

Pseudosubarachnoid hemorrhage in a 42-year-old male with meningitis

Gretchen Coady, MD; Melody Brewer, MD; and Patrick Mailloux, DO

Pseudosubarachnoid hemorrhage is the appearance of subarachnoid hemorrhage on CT imaging with no source of blood found on further evaluation, including lumbar puncture and autopsy. The mechanism leading to this finding is poorly understood, and clinicians should consider this finding in the right clinical setting, such as cryptococcal meningitis. We present a case of an immunocompromised patient found to have a pseudosubarachnoid hemorrhage.

Case report

A 42-year-old male, recently diagnosed with acquired immune deficiency syndrome (AIDS) and a CD-4 count of 35 cells/mm³, presented with abdominal pain, nausea, vomiting, and anorexia. During the course of his hospitalization, the patient's abdominal pain and associated symptoms improved, but he became more somnolent and fatigued. He fell twice while ambulating and after the falls reported having occipital headaches without vision changes. There was no other significant change in mental status, but a new right-eye lateral-gaze palsy was identified. The patient denied having any history of visual disturbances and, other than previous gastrointestinal symptoms with weight loss, the review of systems was negative.

The examination most notably identified an afebrile, somnolent patient with the ability to arouse when stimulated, focus on questions, and follow commands. He demonstrated a right sixth-nerve palsy manifested as a lateral-gaze defect in that eye, in conjunction with both horizontal and vertical nystagmus bilaterally. Muscle tone and strength was slightly reduced in all extremities (measuring 4/5 on the Medical Research Council's grading system).

Due to the repeated falls and possible brain trauma, a noncontrast CT scan of the head (Figs. 1A and 1B) was obtained and was concerning for a subarachnoid hemorrhage (SAH). This finding led to a neurosurgical evaluation with a subsequent CT angiogram of the brain (obtained due to the possible presence of a ruptured aneurysm). The angiogram failed to identify an aneurysm or other source of intracranial bleeding. An external ventricular drain was placed (due to suspicions of hydrocephalus) and allowed for cerebrospinal fluid sampling.

The patient's initial CSF was clear, without xanthochromia, with a white-blood-cell count (WBC) of 2 cells/mm³, red-blood-cell (RBC) count of 73 cells/mm³, and total protein of 5 mg/dL. A second tube's WBC count was 1 cell/mm³, and the RBC count 12 cells/mm³. The CSF culture grew *Cryptococcus neoformans*, with a positive cryptococcal antigen in the blood.

The findings on the CT scan were therefore felt to be consistent with a pseudosubarachnoid hemorrhage (PSAH). This is based on the fact that decreasing RBC count between the first and second tube typically indicates a traumatic tap as the source of the red blood cells. The only way to definitively rule out SAH would be to document clear CSF on a repeat lumbar puncture, which was not performed in this instance. We feel strongly, however, that the combination of a decrement in RBC count in the CSF coupled with a lack of xanthochromia, a negative CT angiogram, and a documented infectious explanation for the findings, provides strong and sufficient evidence against SAH as the mechanism for this patient's CT scan findings.

The patient's mental status continued to deteriorate, and an MRI of the brain (Figs. 2A and 2B) suggested rhombencephalitis with cerebellar swelling contributing to ef-

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The authors are all at the Baystate Medical Center, Springfield MA. Contact Dr. Mailloux at patrick.mailloux@baystatehealth.org.

