OCCASIONAL REVIEW

The diagnosis of subarachnoid haemorrhage

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Lumbar puncture (LP) has for a long time been the mainstay of diagnosis in patients who presented with symptoms or signs of subarachnoid haemorrhage (SAH). At present, computed tomography (CT) has replaced LP for this indication. In this review we shall outline the reasons for this change in diagnostic approach. In the first place, there are drawbacks in starting with an LP. One of these is that patients with SAH may harbour an intracerebral haematoma, even if they are fully conscious, and that withdrawal of cerebrospinal fluid (CSF) may occasionally precipitate brain shift and herniation. Another disadvantage of LP is the difficulty in distinguishing between a traumatic tap and true subarachnoid haemorrhage. Secondly, the use of CT within the first two or three days offers a wealth of information about the origin and extent of the haemorrhage, and about the presence of early complications requiring urgent treatment. An early brain scan also serves as a baseline against which future changes can be measured. The information about the location of the haemorrhage is especially valuable in patients with SAH and a negative angiogram. This is a heterogeneous group of patients which will be discussed separately. The review continues with guidelines about the interpretation of the CSF in patients with a negative CT scan, which is the only remaining indication for LP in the diagnosis of SAH. The final paragraph points out the many pitfalls in the diagnosis of rebleeding by analysis of CSF samples, which makes serial CT scanning by far the preferred method.

The dangers of lumbar puncture

The possibility of a haematoma being present in patients who present with the clinical features of SAH should be considered. In a series of 100 consecutive patients who were suspected of aneurysmal rupture, fifteen had a non-aneurysmal haematoma and eight of these were located in the cerebellum.[1] It is obvious that an LP could have done harm in these patients. In addition, a third of patients with a ruptured aneurysm harbour an intracerebral haematoma,[1-3] and even in patients without neurological signs other than neck stiffness 10 per cent may have a haematoma of at least 30 mm.[4]

Deterioration after LP is not only a theoretical possibility as shown by Duffy, who described severe clinical deterioration in seven of 55 patients with SAH who had LP, before CT scanning and within 12 hours of the bleed. Intracranial haematomas with brain shift was proven by operation or subsequent CT scanning in six of the seven patients, and it was suspected in the remaining patient who stopped breathing at the end of the procedure.[5] Rebleeding may have occurred in some of these patients.

We therefore agree with Hillman that it is advisable to perform a CT scan first in all patients who present within 72 hours of a suspected SAH, even if this requires referral to another centre.[4]

It could be argued that by first performing CT the diagnosis may be delayed and that this may be dangerous if the patient has bacterial meningitis. However, the likelihood of meningitis is extremely small in patients with an adequate history of headache or unconsciousness coming on within seconds and only rarely is the distinction more difficult, for instance, in patients who have been found in a confused state, with marked neck stiffness and moderate fever.

Ideally, patients with suspected SAH should not be admitted to a hospital without CT scanning facilities (and patients with confirmed SAH not to hospitals without a neurosurgical unit).

Traumatic tap or intracranial bleeding?

Neck stiffness following SAH takes three to 12 hours to develop—it is an unreliable test to perform on domiciliary calls. Patients arriving in casualty with a history of "thunderclap headache" a few hours earlier and with equivocal neck stiffness may either have a true SAH or a more usual type of headache with an unusual onset.[6-8] If an LP is performed and blood-stained CSF is found it is difficult to distinguish between a traumatic tap and intracranial bleeding. It is a widely held belief that this distinction can be made by collecting the CSF in three test tubes and by counting the red cells in the consecutive tubes. A decrease in red cells would indicate a traumatic tap. Indeed, a decrease in red cells occurs more often in traumatic punctures than in intracranial bleeding, but it also occurs in patients with a previous bleed.[9] Conversely, a constant number of cells can be seen with traumatic taps. In the individual case therefore no firm diagnosis can be made with this "three tube method".

The opinion of the doctor who carried out

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the LP also appears unreliable. In a study by Buruma *et al* doctors were often unable to decide for themselves whether the blood-stained CSF had resulted from the procedure or not.[9] The form of the red cells changes in the CSF, and this so-called crenation of red cells has been advocated as a means of differentiating a traumatic tap from a true haemorrhage. However, crenation of red cells occurs very soon after red cells have entered the CSF and may therefore be seen in many traumatic taps.[10] Cytology of the CSF is also of limited value. The presence of erythrophages indicates intracranial haemorrhage if an LP was not carried out earlier, but it takes some time before siderophages are demonstrable. Moreover, negative cytology does not exclude a haemorrhage.[9]

