haemorrhage is delayed in 20% of cases, by an average of 4 days. At this late stage, CT scanning is unreliable in detecting SAH. Of the 57 patients in our study whose CT was positive, 38 were investigated within 48 hours. By contrast, three of our 20 patients with normal CT scans were scanned within 48 hours. A normal CT scan does not exclude meningitis which demands urgent but different treatment. In patients with blood-stained CSF but no xanthochromia, the possibility of a “traumatic tap” has to be considered. Nevertheless, only seven of 31 such patients had a normal CT scan. In 20, the CT showed subarachnoid blood and in the remaining four an intracranial haematoma was identified. The presence or absence of xanthochromia was not dependent upon the timing of the lumbar puncture in relation to the bleed. Others have noted that the appearance and subsequent disappearance of xanthochromia is unpredictable.

In inexperienced hands a “traumatic tap”, suggested by the absence of xanthochromia or a decrease in red cell content in consecutive sampling, occurs in up to 20% of lumbar punctures. However, in this study, 31 patients whose ruptured aneurysm was confirmed, had blood stained but xanthochromia free CSF, so that the absence of xanthochromia was not a reliable indicator of a “traumatic tap”. There is, therefore, no doubt that subarachnoid haemorrhage due to a ruptured intracranial aneurysm can occur without CSF xanthochromia.

We conclude that unless there is clouding of consciousness, papilloedema or focal neurological deficit, early lumbar puncture is essential in the investigation of a patient with features suggestive of subarachnoid haemorrhage, particularly when the CT scan is normal. In some patients blood staining, alone, without xanthochromia, may be the only evidence in the CSF of a ruptured cerebral arterial aneurysm and can provide justification for angiography when there is a good history of subarachnoid haemorrhage.
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References


18. MacDonald, Mendelow