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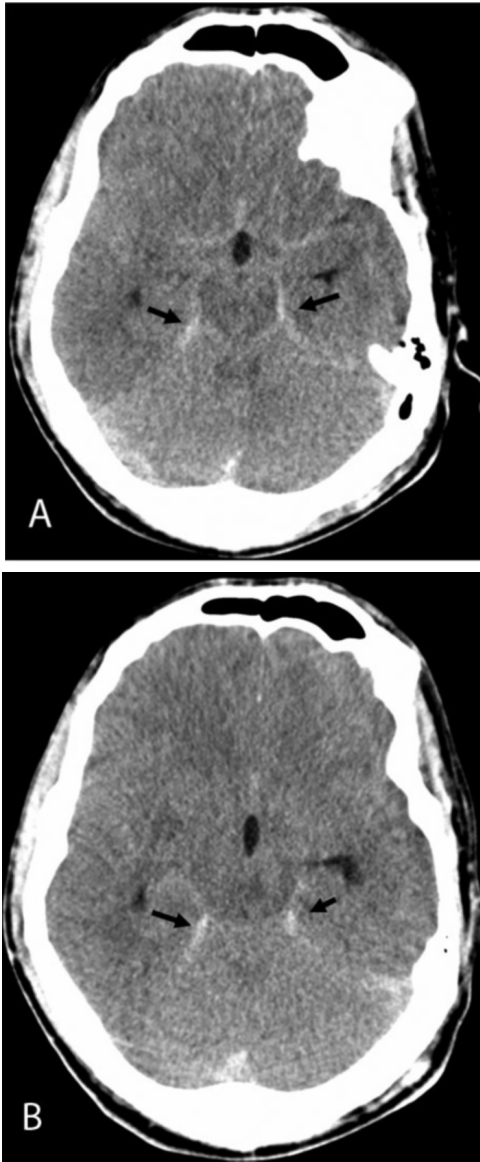


Figure 1. 42-year-old male with meningitis. Noncontrast CT of the head. The two images suggest subarachnoid hemorrhage along the cisterns with effacement of the quadrigeminal cisterns. The arrows highlight the areas thought to be collections of blood.

effacement of the fourth ventricle. His intracranial pressure continued to rise uncontrollably, leading to his death.

Discussion

SAH is defined as the appearance of SAH on CT imaging with no source of blood found on further evaluation, including lumbar puncture and autopsy. The characteristic findings on CT imaging of SAH include hyperintensity of the basilar cisterns, tentorium cerebelli, falx cerebri, sylvian fissures, and cortical sulci (1). Situations noted to cause PSAH include cases of pyogenic leptomeningitis, admini-

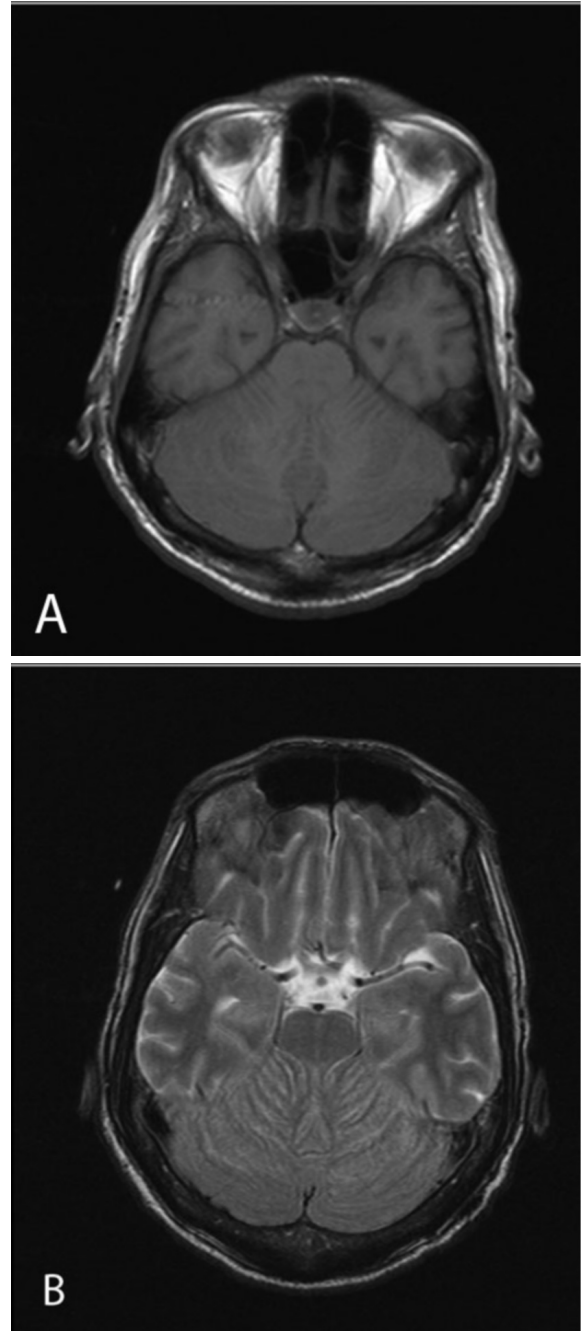


Figure 2. 42-year-old male with meningitis. MRI of the brain. Note marked swelling and enlargement of the cerebellum. The mass effect from the cerebellar swelling contributes to near-complete effacement of the fourth ventricle and effacement of the posterior fossa basal cisterns. The findings suggest rhomboencephalitis, given the history of meningitis and immunodeficiency.

stration of intrathecal contrast material, leakage of high-dose intravenous contrast medium into the subarachnoid spaces, and generalized cerebral edema (2).