The most reliable method for distinguishing a traumatic tap from a genuine bleed is to spin down the CSF and to examine the supernatant fluid for the presence of xanthochromia. Examination of the supernatant should be done with spectrophotometry and not with the naked eye, since direct vision is a rather insensitive method. In a series of 32 CSF samples in which spectrophotometry had detected xanthochromia, this was visible with the naked eye in only half the samples.[11] The pigments that are responsible for the yellow colour of the CSF after a haemorrhage result from lysis of red cells. The average survival of red cells in the CSF is much shorter than in the circulation. This rapid haemolysis has not been fully explained. Recently, it has been suggested that red cells in the subarachnoid space lose their identity as autologous cells and this results in immune mediated haemolysis.[12]

In the classic study by Barrows *et al* the development of xanthochromia was studied by means of spectrophotometry.[13] This technique measures the light intensity in different regions of the visible spectrum (400–700 nm), after transmission of light through the coloured CSF. Three different pigments can be distinguished by their characteristic absorption bands in the spectrum: oxyhaemoglobin, methaemoglobin, and bilirubin. The wavelength where absorption occurs permits qualitative determination, whereas the height of the peak makes quantification possible.

The rate at which the pigments appear in the CSF is of great practical importance, as their presence excludes a traumatic puncture. Oxyhaemoglobin has been detected as early as two hours after the bleeding,[13] but usually it takes a few hours or more for red cells to lyse and for xanthochromia to develop. In two large series of patients with SAH it was shown that xanthochromia could be detected in all patients in whom the CSF was examined at least 12 hours after the haemorrhage.[14 15]

Extra information by CT scanning

Computed tomography not only obviates the need for a potentially hazardous LP, it offers important information other than the mere presence or absence of intracranial blood. This applies particularly if the scan is made as early as possible. On the day of SAH, intracranial blood is detected in about 95% of patients. This proportion declines to 90% after one day, 80% after five days, and 50% after one week.[3 16]

In the first place, if intracranial blood is detected, the site of the haemorrhage often indicates the likely source. In case of an intracerebral haematoma, it is often possible to distinguish primary intraparenchymal bleeding from SAH extending into the brain tissue (fig 1).[1 17] It is not always easy to determine whether a haematoma associated with aneurysm rupture is in fact intraparenchymal or whether it has only distended the subarachnoid space, usually the frontal interhemispheric or sylvian fissure.[18] However, this distinction is of limited importance for practical management, and follow up scans will usually resolve the issue. In case the extravasated blood is confined to the basal cisterns, a ruptured aneurysm can be diagnosed if the subarachnoid clots are most dense at one of the classical aneurysm sites (fig 2): the frontal interhemispheric fissure (aneurysm of the anterior communicating artery), the chiasmatic cistern on one side (aneurysm of the internal carotid artery, usually at the origin of the posterior communicating artery), or the most lateral part of the sylvian fissure (aneurysm of the middle cerebral artery).[1 19]

Bleeding from aneurysms at more unusual sites may be more difficult to recognise; aneurysmal bleeding from a posterior inferior cerebellar artery, for instance, may be easily missed by CT.[20 21] Predicting the site of a ruptured aneurysm from the CT scan is more than an intellectual game. This information is helpful in planning angiography, in particular the order and the extent of selective catheterisation, and it is vital if more than one aneurysm is found.[1 21–23] Even the uncommon event of two aneurysms rupturing at the same time can be correctly documented by CT.[24] Visualisation of the aneurysm itself, after injection of contrast, used to be the exception with CT, indicating that the aneurysm is very large, with or without calcification.[1] High-resolution CT scanning, with 1–5 mm slices, is more sensitive but not infallible.[25] Magnetic resonance imaging (MRI) is not well suited for imaging subarachnoid haemorrhage in the acute stage.[26] After several days or weeks, however, when the CT scan has become normal, MRI may detect subpial deposition of haemosiderin near the source of the haemorrhage.[21 26] In addition, MRI allows visualisation of the aneurysm itself. In two studies, a T2-weighted 2000/80 spin echo sequence, identified an aneurysm in about half the cases, by means of a signal-void area.[27 28]

Not all haemorrhage in the basal cisterns is of aneurysmal origin. A recently recognised form is the perimesencephalic haemorrhage,[29] with extravasated blood mainly or only in the interpeduncular, ambient, or quadrigeminal cisterns (fig 3a). Not only is the pattern of haemorrhage on CT different from that with aneurysmal haemorrhage, the clinical features may also differ; loss of consciousness occurs less often and a gradual onset of headache

