Practical Pearl

Abstract
Subarachnoid hemorrhage (SAH) appears on CT as hyperdensity in the subarachnoid space. In rare circumstances a similar appearance may occur in the absence of subarachnoid blood, a finding that has been termed “pseudo-subarachnoid hemorrhage.” We describe three patients who presented with abrupt alterations in mental status in whom CT falsely suggested SAH, and we review the literature regarding this imaging finding. In contrast to prior reports, all three of our patients had a favorable outcome.

Key Words: Pseudo-subarachnoid hemorrhage; computed tomography (CT); subarachnoid hemorrhage (SAH); cerebral edema; false-positive appearance.

Introduction
Subarachnoid hemorrhage (SAH) is one of the most feared acute neurologic events. Patients typically experience an abrupt headache, frequently associated with vomiting, which may rapidly progress to coma, with or without focal neurologic signs. Accurate diagnosis of SAH is essential, because several relatively unique diagnostic tests and therapies are indicated in the management of patients with SAH that are not routinely applied to patients with other acute neurologic events.

Computed tomography (CT) of the brain is the first diagnostic imaging study performed in individuals suspected of having an SAH. Although CT is less sensitive than magnetic resonance imaging (MRI) for identifying abnormalities such as ischemic stroke, infection, or tumor, its sensitivity for SAH detection has been reported to be as high as 95–98% in patients scanned within 24 hours of symptom onset, and it is considered the study of choice for identification of SAH (1,2). SAH appears on CT as hyperdensity in the subarachnoid space, a finding generally believed to be extremely specific. However, in rare circumstances, a similar appearance may occur in the absence of blood in the subarachnoid space, a finding that has been termed “pseudo-subarachnoid hemorrhage” (3–5). We describe three patients who presented with abrupt alterations in mental status in whom CT falsely suggested SAH and review the literature regarding this imaging finding.

Patient 1
A 22-year-old previously healthy man developed abdominal pain, diarrhea, fever, and headache. One day later he became unconscious and developed generalized convulsions. He was taken to the hospital, where he was found to be unresponsive with a temperature of 102°F and a blood pressure of 80/50 mmHg. Initial CT scan showed diffuse sulcal effacement, obliterated basal cisterns, and a dense linear area in the interhemispheric fissure, suggestive of SAH (see Fig. 1). Lumbar puncture showed an opening pressure of 27 mmHg. Cerebrospinal fluid...
(CSF) examination revealed the following: leukocytes, 44/mm³, with a lymphocytic predominance; erythrocytes 3/mm³, without xanthochromia; normal protein and glucose; and negative Gram stain. Herpes simplex virus polymerase chain reaction (PCR) was negative. Brain MRI performed 1 day later did not show SAH. Cerebral angiogram was not performed. The patient was treated with acyclovir and anticonvulsants, and, by 1 week after symptom onset, he made a complete recovery. Viral serologies eventually were positive for Coxsackie virus, supporting the diagnosis of viral meningoencephalitis. Follow-up CT was normal.

**Patient 2**

A 30-year-old man with a history of mental retardation and episodic headaches presented to an outside hospital with acute severe headache and somnolence. On initial examination, he was somnolent but easily aroused, his neck was supple, and he had no focal neurologic deficits. A head CT showed diffuse sulcal effacement, obliterated basal cisterns, and hyperdensity in the interhemispheric fissure and perichiasmatic, perimesencephalic, and sylvian cisterns (see Fig. 2). Lumbar puncture showed elevated opening pressure, and CSF examination revealed the following: leukocytes 0/mm³; erythrocytes 0/mm³, without xanthochromia; and normal protein and glucose. Immediately after the spinal tap, the patient underwent cerebral angiography, which did not reveal any aneurysms or vascular malformations. During the procedure, the patient acutely deteriorated. On arrival at our institution, he was awake but quadriplegic. Brain MRI showed obliteration of the foramen magnum compatible with tonsillar herniation. Emergency suboccipital decompressive craniectomy was performed, with gradual recovery of his motor function. A follow-up head CT showed resolution of the abnormal density in the subarachnoid space. He eventually required placement of a ventriculoperitoneal shunt to control his intracranial pressure. At a follow-up visit, he had returned to his baseline neurologic status. The presumptive diagnosis was idiopathic intracranial hypertension.

**Patient 3**

A 76-year-old woman with a history of ophtalmic zoster 6 months earlier presented with nausea and vomiting and rapid deterioration in her level of consciousness over several hours. Her family reported that she had been having subtle cognitive problems for several months before this visit. On examination, the patient was afibrile, mildly hypertensive, and comatose with intact brainstem function. She withdrew all limbs to painful stimulation, and some spontaneous