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DISTINGUISHING TRAUMATIC LUMBAR PUNCTURE FROM TRUE SUBARACHNOID HEMORRHAGE

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☐ **Abstract—The lumbar puncture (LP) is a relatively simple diagnostic test. However, significant diagnostic ambiguity can arise when trauma from the needle causes bleeding into the subarachnoid space, especially when trying to make the diagnosis of subarachnoid hemorrhage (SAH). The purpose of this article is to assist emergency physicians in distinguishing traumatic LPs from SAH. To correctly interpret the findings of a traumatic tap, a few concepts must be understood. Timing of the LP in relation to the onset of the SAH affects the results of the cerebrospinal fluid (CFS) analysis; the typical findings will change with time. With a few caveats, xanthochromia, the yellow discoloration of the CSF resulting from hemoglobin catabolism, is often critical in making a diagnosis of SAH. A few of the most essential methods for distinguishing traumatic LP from true SAH include: the "three tube test," opening pressure, and inspection for visual xanthochromia. © 2002 Elsevier Science Inc.**

☐ **Keywords—subarachnoid hemorrhage; lumbar puncture; traumatic tap; xanthochromia**

INTRODUCTION

The lumbar puncture (LP), or spinal tap, is a common Emergency Department (ED) procedure. Cerebrospinal fluid (CSF) is withdrawn from the lumbar theca and is examined for pressure, color, and a variety of cellular and noncellular components. In the ED, LPs are conducted primarily to diagnose bleeding or infection in the central nervous system (CNS), that is, subarachnoid hemorrhage (SAH) or meningitis. The LP is a relatively simple diagnostic test. However, significant diagnostic ambiguity can arise when trauma from the needle causes bleeding into the subarachnoid space. The presence of this blood can lead to confusion about what was present in the subarachnoid space before the needle-induced trauma, also referred to as a traumatic tap. While the precise incidence of traumatic tap is unknown, some authors have estimated that it occurs in approximately 20% of cases (1). In a recent paper by Eskey et al., the frequency of traumatic taps among 1489 bedside procedures, using a cutoff of 1000 cells/mm^3, was 10.1% (2). At present, there is no consensus as to what constitutes a traumatic tap.

Whatever the true incidence of and criteria for diagnosing traumatic tap, distinguishing a traumatic tap from a SAH is critical. For one, mistaking a true SAH for a traumatic tap has significant morbidity and mortality associated with it. Roughly two-thirds of untreated SAH patients die or have serious neurologic disabilities as a consequence, many of these in the first days following initial rupture (3). On the contrary, when properly diagnosed, the outcome is largely a function of the clinical condition at presentation (4). Thus, well-appearing patients who are promptly diagnosed have an excellent outcome. On the other hand, incorrectly concluding that a traumatic tap is a true SAH will expose the patient to potentially risky procedures such as angiography. To carry out this scenario one step farther, if the angiogram

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showed an incidental aneurysm (which occurs in approximately 1–2% of the general population), unnecessary surgical intervention could be carried out (3).

Therefore, it is essential to accurately interpret the findings of an LP. The purpose of this article is to assist emergency physicians in distinguishing traumatic LPs from SAH.

When patients present with the possibility of a SAH, the standard first test is noncontrast computed tomography (CT) scan of the head. During the first 24 h after the hemorrhage, this test is approximately 95% sensitive, perhaps even higher with modern scanners and when performed in the first 12 h. With longer intervals between the onset of the headache and the CT scan, the sensitivity declines such that after 1 week it is only 50% sensitive; even for early presenting patients, authorities recommend an LP to follow up CT scans that are negative, equivocal, or technically inadequate (5–10). Some have advocated an LP-first strategy on theoretical grounds but this has not been well studied clinically (11). However, even if that strategy were to be implemented, the issue of traumatic taps would still exist.