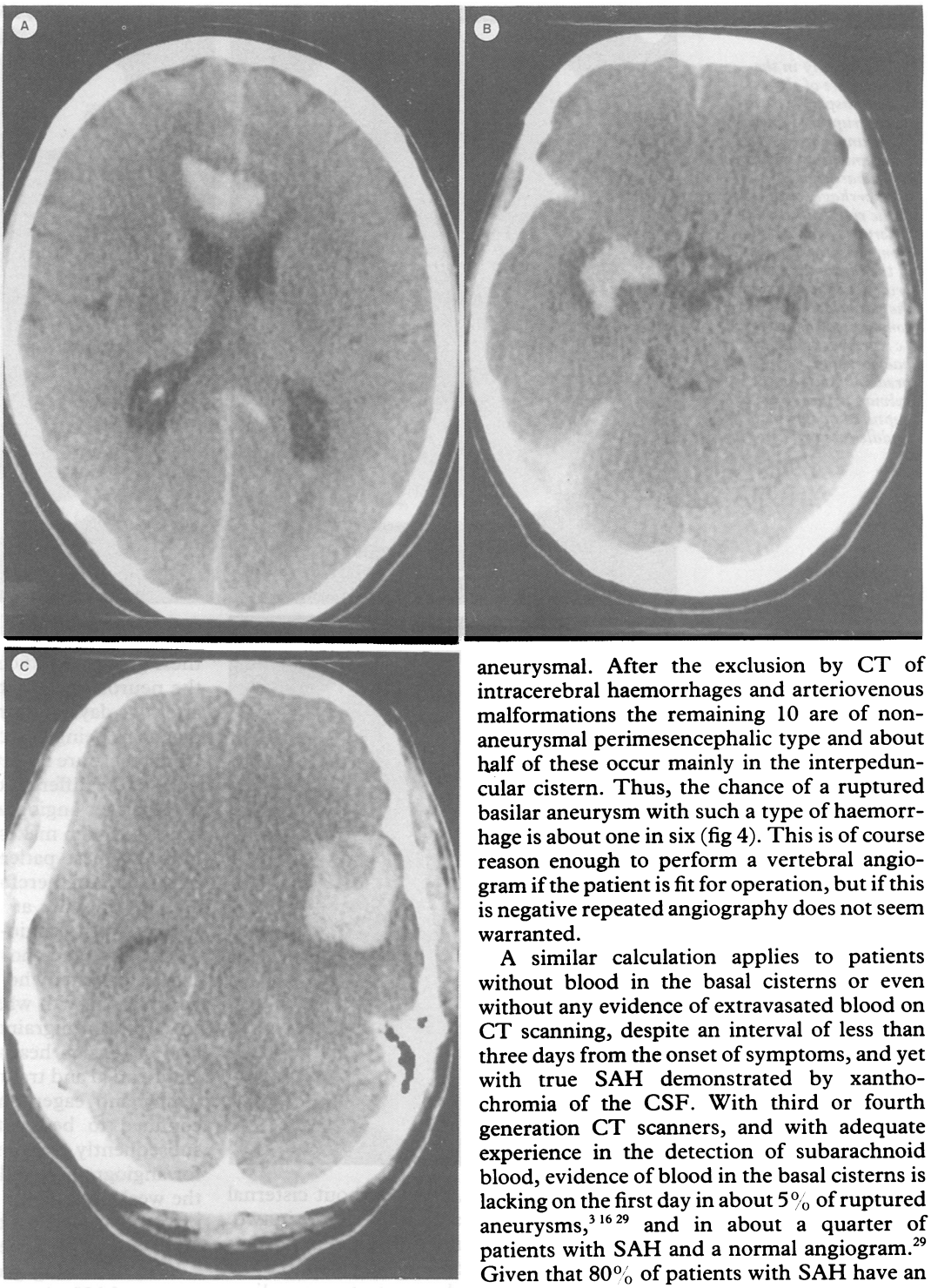

Figure 1 (a) Bifrontal haematoma, from ruptured aneurysm of the anterior communicating artery; (b) haematoma in the medial part of the temporal lobe, from ruptured aneurysm of the internal carotid artery, at the junction with the posterior communicating artery; (c) haematoma in the lateral part of the temporal lobe, from ruptured aneurysm of the middle cerebral artery (the aneurysm itself is visible as a less dense area within the haematoma).

