LETTERS TO THE EDITOR

A prospective follow up of thunderclap headache mimicking subarachnoid haemorrhage

Subarachnoid haemorrhage frequently presents with the sudden onset of a severe and unusual headache, but in a number of patients presenting with this classic history the CSF is clear and CT shows no subarachnoid blood. Such headaches have been termed thunderclap headaches; their natural history is not fully known. A normal CSF examination is usually sufficient to exclude a subarachnoid haemorrhage.

Day and Raskin, however, reported a patient with an onset of headache mimicking subarachnoid haemorrhage and normal CSF who had angiography performed and normal.

This recommendation prompted Wijdicks et al to follow up patients who had presented some years previously with thunderclap headache and normal CT and CSF results. After a mean follow up of 3.5 years no patient had shown clinical evidence of subarachnoid haemorrhage, although 17% had experienced recurrent thunderclap headaches.

The study used a retrospective collection of patients from case records. This proportion of cases of the subsequent history differed from that in the case notes; furthermore a few patients had angiography at the time of presentation, which excluded an underlying aneurysm before the period of follow up. In a recent study, none of 14 thunderclap headache patients followed for at least 18 months developed subarachnoid haemorrhage; however, eight had angiography at the time of presentation, which excluded an underlying aneurysm before the period of follow up. Prospective studies in patients not subjected to angiography at the time of headache are required.

Patients were identified who presented to a district general hospital with the sudden onset of severe and unusual headaches, with a provisional diagnosis of subarachnoid haemorrhage, and in whom lumbar puncture was thus urgently required. The criteria for inclusion of a patient were: the history of a thunderclap headache; a sudden increase in pain intensity within 1 minute, and normal CSF examination within 48 hours of onset of headache. All patients were interviewed at the time of presentation. Follow up was by telephone interview.

The clinical features were compared with those obtained from records of patients presenting to a regional neurological unit in the same hospital with subarachnoid haemorrhage with no focal neurological symptoms or signs or impairment of conscious level at presentation. Differences between groups were examined using the chi-square test for differences between proportions and student t test for continuous variables.

Sixteen patients with thunderclap headache presenting over a 16 months period were identified. Eight were male. Mean age at time of presentation was 42 years (range 29-63). All presented within 24 hours of onset of headache. All described the headache as the worst headache they had experienced. Site of onset, nature, duration, precipitating and possible precipitating events are shown in the table. Three patients had experienced a similar thunderclap headache previously, one eight days before and two the previous day; in all patients this was described as being very similar to the presenting headache. Six patients had previously experienced headaches more frequently than monthly, although never like the presenting headache.

Three of these described a range and the others described non-specific headach. Co-existent disease was hypothyroidism and diabetes mellitus in individual cases. Twelve patients had a brain CT within 48 hours of onset of headache and in all cases this was normal.

All patients were followed up by a telephone interview after a mean of 24 months, with a range of 14-24, median 21. None had had a clinical subarachnoid haemorrhage or had developed focal neurological deficits in the follow up period. Four experienced a single recurrence of the thunderclap headache; at three weeks and three, 11 and 17 months. Eight patients experienced recurrent headaches monthly or more frequently; four of these described common migraine and four non-specific headaches. One of the latter experienced daily headaches and MRI was performed, which was normal. Most patients returned to work within two weeks, but two remained off work for prolonged periods; one with frequent non-specific headaches returned to work after six months, while the other had not done so at the time of follow up at 24 months; she continued to complain of generalised aching and poor concentration, and had been given a diagnosis of ‘fibromyalgia’.

The clinical features of the presenting symptoms of 37 patients presenting with subarachnoid haemorrhage were reviewed. Thunderclap headache is a benign condition in the subarachnoid group (60% versus 25%; p < 0.05). There was no significant difference between the two groups in: history of previous similar headache, precipitating factors, time of onset of headache, replication nausea, photophobia, or collapse at onset (table). Vomiting at onset was more frequent in the subarachnoid group (68% versus 43%; p < 0.05). Mean systolic and diastolic blood pressure at presentation in the subarachnoid group (177 versus 133 mm Hg p < 0.005, and 107 versus 81 mm Hg p < 0.005); for the blood pressure comparison in the subarachnoid group only those 10 cases presenting initially to the University Hospital were included, as blood pressure at initial presentation was not available for some cases referred from other hospitals.