One last point that is critical to appreciate is the concept of how timing affects the results of the CSF analysis. The typical findings will change with time. Red blood cells will be present in the first hours after rupture of an aneurysm but then disappear over days to weeks. Xanthochromia, the yellow discoloration of the CSF resulting from hemoglobin catabolism, is absent during the first hours and then, once formed, remains present for a couple of weeks. Precisely when it develops and when it disappears will depend on the amount of pigment released and on how it is measured. Therefore, in diagnosing patients with SAH, the time from onset of symptoms to the time the CT scan and the LP are performed will affect the results of both of these tests.

WHAT IS A TRAUMATIC TAP?

Traumatic taps are likely caused by puncture of the venous plexuses located dorsally and ventrally to the spinal sac or vessels that accompany the cauda equina. The sensitivity of detecting blood in the CSF via LP is considered 100%; given an estimated 20% traumatic tap incidence, the specificity is only 80% (1). The specificity can be significantly improved by establishing methods to clearly identify a traumatic tap.

However, this begs the question of how many cells it takes to be considered traumatic. In fact, there is no generally agreed upon standard. In theory, even a single RBC could be considered due to trauma. This issue is compounded by the fact that there is also no specific threshold for the number of RBCs in the CSF to be called a SAH. The diagnosis of SAH has been reported with as few as "a few hundred" cells, so the issue of distinguishing needle-induced trauma from SAH can be germane even at fairly low numbers of erythrocytes (12).

There are a variety of methods for making this distinction, some of them based on data, but many of them simply medical folklore. Table 1 lists the various criteria that have been used.

OPENING PRESSURE

The opening pressure should be routinely measured in all adults who undergo LP. This can be done most easily and accurately when the patient is in the lateral decubitus position. If the LP is performed sitting up, the patient can be laid down after the needle is in the lumbar theca. Generally, when the sitting position is used because of difficulty obtaining CSF lying down, the CSF first may be collected in the sitting position and then if a pressure measurement is desired, the patient can be gently tilted back to the lateral decubitus position to obtain a pressure reading, in this case, a proxy for the opening pressure. Special care should be taken to avoid dislodging the needle from the subarachnoid space.

It may take 2–3 min for the pressure to equilibrate in the manometer. Conventional teaching is that the legs and neck should be unbent but recent data suggest that this may be unnecessary (13). The importance of measuring the opening pressure is three-fold (7). The first two are to aid in diagnosing relatively rare conditions—pseudotumor cerebri and cerebral venous sinus thrombosis. The last is to assist in the distinction between traumatic LP and true SAH. In the days before CT scan, LP was the routine diagnostic test. In Walton's series published in the pre-CT era, 60% of the 213 patients with SAH whose CSF pressures were measured had elevated pressure, as defined by higher than 20 cm of H_2O (14). Therefore, in the correct clinical setting, finding an elevated opening pressure with bloody CSF is strongly suggestive of a true SAH.

The CSF can contain approximately 400 red blood cells (RBC) and appear clear to the naked eye. Therefore, it is imperative to measure the opening pressure on all patients, because the initial appearance of the CSF is not enough to tell if the fluid contains sufficient RBCs to be of concern.

"THREE-TUBE" TEST

Named before the four tubes that are contained in most present-day LP kits, this is a classic test to seek diminishing numbers of RBCs in the last as compared with the

Table 1. Methods for Distinguishing Traumatic LP from True SAH

CSF Finding	Traumatic LP	True SAH	Data	Comments*
Opening pressure	Normal	Elevated in 60% of cases	[14], [17], [7]	A
"3-tube test"	Initially bloody with gradual clearing	Persistently bloody	[17], [7]	B
Visual inspection for xanthochromia	Clear	Xanthochromia	[14], [17]	C
Spectrophotometry for xanthochromia	No hemoglobin breakdown products	Presence of hemoglobin breakdown products	[14], [17], [7], [10]	C
RBC count	Diminishing in progressive tubes	Persistent (NB: there is no specific threshold number)	[14], [17], [7]	B
WBC count	Proportional to peripheral blood	Proportional to peripheral blood initially, then relatively increased later	[14], [17]	B
Clot formation	Occurs rarely	Absent	[17]	D
D-dimer level	Absent	Present	[8, 23]	D
Crenated RBCs	Absent	Present	[24]	D
Erythrophages	Absent	Present	[8]	E
Repeat LP at higher interspace	Usually clear	Findings similar to those of the first tap	[17]	E

