

A Case Report of Pseudo-Subarachnoid Hemorrhage

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ABSTRACT

Background: The authors report an unusual case of the radiological appearance of diffuse subarachnoid hemorrhage on brain computed tomographic (CT) scan in a patient with post-resuscitation anoxic encephalopathy.

Clinical presentation: A young man with chronic renal failure collapsed unconscious while starting hemodialysis and was revived after resuscitation for almost one hour. He was shifted to intensive care unit and subsequent CT scan after 3 days showed a picture compatible with diffuse subarachnoid hemorrhage. The subsequent lumbar puncture showed no evidence of subarachnoid hemorrhage while the attenuation at the basal cistern on CT brain was consistent with pseudosubarachnoid hemorrhage. A four vessel angiography done subsequently confirmed anoxic encephalopathy.

Conclusion: Anoxic encephalopathy can mimic diffuse subarachnoid hemorrhage on CT.

INTRODUCTION

The computed tomographic (CT) finding of pseudosubarachnoid hemorrhage mimics that of subarachnoid hemorrhage (SAH). In brain swelling due to acute hypoxic anoxic encephalopathy, the increased attenuation in the basal cisterns and subarachnoid spaces on CT scans is similar to findings of acute SAH and these SAH-like findings were first described by Spiegel et al in 1986 (1). They reported that 10 patients with marked brain edema associated with a brain tumor or cerebral infarction show-

ed SAH-like appearance along the interhemispheric fissure and tentorium cerebelli on CT without presence of SAH on autopsy. In 1998, on reviewing head CT examinations of 100 comatose patients with brain edema, Avrahami et al found SAH-like findings along the cisterns and sulci in all of them and concluded that a CT diagnosis of SAH was unlikely. They proposed the term "pseudo-subarachnoid hemorrhage" (pseudo-SAH) for this phenomenon (2).

The authors attempt to report a similar case report of pseudo-SAH in a young man who presented with prolonged post cardiopulmo-

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nary resuscitation and was eventually found to have anoxic encephalopathy. □

CASE REPORT

A young man with chronic renal failure collapsed unconscious while starting hemodialysis and was revived after resuscitation for almost one hour. He also suffered some nasal trauma on falling down with some bleeding from nose. He was shifted to intensive care unit on mechanical ventilatory support. In light of low Glasgow coma scale of 3 and history of fall with bleeding from nose a CT scan was done to rule out head trauma after 1 day as patient was hemodynamically stabilized. The CT brain showed severe brain edema and loss of sulci with hyper attenuated cisterns and cerebral sulci, a picture compatible with diffuse SAH (Figure 1). He was put on continuous venovenous hemofiltration for his renal failure and his metabolic abnormalities were corrected in intensive care unit. He remained in a comatose state and no neurological response. The subsequent lumbar puncture showed no evidence of SAH while the attenuation co-efficient at the basal cistern on CT brain was 27-43 HU (Hounsfield unit) with a Mean of 32 to 37 HU consistent with pseudo subarachnoid hemorrhage. A four vessel angiography done subsequently confirmed anoxic encephalopathy and brain death. His electroencephalogram and brain-

stem neurological signs were also consistent with brain death. □

DISCUSSION

Subarachnoid hemorrhage (SAH) is a medical emergency and its early diagnosis is extremely important. In brain computerized tomography (CT) it appears with an increased density in the basal cisterns and the subarachnoid space. A few neurological diseases like hypoxic encephalopathy, hyperperfusion radical encephalopathy, extensive infarctions, viral meningoencephalitis, purulent meningitis, bilateral subdural hematoma and idiopathic intracranial hypertension have been described in medical literature that rarely mimic a tomographic pattern typical of SAH, without clinical or pathological evidence of SAH; a condition termed as pseudosubarachnoid hemorrhage (3-7). The mechanism for the development of a pseudo-SAH has not been fully understood. Some authors have suggested that severe brain edema compresses the dural sinuses, compromising the venous drainage from the brain and resulting in engorgement of the superficial veins, which stand out against the edematous low-attenuated brain parenchyma, mimicking an SAH (1). However Given et al proposed a similar mechanism but included narrowing or disappearance of hypoattenuated CSF space as a factor generating a pseudo-SAH (2).

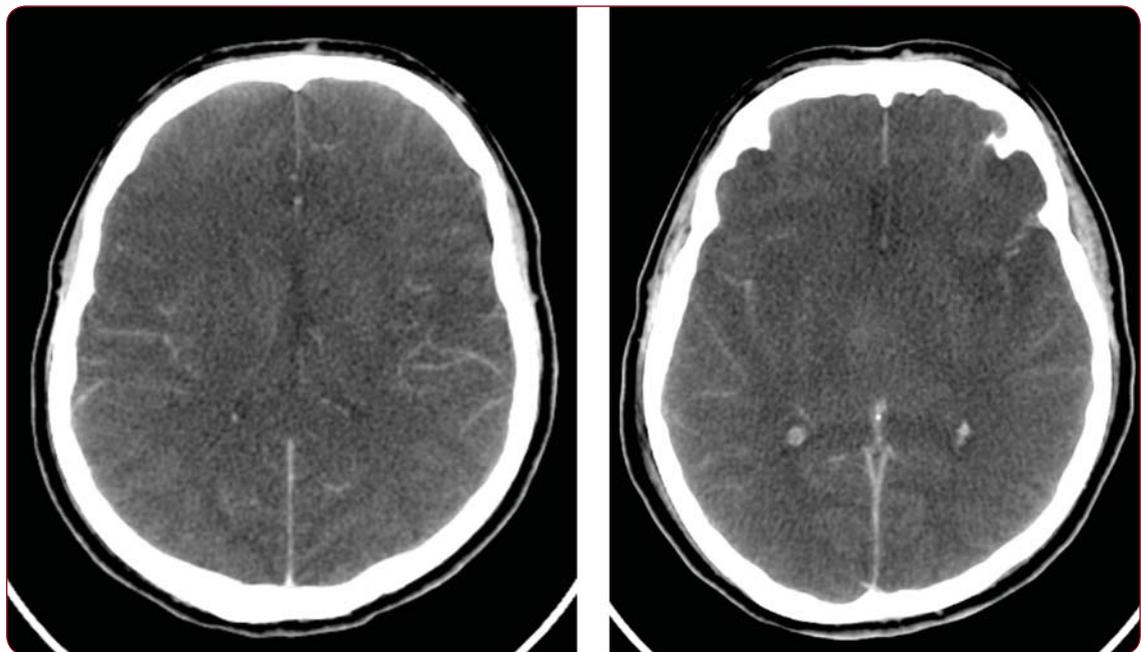


FIGURE 1. CT Brain showing subarachnoid hemorrhage like appearance

A pseudo-SAH finding is a CT pseudo lesion that shows SAH like findings, in which the cisterns and cerebral sulci appear hyperattenuated relative to the brain parenchyma. This is a synergistic result of distention of the superficial vessels arising from elevated intracranial pressure and severe brain edema manifesting as hypoattenuated parenchyma (8). Following resuscitation, pseudo-SAH may develop within 3 days after the onset of cardiopulmonary arrest in approximately 20% of patients (9). In an ana-

lysis of 7 cases of pseudo-SAH confirmed by necropsy or lumbar puncture study, the attenuation coefficient in the basal cisterns was 21 to 44 HU (mean, 29-33 HU), which was clearly lower than in all control cases consisting of patients with confirmed SAH having 60-70 HU (2). Our patient also had attenuation co-efficient in the basal cisterns of 32-37 HU (Mean) which is consistent with diagnosis of pseudo-SAH on CT imaging.



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