(minutes rather than seconds) occurs more often than with aneurysms. Together with the excellent prognosis this suggests a venous or capillary source of bleeding.[29] This type of haemorrhage is not rare, occurring in about 10% of all subarachnoid haemorrhages, and in half the patients with a negative angiogram (table). It is also rather specific. Basilar aneurysms may also produce an isolated clot in the interpeduncular cistern, but these aneurysms constitute only one out of 10 ruptured aneurysms, and in turn only one in eight haemorrhages from basilar aneurysms do not extend into the chiasmatic and other basal cisterns (fig 3b).[29] For every 100 SAHs 80 are aneurysmal. After the exclusion by CT of intracerebral haemorrhages and arteriovenous malformations the remaining 10 are of non-aneurysmal perimesencephalic type and about half of these occur mainly in the interpeduncular cistern. Thus, the chance of a ruptured basilar aneurysm with such a type of haemorrhage is about one in six (fig 4). This is of course reason enough to perform a vertebral angiogram if the patient is fit for operation, but if this is negative repeated angiography does not seem warranted.

A similar calculation applies to patients without blood in the basal cisterns or even without any evidence of extravasated blood on CT scanning, despite an interval of less than three days from the onset of symptoms, and yet with true SAH demonstrated by xanthochromia of the CSF. With third or fourth generation CT scanners, and with adequate experience in the detection of subarachnoid blood, evidence of blood in the basal cisterns is lacking on the first day in about 5% of ruptured aneurysms,[3 16 29] and in about a quarter of patients with SAH and a normal angiogram.[29] Given that 80% of patients with SAH have an aneurysm, the chance of an aneurysm in

Table Patterns of bleeding on CT and results of angiography in 120 consecutive patients with subarachnoid haemorrhage. Patients with primary intracerebral haemorrhage have been excluded.[24] CT was performed within five days in all patients, and within three days in most

	Angiography	
	Aneurysm (n = 92)	*No aneurysm (n = 28)*
Pattern of haemorrhage on CT:		
Basal cisterns, compatible with aneurysm	85	7
Predominantly around midbrain	1	13
Aspecific or no evidence of haemorrhage	6	8