* Comments:
A. Should be routinely conducted in adults; useful when positive and also helps to make other diagnoses.
B. Should be routinely conducted; can be false positive (see text) but very helpful if RBC count is zero in last tube.
C. Should be routinely conducted; can be false positive (see text) but is the best criterion available. When measured by spectrophotometry, this is extremely sensitive but unclear how frequently there are false positives.
D. Not recommended to be conducted routinely; usually not helpful.
E. Not recommended to be conducted routinely; may be useful in some cases.

first tube. A couple of studies have tested this theory. Buruma et al. found a RBC decrease in three consecutive tubes in a group of traumatic taps ($p < 0.00012$) but were unable to demonstrate a decrease in a group of patients with true hemorrhage ($p < 0.25$) (15). These investigators lumped together several kinds of CT-diagnosed intracranial hemorrhage (contusion, intraparenchymal and subarachnoid). Of the 25 patients with true hemorrhage, only 2 had SAH. Therefore, their findings may not extrapolate to all patients with SAH. Tourtellotte and colleagues, in their study of induced traumatic taps, found consecutive tubes to always have fewer RBCs (16). If there is hemorrhagic spinal fluid, it is recommended to collect 0.5 cc in the first tube (just enough for analysis) and collect extra in the subsequent tubes so as to accentuate the difference between the first and last tubes, thereby making the distinction between traumatic tap and SAH easier. A true SAH should have generally equal discoloration in the various tubes.

Despite the inherent logic, there are some problems with this test. Consider the example of a patient whose CSF contains 5000 RBCs in the first tube and 700 in the last. This might represent a purely traumatic tap with gradual but incomplete clearing. However, it might also represent a traumatic tap in a patient with a true SAH who had 700 RBCs in the CSF pre-traumatic LP.

That said, if the count in the last tube is zero, or trends very close to zero, that is powerful evidence that the tap was traumatic. As a practical point, when confronted with this situation, it is the authors' practice to let a few extra milliliters of CSF drip out in the third tube, to make it more likely that the count in the final or fourth tube will approach zero. One other practical point is that if the CSF is initially clear (zero RBCs in tube 1) but then an impatient operator adjusts the needle position to speed the fluid recovery and the fluid turns bloody, this can be confidently ascribed to needle-induced trauma as well.

VISUAL INSPECTION FOR XANTHOCHROMIA

The definition of xanthochromia has been debated over the years. In part, this is because there are different pigments that cause xanthochromia and different methods by which to measure them—visually or by spectrophotometry. First described by Milan and Chiray in 1902, xanthochromia literally means yellow colored CSF (9). This occurs from catabolism of hemoglobin from RBCs in the CSF. The two major pigments are oxyhemoglobin and bilirubin (methemoglobin is occasionally present).

This pathophysiology of RBC lysis (see Figure 1) into its major pigment metabolites is important to understand when attempting to distinguish a traumatic tap from a SAH. Hemoglobin from lysed RBCs in the CSF can form oxyhemoglobin either in vivo or in vitro. This occurs in the first hours after the cells enter the CSF and tends to give the CSF more of a pink or pink–orange color, although at low concentrations, the distinction in color

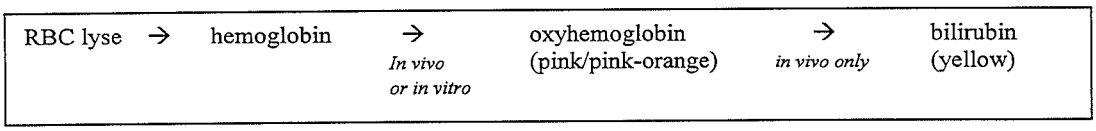

Figure 1. Pathophysiology of Xanthochromia formation.

