ELSEVIER

Canadian Association of Radiologists Journal xx (2013) 1–7

CANADIAN ASSOCIATION OF RADIOLOGISTS JOURNAL

www.carjonline.org

Neuroradiology / Neuroradiologie

Pseudo-Subarachnoid Hemorrhage: A Potential Imaging Pitfall

Chun-Yu Lin, MD[a], Ping-Hong Lai, MD[b,c,*], Jui-Hsun Fu, MD[b], Po-Chin Wang, MD[b,c], Huay-Ben Pan, MD[b,c]

[a]*Department of Medical Imaging, Taipei Medical University Hospital, Taipei Medical University, Taipei, Taiwan*
[b]*Department of Radiology, Kaohsiung Veterans General Hospital, Kaohsiung, Taiwan*
[c]*School of Medicine, National Yang-Ming University, Taipei, Taiwan*

Key Words: Pseudo-subarachnoid hemorrhage; Subarachnoid hemorrhage mimics

Increased density of the basal cisterns and subarachnoid spaces on computed tomographies (CT) is a characteristic finding of acute subarachnoid hemorrhage (SAH) [1]. Excluding head injury, SAH leads to the performance of angiographic studies to rule out vascular lesions. Thus, recognition of the radiologic feature of pseudo-SAH on CT is important to avoid unnecessary testing and treatment delay. Several mimics of SAH have been reported: diffuse cerebral oedema due to various causes, purulent meningitis, cerebral infarction, subdural hematoma (SDH), leakage of intravenous contrast into the subarachnoid space, status epilepticus, intracranial hypotension, intrathecally administered contrast medium, and polycythemia [2–20]. This pictorial essay presents the varied manifestation of radiologic mimics of SAH.

Diffuse Cerebral Oedema

Several etiologies could result in diffuse brain oedema, including, for example, hypoxic ischemic encephalopathy, cardiopulmonary arrest, metabolic encephalopathy, trauma. The cerebral cortex becomes displaced into cerebrospinal fluid (CSF) space and veins become congested. The increased tissue attenuation immediately deep to the dura and superficially within the cerebral sulci results in hyperdensity on precontrast CT, which simulates the appearance of SAH [2–4]. In addition, the decreased attenuation of brain parenchyma due to oedema increases ‘‘contrast’’ between the parenchyma and the cistern. A mass effect of the fourth ventricle and poor grey-white matter differentiation may imply this situation (Figure 1) [2].

Purulent Meningitis

Infectious meningitis, especially tuberculous meningitis, has been reported to show increased CT attenuation over basal cistern. Toxins elaborated by the organism lead to breakdown of the brain-blood barrier. The leak of proteinaceous material due to brain-blood barrier disruption and purulent material in the subarachnoid space may lead to increased attenuation and mimic SAH [5,6]. Clinical history, CSF analysis, and enhancement of the basal cistern in postcontrast CT are clues for the diagnosis (Figure 2).

Infarction

Large cerebral or cerebellar hemispheric infarction, which may present with brain oedema, mass effect, and effacement of basal cistern, could have a pseudo-SAH appearance (Figure 3) [7,8]. Thunderclap headache might be the first sign of SAH, but sometimes it is accompanied by ischemic stroke, retroclival hematoma, and spontaneous intracranial hypotension [21]. Also a large amount of SAH may sometimes cause vasospasm and result in secondary cerebral infarction; the risk for vasospasm is usually between days 5 and 10 after SAH [9].

SDH

The pseudo-SAH appearance was secondary to the mass effect caused by the unilateral or bilateral SDHs. These large

* Address for correspondence: Ping-Hong Lai, MD, Department of Radiology, Kaohsiung Veterans General Hospital, 386 Ta-Chung First Rd, Kaohsiung, 813, Taiwan.

E-mail address: phlai@vghks.gov.tw (P.-H. Lai).