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Even though many cases of PSAH exist, the pathophysiology still remains one of hypothesis. In meningitis, it is suspected that the presence of toxins leads to the breakdown of the blood-brain barrier, thus allowing proteinaceous material to leak into the subarachnoid space (2). However, this elevation in protein has only been seen to cause significant CSF hyperintensity in the most severe of meningitis cases (2). In the case of cerebral edema, it is believed that the increase in intracranial pressure and brain swelling narrows the subarachnoid spaces and displaces CSF, causing loss of the low attenuation associated with CSF (2). In addition, the increased pressure is felt to cause engorgement and dilation of the superficial venous structures, appearing to increase the blood to those areas as well as to increase flow of any contrast in the vessels (2). The final effect that cerebral edema shows is a decrease in the attenuation of the brain parenchyma, leading to the appearance of increased differentiation between the CSF and the parenchyma (3).

A previous case report determined that PSAH appearance in CNS infection was related to thick enhancement of the subarachnoid spaces, explained by substantial mucoid exudate ultimately determined on histological examination (4). This was specifically noted in a case of cryptococcal meningitis.

In all cases of PSAH, the diagnosis is one of exclusion. The main diagnosis of concern is a ruptured aneurysm leading to a SAH. An angiogram, typically via CT scan, is obtained to evaluate the cerebral vasculature; if no aneurysm is found, then further investigation may be required to differentiate the etiology of the findings. A sample of CSF, to look for red blood cells and xanthochromia, may be obtained to rule out the presence of blood and therefore confirm the diagnosis of PSAH. CSF xanthochromia is recognized as a useful test for confirming the diagnosis of SAH with a sensitivity of 93–100% (5, 6)

Hounsfield units may also prove useful in differentiating between the hyperintensity of SAH and PSAH. The hyperintensity of SAH is reported to range between 60 and 70 Hounsfield units, whereas that of PSAH is reported to range between 29 and 33 Hounsfield units (1). Our patient demonstrated a range of 42 to 50 Hounsfield units, making it difficult to demonstrate a clear delineation on the CT scan findings alone.

In the case above, the patient was diagnosed with PSAH in the setting of cryptococcal meningitis caused by *Cryptococcus neoformans*, the most common fungal CNS infection (4). *Cryptococcus* is an encapsulating yeast predominantly infecting immunocompromised individuals. When CT is completed in this setting, a normal study is the most common finding (4). In rare cases, the findings can include a dilated perivascular space, gelatinous pseudocysts, enhancing intra-axial or leptomeningeal cryptococcomas, and meningeal enhancement (4). Meningeal enhancement is seen infrequently because the inflammatory response seen with cryptococcal meningitis is usually mild (4). In three reported cases of cryptococcal meningitis, the patients were noted to have significant mucoid exudate on histological

examination, ultimately seen as thick enhancement of the subarachnoid spaces (4).

Based on the information above, it is possible to understand how cryptococcal meningitis can present as SAH on CT. This disease, as documented in prior studies, can cause cerebral edema, vascular congestion, and mucous production, leading to thick enhancement of the subarachnoid spaces (4), all of which can appear as SAH on CT imaging. It appears that the most commonly hypothesized cause of PSAH is cerebral edema when the subarachnoid space is narrowed and the superficial venous structures become engorged and dilated, in addition to the decrease in attenuation of the parenchyma during edema (1). As the pathophysiology of PSAH is further understood, various CNS infections, as well as other brain injuries, could present as such on CT imaging. It is a diagnosis physicians should be aware of as they treat and evaluate their patients with presumed SAH. It would be interesting to see further studies using the Hounsfield units to differentiate between PSAH and SAH, as it could save expense and discomfort in unnecessary studies as well as time to diagnosis and appropriate treatment.

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