with yellow may be difficult. Oxyhemoglobin is then metabolized to bilirubin, which imparts the classic yellow discoloration of xanthochromia. Unlike oxyhemoglobin, bilirubin is believed to be formed by an enzyme-dependent, in-vivo process. Therefore, the presence of bilirubin means that the blood was present before the LP. Macrophages and other cells in the leptomeninges contain heme oxygenases, which are induced by the presence of hemoglobin and metabolize oxyhemoglobin into bilirubin (17).

Two studies demonstrate this phenomenon. Barrows et al. added RBCs to clear CSF and demonstrated the presence of oxyhemoglobin and the lack of bilirubin even after extended incubation periods (18). Tourtellotte et al. looked at the CSF of purposeful traumatic taps (16). In six different patients, a standard LP was performed and clear CSF was obtained in the first tube and then the needle was advanced to cause a traumatic tap. It was shown that oxyhemoglobin may be present (even in the supernatant if the sample is very hemorrhagic) but bilirubin is never present in a traumatic tap; the absence of bilirubin was verified by spectrophotometry and a negative diazo reaction.

CSF from a traumatic tap may contain oxyhemoglobin but not bilirubin; CSF from true SAH will contain both pigments. However, there are a few important caveats: timing, technique of measurement of xanthochromia, and false positives.

Bilirubin requires time to develop and, therefore, xanthochromia may not be present in the first hours following rupture of the aneurysm. This was observed clinically by Walton who used the method of visual inspection (14). Among 49 patients with SAH tapped in the first 6 h after onset of symptoms, all had bloody CSF but only 10 of them had xanthochromia. Of the 40 patients tapped from between 6 h and 12 h after the ictus, all still had bloody CSF and 26 of the 40 (65%) had xanthochromic CSF. All cases of SAH presenting between 12 h and 14 days of symptom onset that were evaluated for xanthochromia actually had xanthochromic CSF. Absence of xanthochromia is not a reliable criterion to exclude SAH in the first 12 h after the ictus.

The determination of "yellow discoloration" of CSF, termed xanthochromia, is based on a subjective assessment using the naked eye. Xanthochromia is usually equated with SAH because it is assumed that bilirubin is present. Occasionally, however, discoloration of CSF can occur in other instances in which the naked eye may not be able to distinguish. If sufficient serum enters the CSF from a traumatic tap (usually in excess of 100,000 RBCs per cubic milliliter), the supernatant can appear faintly yellow. Similarly, oxyhemoglobin at low concentrations can appear a faint yellow. In both instances, xanthochromia may be because of a traumatic tap. Given that it is very difficult to visually identify true xanthochromia with certainty, accuracy can be maximized if visual inspection is standardized in the following way. The CSF should be centrifuged as soon as possible and placed in a glass tube. A second glass tube should be filled with an equal volume of water and the two samples should be compared against a pure white background, such as a piece of filter paper. This examination should take place as soon as possible after the LP is completed.

There are a few causes of xanthochromia that are not because of SAH. False positive readings can be caused by the following (also, see Table 2): jaundice, high CSF protein concentration, rifampin intake, or excess carotenoid intake (17). A minimum serum total bilirubin level of 10–15 mg/dL is usually required before there is enough bilirubin in the CSF to cause discoloration. Interestingly, though, the amount of bilirubin in the CSF does not correlate with serum levels. A CSF protein concentration of 150 mg/dL or higher is xanthochromic because of the albumin-bound bilirubin. Both rifampin and carotenoids can impart an orange discoloration to body fluids, including the CSF (17).