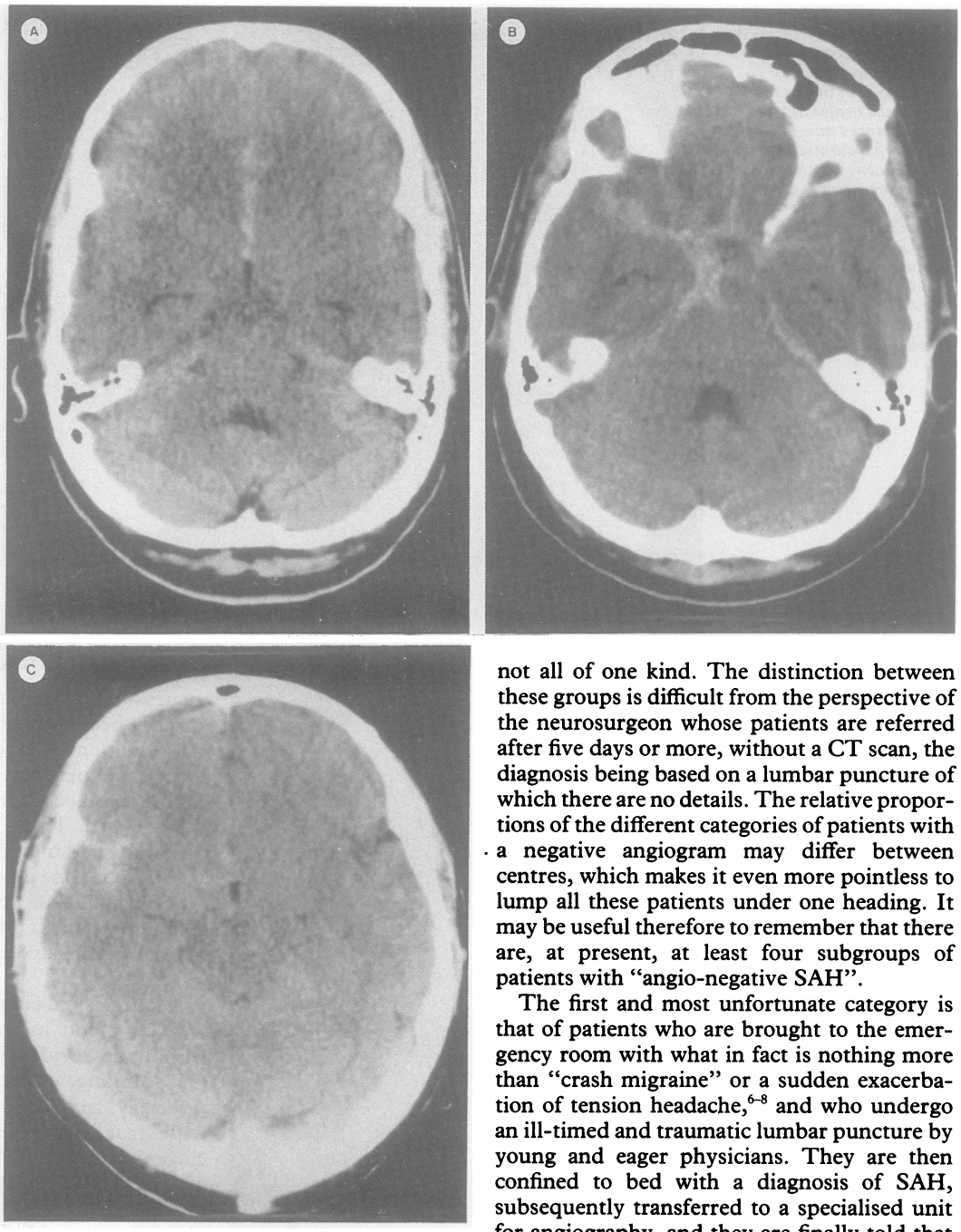

Figure 2 (a) Subarachnoid haemorrhage, predominantly in the frontal part of the interhemispheric fissure, from a ruptured aneurysm of the anterior communicating aneurysm; (b) subarachnoid haemorrhage, with centre in the right suprasellar cisterns (to reader's left), from a ruptured aneurysm of the internal carotid artery, at the junction with the posterior communicating artery; (c) subarachnoid haemorrhage, predominantly in the sylvian fissure, from a ruptured aneurysm of the middle cerebral artery.

patients with true SAH but without cisternal blood on an early CT scan is about one in two.

Early CT scanning not only gives important diagnostic information on the source of haemorrhage, it also demonstrates complications that may require urgent treatment, especially acute hydrocephalus[30] or a life-threatening haematoma.[31] Finally, the initial CT scan provides an important baseline for the diagnosis of future complications, particularly rebleeding, infarction or hydrocephalus.

"Angiogram-negative SAH": a mixed bag

The fate of patients with the diagnosis of subarachnoid haemorrhage but a negative angiogram has been the subject of a spate of publications that have reported follow up studies. We shall abstain from listing these studies, as it is abundantly clear from the preceding paragraphs that these patients are not all of one kind. The distinction between these groups is difficult from the perspective of the neurosurgeon whose patients are referred after five days or more, without a CT scan, the diagnosis being based on a lumbar puncture of which there are no details. The relative proportions of the different categories of patients with a negative angiogram may differ between centres, which makes it even more pointless to lump all these patients under one heading. It may be useful therefore to remember that there are, at present, at least four subgroups of patients with "angio-negative SAH".

The first and most unfortunate category is that of patients who are brought to the emergency room with what in fact is nothing more than "crash migraine" or a sudden exacerbation of tension headache,[6-8] and who undergo an ill-timed and traumatic lumbar puncture by young and eager physicians. They are then confined to bed with a diagnosis of SAH, subsequently transferred to a specialised unit for angiography, and they are finally told that the weak spot in one of their brain vessels has probably healed but that they would be wise to take things a bit easier in the future.

The remaining groups of patients all have a true SAH, but without a demonstrable aneurysm. Again, it is assumed that on the basis of the CT scan primary intracerebral haemorrhages consistent with either the "hypertensive" type or with arteriovenous malformations, cryptic or overt, have been ruled out before. About half of these patients show blood on CT around the midbrain, unlikely to be compatible with aneurysmal rupture (fig 3a). The localised nature of the extravasated blood, the less explosive onset of the symptoms in some cases, and the excellent prognosis all make it improbable that an occult arterial aneurysm underlies these haemorrhages.[29] The third group of patients, making up about one quarter of patients with true SAH and a normal angiogram, shows no evidence of blood in the