No patient with thunderclap headache developed a subarachnoid haemorrhage or signs of an underlying cerebral aneurysm during the period of follow up. This supports the previous finding of Wijdicks et al that thunderclap headache is a benign condition.

In the case described by Day and Raskin the association of thunderclap headache with an unruptured cerebral aneurysm may be coincidental; even if they are related this association may not be attributable to routine angiography would almost certainly outweigh the benefit.

The pathophysiological mechanism underlying thunderclap headache remains uncertain. It may represent an asymptomatic form of migraine in some cases may herald the onset of more typical attacks. This association has been supported by Salloum et al who noted a history of migraine in three out of seven patients with thunderclap headache; all patients, however, had angiography at the time of presentation therefore excluding structural vascular lesions. Of the 16 patients in this study had a past history of migraine, a proportion not significantly different from a normal population, while in one case the thunderclap headache preceded the onset of common migraine. Therefore although a migrainous process may underlie some episodes of thunderclap headache, it is unlikely to account for all cases. A benign explosive onset headache is well described after exertion and during sexual activity, in the latter had been ascribed to a hyperdynamic circulation; this may account for some cases of thunderclap headache although, in this study, the majority of cases occurred while at rest. It seems likely that a number of different factors may be responsible for the headaches in different patients.

The comparison of clinical features with

Table. Comparison of patient characteristics and clinical features of the thunderclap headache and subarachnoid haemorrhage groups (*p < 0.05; **p < 0.005)

<table>
<thead>
<tr>
<th>Thunderclap headache (n = 18)</th>
<th>Subarachnoid haemorrhage (n = 37)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex (% male)</strong></td>
<td>50 (25%)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>42</td>
</tr>
<tr>
<td>Smokers</td>
<td>4 (25%)</td>
</tr>
<tr>
<td>Previous similar headache</td>
<td>3 (19%)</td>
</tr>
<tr>
<td>Precipitating event</td>
<td>3 (19%)</td>
</tr>
<tr>
<td>Coitus</td>
<td>1</td>
</tr>
<tr>
<td>Standing on toilet</td>
<td>0</td>
</tr>
<tr>
<td>Lifting</td>
<td>0</td>
</tr>
<tr>
<td>Diving/swimming</td>
<td>0</td>
</tr>
<tr>
<td>Site of onset</td>
<td></td>
</tr>
<tr>
<td>Occipital</td>
<td>8 (50%)</td>
</tr>
<tr>
<td>Frontal</td>
<td>6 (36%)</td>
</tr>
<tr>
<td>Temporal</td>
<td>3 (19%)</td>
</tr>
<tr>
<td>Parietal</td>
<td>0</td>
</tr>
<tr>
<td>Vertex</td>
<td>0</td>
</tr>
<tr>
<td>Generalised</td>
<td>1 (6%)</td>
</tr>
<tr>
<td>accompanying features</td>
<td></td>
</tr>
</tbody>
</table>
| Nausea | 14 (88%) | 23 (60%)
| Vomiting at onset | 7 (44%) | 25 (68%)
| Photophobia | 8 (50%) | 15 (41%)
| Collapse at onset | 1 (6%) | 6 (16%)
| Blood pressure on admission (mm Hg): | | |
| Systolic Mean | 133 | 177** |
| Range | 100-180 | 120-280 |
| Diastolic Mean | 81 | 107 |
| Range | 60-90 | 70-125 |
those of subarachnoid haemorrhage demonstrate that, although a history of smoking, vomiting at onset and a raised blood pressure at presentation, favour a diagnosis of subarachnoid haemorrhage, there is considerable overlap between the two conditions. Furthermore, factors known to precipitate benign headaches, such as exertion and sexual activity, may also precipitate subarachnoid haemorrhage. Therefore reliable clinical differentiation between the two conditions is not possible and in all cases of sudden onset unusual headache it is important that lumbar puncture and CT are performed; if these are normal the patient can then be reassured, and angiography is not routinely necessary.

HS MARKUS
Department of Medicine, University Hospital, Queen's Medical Centre, Nottingham, UK

Correspondence to: Dr Markus, Department of Neurology, Middlesex Hospital, Mortimer Street, London W1N 8AA, UK.


Differential diagnosis of spontaneous and traumatic intracranial haemorrhage

The introduction of computed tomography (CT) revolutionised the diagnosis of head injury, as intracranial haemorrhages and lesions of the brain can be directly visualised. The cause of lesions shown by CT, however, can be difficult to interpret especially if information on the circumstances of the accident is missing and if the patient has retrograde amnesia. We report two patients in whom lack of information resulted in considerable diagnostic difficulties.