SPECTOPHOTOMETRY FOR XANTHOCHROMIA

Spectrophotometry is a method of quantifying the amount of a certain substance, such as bilirubin, in a liquid by detecting the optical density at the substance's

Table 2. Xanthochromia: other causes

Jaundice
Increased CSF protein
Rifampin
Excess carotenoids

specific wavelength or absorption band. There are multiple advantages to spectrophotometry. It is useful when there is too little bilirubin to be detected by the naked eye. Also, when the supernatant is pigmented, it may be unclear if the color change is because of oxyhemoglobin alone or a combination of oxyhemoglobin and bilirubin. Finally, it is an objective finding that can be officially documented. For all these reasons it has been argued that visual inspection alone for xanthochromia is not sufficient and, to avoid unnecessary risks of angiography, spectrophotometry is necessary to identify bilirubin, true xanthochromia, and diagnose SAH.

Spectrophotometry is a very sensitive test. Vermeulen et al. demonstrated xanthochromia in all 111 patients with SAH who had lumbar punctures 12 h to 2 weeks after the ictus (10). It should be noted that this study was of a population of patients with SAH, all of whom had blood visible on CT scan. Patients who require an LP to diagnose SAH are patients whose CT scans are negative; therefore, extrapolating data from patients whose CT scans were positive (and who presumably had a larger amount of hemoglobin in their CSF) to those whose CT scans are negative (with less blood) may not be valid.

For all its objectivity, spectrophotometry may not be a very specific test. In a study by Morgenstern et al., spectrophotometry was positive in 20 LPs, only 2 of which were given the diagnosis of SAH (19). The remaining 18 patients were followed for a mean of 24.4 months and, of the 16 available for contact, all were alive, well, and free of symptom recurrence and subsequent bleed. These false positive spectrophotometry results in 18 of 79 lumbar punctures make the test nonspecific with a low positive predictive value. Of note, among the 18 false positive spectrophotometry results, only 1 demonstrated visual xanthochromia.

While spectrophotometry offers several advantages as well as possible disadvantages, the reality is that this test is simply not available in the vast majority of hospitals in the United States. In one survey of over 800 hospitals, 99% of them used the visual method (20). We have surveyed over 2500 hospital laboratories in North America and found similar results (21). Even in parts of Europe, the use of visual inspection is common (22).

If xanthochromia is present by any method, this strongly suggests a true SAH and the patient should be further evaluated. If xanthochromia is absent visually in the setting of bloody CSF, then spectrophotometry should be carried out if available. In the setting of bloody fluid without xanthochromia (assessed visually) and no availability of spectrophotometry, neurosurgical consultation or noninvasive cerebrovascular imaging should be conducted.

RBC COUNT

There is no criterion standard for how many RBCs in the CSF are needed to definitively diagnose a SAH. Even the concept that zero RBCs excludes a SAH must be carefully analyzed. The CSF formula is a dynamic process and, therefore, while RBCs would be universally present in the first hours or couple of days after a hemorrhage, they gradually lyse and disappear. In Walton's study of 286 cases of SAH, blood was present in all cases presenting in the first 12 h and in 33 of 36 cases presenting between 12 h and 24 h after the ictus (14). As the number of days increased, there was an increase in the number of cases without blood. Of note, two weeks after the ictus, the majority of patients did not have blood in their CSF. SAH has been reported in a patient with "only a couple of hundred erythrocytes" in the CSF (12).

WBC COUNT

The "typical cellular reaction" that occurs with a SAH is well described by Walton. Initially, the number of white blood cells (WBC) is proportional to the number of RBCs in peripheral blood (useful approximation is 1 WBC for every 700 RBCs, assuming the absence of leukocytosis and anemia) (17). Then there is an increase in the polymorphonuclear (PMN) cells followed by an increase in large mononuclear cells occurring approximately 3 to 5 days after the ictus. The lymphocytosis may persist for weeks. This reaction is believed to be a meningeal response to the products of RBC hemolysis. In a traumatic tap of normal CSF, the WBC count will be proportional to the number of RBCs.

Thus, a disproportionately large number of white cells suggest either a SAH that has developed a cellular reaction, or alternatively, meningitis with a traumatic tap.