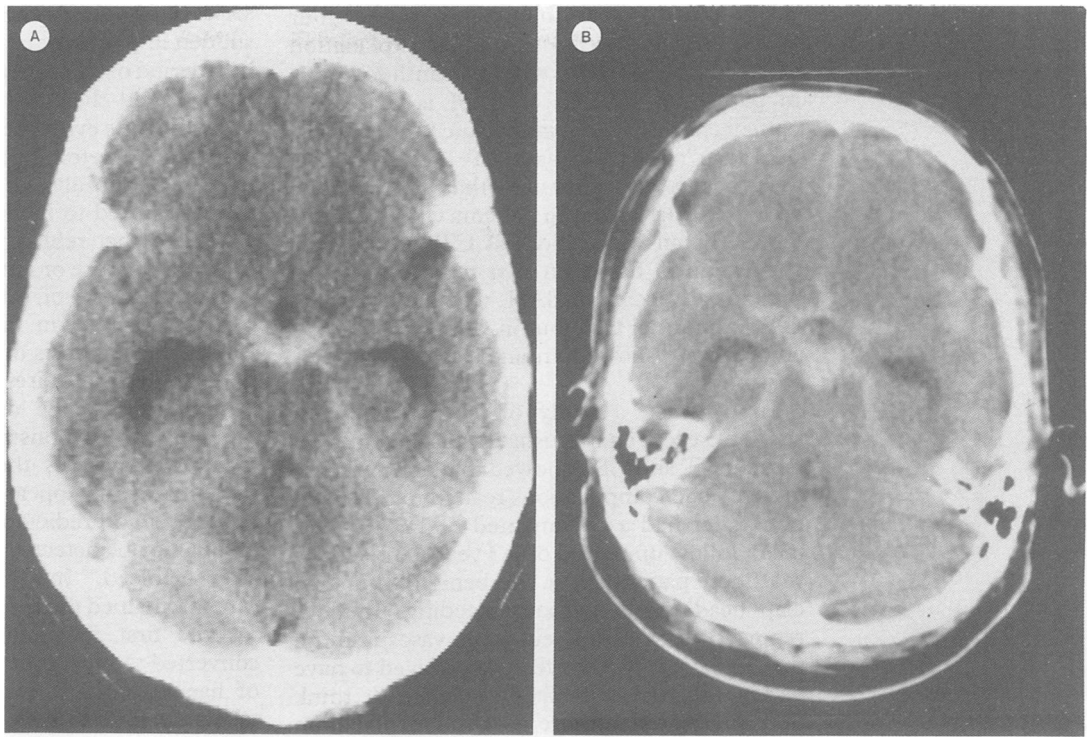

Figure 3 (a) Non-aneurysmal perimesencephalic haemorrhage, with extravasated blood predominantly in the interpeduncular fossa, and to a lesser extent in the ambient cisterns; (b) subarachnoid haemorrhage from ruptured aneurysm of the basilar artery, with extravasated blood predominantly in the interpeduncular fossa, but also more anteriorly, in the chiasmatic cistern, the Sylvian fissures, and the frontal part of the interhemispheric fissure.

basal cisterns on CT, or no blood at all.[29] In these patients the cause of bleeding is even more elusive than in the perimesencephalic group, but it may be the same. The fate of these patients has not been separately investigated, but the impression is that they do well.

The fourth and last category are patients with a pattern of bleeding on CT, that is typical of ruptured aneurysm but in whom angiography fails to demonstrate the expected aneurysm. This group constitutes the last quarter of patients with SAH and normal angiography, and about 5% of all patients with cisternal haemorrhage (table). It may be that occasional observations of local thrombosis,[32] vasospasm[33] or rebleeding[34] apply only to these patients, and that these harbour "occult" aneurysms, despite a normal angiogram. It is this category of patients in which repeated angiography seems most indicated.

Lumbar puncture after a negative CT scan

Examination of the CSF is still important if CT scanning provides no evidence of subarachnoid or other intracranial haemorrhage. Yet, if the patient is seen soon after the headache, we wish to emphasise again that it is vital to *defer lumbar puncture to at least 12 hours after the onset of symptoms* and that xanthochromia should be examined not with the naked eye but with spectrophotometry. If LP is carried out too early, xanthochromia may not have had enough time to develop and it is then impossible to distinguish between a traumatic lumbar puncture and a genuine SAH. Blood artificially introduced into the CSF will later lead to the appearance of pigments, and repeated CSF studies can never solve the problem that has been created by an LP that has been performed too soon.

CSF examination in the diagnosis of SAH is also important when the patient presents late after a suspected SAH. The 90% probability[3 16] of detecting blood in the basal cisterns on CT in the first two days after an SAH decreases rapidly thereafter. In contrast, all patients with a true SAH have CSF xanthochromia between 12 hours and two weeks after the bleeding.[14 15] Xanthochromia is still present in over 70% of patients after three weeks, and even after four weeks this proportion is still over 40%.[15] These findings contrast with another study[35] where xanthochromia was investigated with the naked eye, which explains the high proportion of patients with SAH who had no xanthochromia.