http://dx.doi.org/10.1016/j.carj.2013.07.003

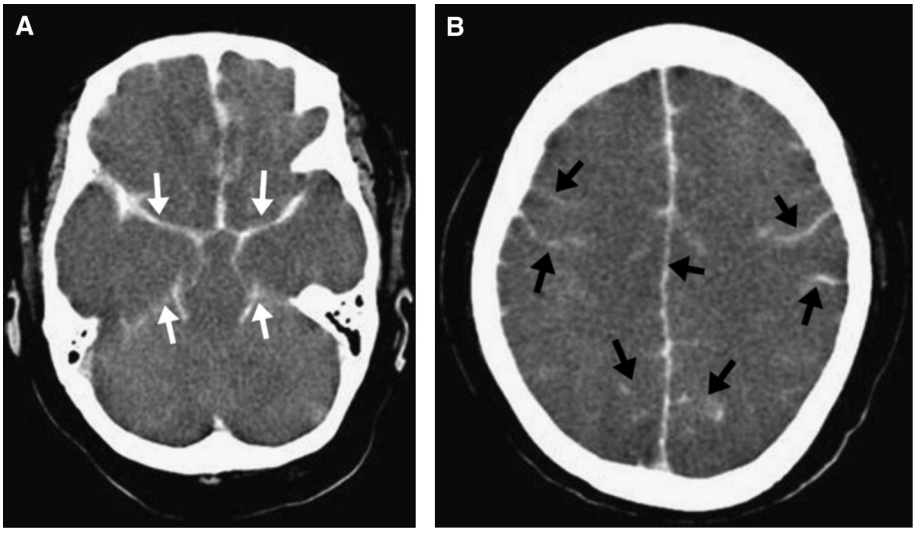

Figure 1. A 47-year-old man with hypoxic ischemic encephalopathy. (A, B) Precontrast computed tomography, showing uncal herniation, increased attenuation over basal cisterns and sylvian fissures, and tentorium (white arrows) and cerebrospinal fluid space (black arrows). Diffuse low attenuation over the bilateral hemisphere with poor grey-white matter differentiation is characteristic for hypoxic ischemic encephalopathy.

SDHs compressed the cerebral hemispheres and caused diffuse effacement of the cortical sulci, with a decrease in the size of CSF space (Figure 4). Hydrocephalus would be expected as a result of obstruction of cerebral fluid reabsorption in true SAH as opposed to slit-shaped ventricles as a result of mass effect in pseudo-SAH [10–12]. After evacuation of the SDH, the CSF space returned to its normal size with the disappearance of the hyperdensity [11].

Contrast Extravasation

Contrast neurotoxicity seems to be related to the chemical or ionic properties and its hyperosmolarity, lipid solubility, and viscosity. Contrast extravasation in the brain has been reported as an uncommon complication of angiography and accompanied with transient cortical blindness, confusion, amnesia, seizure, or neurologic deficits [13,14]. Besides angiography, including coronary, carotid, spinal, renal, and aortography, an interventional procedure such as transjugular intrahepatic portosystemic shunt also can cause the pseudo-SAH appearance (Figure 5). The reduced clearance contrast in renal failure and overdose contrast usage may be clinical factors [13]. The majority of patients recovers without any neurologic deficit, and follow-up CT findings return to normal. In such situations, clinical examination and serial neurologic imaging studies can effectively help in the differential diagnosis.

Status Epilepticus

Transient or permanent changes may occur in the brain after status epilepticus, including focal gyral swelling,

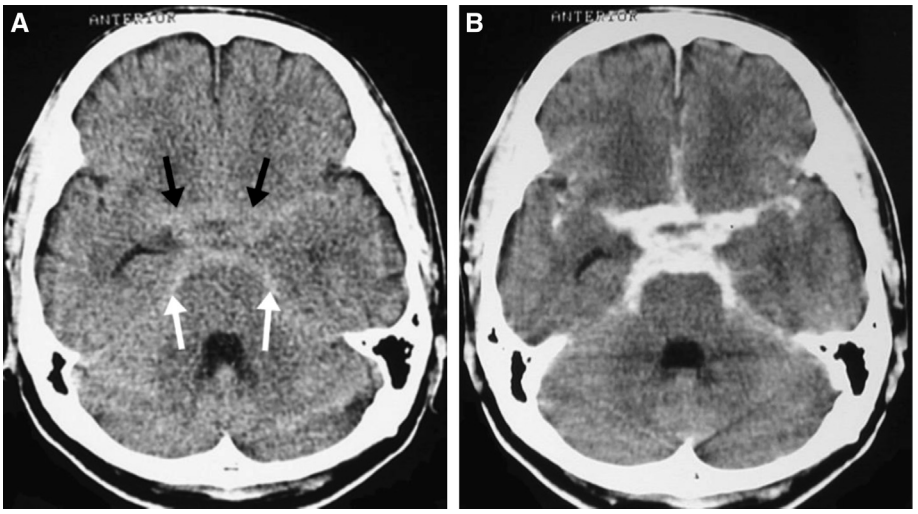

Figure 2. A 55-year-old man with tuberculous meningitis. (A) Precontrast computed tomography (CT), showing increased attenuation over basal cisterns (black arrows) and along the tentorium (white arrows). (B) Postcontrast CT, showing vivid enhancement in the same regions.