CLOT FORMATION

Occasionally, if enough RBCs are introduced into the CSF sample via a traumatic tap, the blood will clot. This has been shown to be the case when there are greater than 200,000 RBCs in the sample. This is a fairly specific test because the bloody CSF from a SAH should not clot presumably because of defibrination. Clot formation is an indicator of a traumatic tap but cannot rule in or rule out SAH.

D-DIMER TEST

The D-dimer assay recently has been considered as a method of differentiating SAH from traumatic taps.

There are a few studies in the literature with conflicting conclusions about the reliability of the D-dimer assay. The one study that sparked interest in the assay was by Lang et al. (23). D-dimer test was able to accurately distinguish SAH from traumatic taps and normal CSF in 40 cases; there was a positive assay in all 6 patients with SAH and there was a negative assay in all 14 patients with traumatic taps and all 20 patients with normal CSF.

The utility of the D-dimer assay was also tested by Page et al. (8). Of 12 patients with confirmed SAH, 3 had negative D-dimer values, reducing the sensitivity of the test tremendously. All false negative results were among

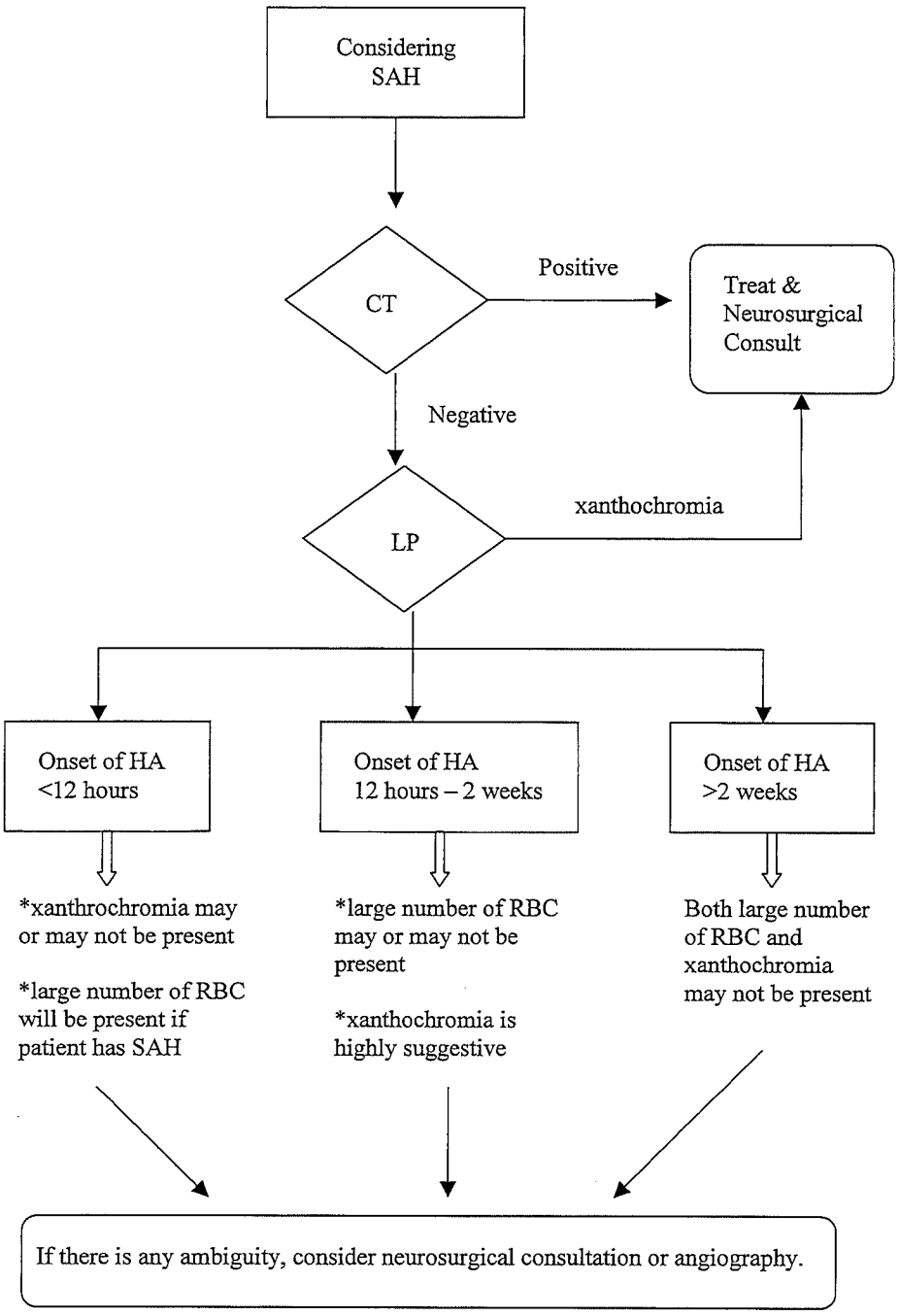

Figure 2. Algorithm for evaluation of possible SAH.

patients who presented more than 4 days after the ictus. There were many false positives as well, including one case that was believed to be a traumatic tap. Morgenstern et al. also found the D-dimer assay to be less than optimal: among the 4 positive results, 3 were false positives (19).

At this junction, the D-dimer assay cannot be considered a reliable method of differentiating SAH from traumatic taps. Further studies need to be conducted, possibly taking into account timing of the ictus and the methodology by which D-dimer measurement is performed.

OTHER TESTS

Other tests, such as microscopic examination for crenated RBCs and erythrophages, are of historic interest only (8,24).

REPEAT LP AT HIGHER INTERSPACE

When unable to distinguish a traumatic tap from possible SAH, repeating the lumbar puncture at a higher interspace is a useful test if the second tap drains clear CSF. It can be concluded that the first tap was traumatic and the patient does not have a SAH. Although the patient is subjected to the risks of a second LP, it may be worthwhile to definitely rule out a life-threatening SAH and potentially avert the higher risks of angiography.