Could an SAH have occurred in patients with symptoms suspected of SAH who have blood-stained CSF but no xanthochromia? Although one study suggested that this may occur,[35] this is extremely unlikely. In a centre with approximately 80 patients with a proven SAH admitted each year, only 12 patients in a six year period had bloodstained CSF but no xanthochromia. In three of these 12 patients angiography was performed; all three were negative. All 12 patients survived without disability and were not admitted again for an

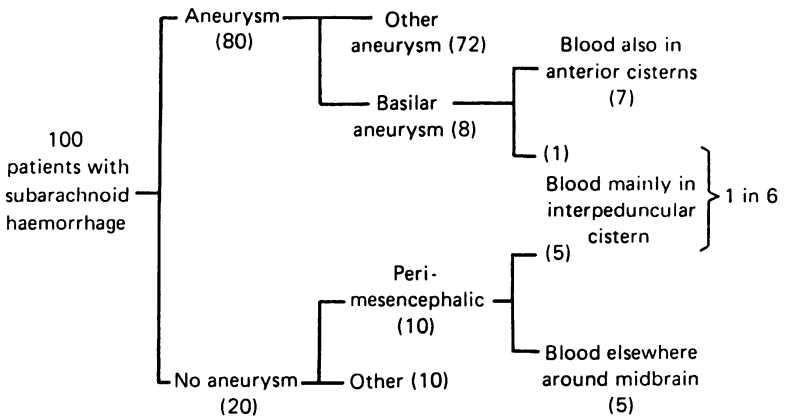

Figure 4 The chance of a ruptured aneurysm in patients with an isolated clot in the interpeduncular cistern on CT is 1 in 6.

SAH during a mean follow up period of four years.[15] Again, the most probable explanation for bloodstained CSF without xanthochromia in patients suspected of SAH is that these patients had a non-haemorrhagic "thunderclap headache" and a traumatic LP.

It has been suggested that angiography should be carried out in patients with a suspected SAH but with bloodless CT or LP. This suggestion is based on two reports of single patients suspected of SAH, who had a normal CT and normal CSF but in whom subsequent angiography showed an aneurysm with vasospasm.[36 37]

In contrast, a study of 71 patients with sudden, severe and unusual headache but with normal CT and CSF showed that none of these patients had complications related to SAH and none later had a documented SAH, during a mean follow up period of 3·3 years.[6] It is likely that these patients have had benign "thunderclap headaches", without bleeding. In both reported cases with aneurysms, vasospasm on the angiogram was the feature believed to have a causal relation with the headache. We think that migraine is a more probable explanation for both the headache and the vasospasm.[38] A small proportion of adults harbour asymptomatic aneurysms,[39] and indiscriminate angiography is bound to uncover some of these. We agree that angiography should be done in patients without blood on CT and even without CSF xanthochromia if these patients present after more than two weeks of the suspected bleeding, since not only the blood on CT but also xanthochromia of the CSF may have disappeared by that time.[15]

The diagnosis of rebleeding

If a patient with a recently ruptured aneurysm complains of a sudden increase of headache and then abruptly becomes deeply unconscious, with wide, non-reacting pupils and apnoea, there is no diagnostic problem. However, other complications may mimick rebleeding.[40] Deterioration of consciousness and focal neurological signs may have various causes, particularly delayed cerebral ischaemia. Furthermore, the clinical picture is often clouded by the previous haemorrhage. Comatose patients do not report an increase of headache, and "increased neck stiffness" is often equivocal. Finally, close observation of the patient around the clock for several weeks is not always practicable, with the result that the onset of any deterioration is not always documented. Even if a sudden loss of consciousness (within minutes) is witnessed in a patient with SAH, only two out of three turn out to have a rebleed on CT. If the fatal cases are excluded an even larger proportion of abrupt episodes, nearly 50%, was not caused by rebleeding.[41] Other causes of acute deterioration were epilepsy, ventricular fibrillation (in only one of 62 episodes of deterioration) and, surprisingly, cerebral ischaemia. Cerebral ischaemia after SAH usually presents with a gradual onset of deterioration. In contrast three patients in this series of 62 episodes mimicking a rebleed had a sudden impairment of consciousness, followed by a period of a few hours in which there was no change, and then had further gradual deterioration while evidence of infarction developed on CT.[41] Therefore, rebleeding cannot be diagnosed on purely clinical grounds. These problems used to be often dismissed with the statement that rebleeding "was confirmed by lumbar puncture or autopsy".