The first patient, a woman aged 21 years, fell off her horse and was unconscious for a few minutes. She stated that the horse had shied and she had been unable to stay in the saddle. She did not have retrograde amnesia. At the time of the examination she had a visible and palpable haematoma on the back of her head with multiple scrapes. There were no focal neurological signs. The CT scan showed a haemorrhage in the right parietal region. Nine days later, a scan showed an inhomogenous structure with possible calcification (figure 1). The MRI showed a cavernous angioma in the right parietal region.

The second patient, a woman aged 51 years, suffered an accident while riding in a car on the autobahn. A tyre broke loose on a truck driving in the opposite direction and rolled into the car in which she was riding. She lost consciousness immediately and was subsequently intubated by a doctor in the emergency ambulance. At the time of admis-
sion she had a considerable scalp injury and a haematoma around the left eye. She localised to pain stimulation and did not show any focal neurological signs. Radiographs showed a left orbital roof fracture, a compression fracture of the third thoracic vertebra, and a ventral separation of the massa lateralis of the second cervical vertebra. In addition to the left orbital roof fracture, a CT scan showed extensive subarachnoid haemorrhage in the basal cisterns (figure 2), the interhemispheric cistern, and cortical subarachnoid space with intraventricular haemorrhage in the third and fourth ventricles. After intensive treatment she showed considerable improvement and was extubated after six days and slowly mobilised. Two weeks later the orbital fracture was closed surgically. A few hours later there was increasing loss of consciousness and headaches. The CT scan showed a new, fresh subarachnoid haemorrhage with ventricular haemorrhage. Angiography showed a partially aneurysm of the supracerebral part of the right internal carotid artery.

The differentiation between a spontaneous and a traumatic intracranial haemorrhage has important consequences. A spontaneous intracerebral haematoma of atypical localisation or a spontaneous subarachnoid haemorrhage require angiography to exclude or confirm a vascular lesion. Due to the great risk of recurrent haemorrhages early use of surgical source is recommended.1 In contrast, traumatic subarachnoid haemorrhage does not require any surgical or other treatment. Difficulties arise in two main circumstances: either the patient suffers an accident for unknown reasons and the CT scan shows changes which are unusual for injury (patient 1), or the exact circumstances of the accident are known and apparently confirm injury but the CT scan shows another source for the intracranial haemorrhage (patient 2). Only the neuroradiological findings (patient 1) or the clinical progress (patient 2) led to a correct diagnosis of the vascular malformation. The injury must have led to haemorrhage from the cavernous angioma and the subarachnoid aneurysm. In addition to failure to detect some consequences of injury1 or detection only after a time delay1 the CT findings may lead to a misdiagnosis. The accident may result in a rupture of a vascular malformation or spontaneous haemorrhage may cause the accident. In such cases knowledge of the circumstances of the accident and careful analysis of the neuroradiological findings may lead to a correct diagnosis.

P BERLIT
K TORNOW
Departments of Neurology and Neuroangiography, University of Heidelberg, and Deutsches Krebsforschungszentrum, Heidelberg, Germany

Correspondence to: Professor Berlit, Department of Neurology.


Intracranial haemorrhage and death after iohexol myelography

We report a case of intracranial subarachnoid haemorrhage 12 hours after lumbar iohexol (Omnipaque, NYCOMED AS, Oslo) myelography.

A 35 year old healthy man was admitted having had sciatia for two months. Lumbar myelography was performed at the L4-L5 level and was confined to the lumbar and dorsal regions. The cerebrospinal fluid (CSF) pressure and biochemical content were normal. Five ml of clear CSF was drained and 10 ml of iohexol (concentration 300 mg/ml) injected. The myelography of a prolapsed disc was completed. The biochemical analysis of the CSF sample showed no abnormality. After the procedure the patient was confined to complete bed rest with the head end of the bed elevated. He was closely monitored and remained asymptomatic for 12 hours when, after vomiting, he started behaving abnormally and talked incoherently. He was febrile and had neck stiffness; meningeal signs were suspected. A second lumbar puncture showed evidence of subarachnoid haemorrhage. He then had convulsions and lost consciousness, became breathless, and died two hours later. A complete postmortem examination was performed. The brain weighed 1400 g. The
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H S Markus

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