CONCLUSION

Blood in the CSF after a lumbar puncture can present a very real diagnostic dilemma for the emergency physician. One would not want to mistake a SAH for a traumatic tap or vice versa. Unfortunately, there is no exact definition of a traumatic tap. There is no specific percentage drop or cutoff for the number of RBCs. To make matters worse, there is no criterion standard for the number of RBCs present in a SAH. Therefore, the emergency physician must carefully interpret all the data in the context of each individual case, especially with regard to timing, to distinguish a traumatic tap from SAH. A proposed algorithm to work-up a case of possible SAH is presented in Figure 2.

There are a few guiding principles that should be kept in mind. A CT scan is very sensitive in early presenting patients. Because this test is noninvasive, widely available, and inexpensive, and there is wide experience with its interpretation, it remains the first-line diagnostic test. However, if it is negative, a lumbar puncture to analyze the CSF should be performed. The CSF findings vary directly with time from the onset of the headache.

Among patients with SAH presenting within the first 12 h, all CSF samples will be bloody and roughly half will already have visually apparent xanthochromia. Of patients presenting more than 12 h but within 2 weeks of the ictus, many but not all CSF samples will have blood but almost invariably they will have xanthochromia (by visual or spectrophotometry). And among patients presenting later than 2 weeks, the CSF will likely not contain blood and may also lack xanthochromia.

All CSF samples should be assessed for xanthochromia as it is one of the best tools to distinguish traumatic lumbar puncture from SAH. Visually assessed xanthochromia is not 100% specific because it is subjective and discoloration secondary to oxyhemoglobin or other substances can be mistaken for bilirubin. While xanthochromia measured by spectrophotometry may be more accurate for the detection of bilirubin, two problems exist. The specificity of the test has been brought into question, and the reality is that the test is not available in the vast majority of hospitals in North America (19). Therefore, it is important to know what method one's hospital laboratory uses and to understand the limitations of that methodology.

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