The diagnosis of rebleeding by analysis of CSF samples is, in fact, extremely difficult. Just as the diagnosis of a first haemorrhage by CSF analysis requires the demonstration of xanthochromia, the diagnosis of rebleeding requires the demonstration of "fresh" pigments. This raises the question of whether blood pigments appear in and disappear from the CSF in a predictable fashion. Oxyhaemoglobin may be detected in the CSF a few hours after the bleed.[13] In the early study by Barrows *et al* it remained the predominant pigment only for the first few days, after which it was converted to bilirubin, the iron-free derivative of haemoglobin.[13] This conversion does not take place in test tubes, as it is dependent upon the enzyme haem oxygenase, which is present in macrophages, the arachnoid membrane, and the choroid plexus.[42] In a later study, however, the relative concentration of oxyhaemoglobin was found to increase spontaneously after an initial decrease, and oxyhaemoglobin could still be the predominant pigment as late as three weeks after the initial haemorrhage.[43] The same fluctuating pattern was observed when the haemoglobin concentration was serially measured.[43] Therefore, measurement of neither the proportion of oxyhaemoglobin nor the haemoglobin concentration can help in the diagnosis of rebleeding. It has also been suggested that the total amount of pigments from red cell lysis, the xanthochromic index, decreases after the first few days following the haemorrhage.[44] Rebleeding would become apparent by the interruption of this normal resolution pattern. However, the xanthochromic index may continue to rise up to the sixteenth day after SAH.[43] The marked differences in the velocity of pigment clearance from the CSF have been attributed to advanced age, diabetes, and atherosclerosis.[45] Since most rebleeds occur within this period[46] a suspected rebleed cannot be confirmed merely by demonstrating an increase in xanthochromia.

These findings make the often reported statement that rebleeding was confirmed by LP barely tenable. If rebleeding is suspected and LP is repeated, the demonstration of bloodstained CSF does not prove rebleeding, as the CSF may remain blood-stained for as long as four weeks after the initial bleed.[12] The bloodstaining may also have resulted from a traumatic tap, and the distinction between a traumatic tap and recurrent bleeding is impossible to make since the CSF supernatant was already xanthochromic before the suspected rebleed. In summary, examination of the CSF can only be of help in the diagnosis of rebleeding if two earlier LPs had demonstrated a decrease of the xanthochromic index, which is

then followed by an increase.[43] It is clear that in practice this method can seldom be applied.

Apart from the unpredictable clearance of pigments the diagnosis of rebleeding by CSF analysis is also difficult because LP cannot always be performed after a rebleed. Since "fresh" xanthochromia needs time to develop, the LP has to be postponed to at least 12 hours after the rebleed. Meanwhile the patient may have died. In addition, the risk of LP in patients with an unsuspected haematoma may be even greater than after a first bleed. In a consecutive series of 17 patients with CT evidence of rebleeding, it was possible in only six patients to carry out an LP and compare the results of spectrophotometry with two previous samples (the diagnosis was confirmed in five).[43] In short, the diagnosis of rebleeding by LP is hardly possible.

Necropsy, if feasible, does not settle the matter either. The pathologist can confidently diagnose rebleeding only if the interval between the first haemorrhage and rebleeding was not too short, and that between rebleeding and death was not too long.[40] The present accepted standard in the diagnosis of rebleeding consists of the demonstration of a fresh haemorrhage on CT scanning or at necropsy, not previously demonstrated on CT scan. Such comparisons are quite sensitive, as even three or more successive haemorrhages can be reliably distinguished.[41] Nevertheless, early rebleeds cannot be diagnosed if these occur before the admission CT scan.[46]

Conclusions

1 Lumbar puncture after SAH may precipitate brain shift and herniation from an unsuspected haematoma, even in patients who are fully conscious.

2 Subarachnoid haemorrhage can be reliably diagnosed by analysis of the CSF only after examination of the supernatant fluid with a spectrophotometer. This implies that lumbar puncture should be postponed until at least 12 hours after the onset of symptoms. If lumbar puncture is performed earlier, a traumatic tap may unnecessarily stigmatise patients with benign forms of "thunderclap headache".

3 Computed tomography (CT) scanning is a safe and sensitive method for demonstrating subarachnoid blood, but should preferably be performed within three days. Important additional information provided by CT is: firstly, the demonstration of non-aneurysmal sources of haemorrhage ("hypertensive" haemorrhages, arteriovenous malformations, cryptic haemorrhages around the midbrain); secondly, the demonstration of early complications requiring urgent treatment; thirdly, evidence about the probable site of ruptured aneurysms, and fourthly, establishment of a baseline for future changes.

4 Patients with negative angiograms are not a single category. This group consists of patients without SAH but with a traumatic lumbar puncture, patients with various types of non-aneurysmal subarachnoid haemorrhage, and finally a few patients with "occult" aneurysms.

5 Lumbar puncture is indicated in SAH only if CT scanning is negative. If LP is carried out between 12 hours and two weeks after the haemorrhage, xanthochromia of the CSF can be demonstrated in all patients with SAH, provided xanthochromia is investigated by spectrophotometry. Even after three weeks xanthochromia is still present in over 70% of patients, and after four weeks in over 40%.

6 Rebleeding is the cause of acute deterioration in only two out of three cases with sudden loss of consciousness; the diagnosis cannot be confirmed by CSF analysis, except under exceptional circumstances. Necropsy is also fallible in this respect. Serial CT scanning is mandatory for a confident diagnosis of rebleeding.

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