



BMH Med. J. 2024;11(4):58-60. **Brief Review**

Pseudo - Subarachnoid Hemorrhage: Diagnostic Challenges and Clinical Implications

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Abstract

Pseudo-subarachnoid hemorrhage (pseudo-SAH) is a radiologic phenomenon where imaging characteristics mimic those of true subarachnoid hemorrhage (SAH), despite the absence of actual hemorrhage. This paper aims to elucidate the underlying mechanisms, diagnostic challenges, and clinical implications of pseudo-SAH, with a particular focus on differentiating it from true SAH using imaging modalities and clinical context.

Introduction

Subarachnoid hemorrhage (SAH) is a critical condition with high morbidity and mortality, necessitating prompt and accurate diagnosis. However, certain conditions can mimic the appearance of SAH on imaging studies, leading to potential misdiagnosis and inappropriate management. Pseudo-SAH is one such phenomenon, often seen in patients with diffuse cerebral edema, intracranial hypotension, or following cardiopulmonary arrest. This paper reviews the etiology, diagnostic criteria, and implications of pseudo-SAH, highlighting the importance of accurate differentiation from true SAH [1,2].

Etiology and Pathophysiology

Pseudo-SAH can arise from various conditions, including:

Diffuse Cerebral Edema: Severe brain swelling can increase the attenuation of the cerebrospinal fluid (CSF) spaces on CT, mimicking SAH [3].

Intracranial Hypotension: Reduction in CSF volume can lead to dural venous sinus engorgement and increased attenuation of the basal cisterns.

Post-Cardiac Arrest: Global cerebral ischemia following cardiac arrest can cause brain swelling and increased attenuation of the CSF spaces.

Meningitis and Encephalitis: Severe infections can lead to brain swelling and increased protein content in the CSF, leading to higher attenuation on imaging.

Diagnostic Imaging

Computed Tomography (CT)

CT imaging is the first-line modality for diagnosing SAH. In pseudo-SAH, the increased attenuation in the basal cisterns and cortical sulci can mimic true hemorrhage. Key features include [4]:

Basal Cisterns and Cortical Sulci: Hyperattenuation in these areas without the presence of true blood.

Brain Parenchyma: Diffuse cerebral edema may be present, with loss of gray-white matter differentiation.

Magnetic Resonance Imaging (MRI)

MRI can provide additional clues to differentiate pseudo-SAH from true SAH:

FLAIR Sequence: May show hyperintense signal in the basal cisterns due to edema rather than blood.

Diffusion-Weighted Imaging (DWI): Can reveal areas of restricted diffusion indicative of cytotoxic edema.

Susceptibility-Weighted Imaging (SWI): Helps in identifying true blood products through susceptibility effects, which are absent in pseudo-SAH.

Angiography

Digital subtraction angiography (DSA) may be performed to rule out vascular causes of true SAH, such as aneurysms or arteriovenous malformations. In pseudo-SAH, DSA typically shows no vascular abnormalities.

Clinical Presentation

The clinical presentation of patients with pseudo-SAH varies based on the underlying cause:

Diffuse Cerebral Edema: Patients may present with altered mental status, coma, or signs of increased intracranial pressure [5].

Intracranial Hypotension: Symptoms include orthostatic headaches, nausea, and vomiting.

Post-Cardiac Arrest: Patients typically present with global hypoxic-ischemic encephalopathy and coma.

Meningitis/Encephalitis: Symptoms include fever, headache, neck stiffness, and altered mental status.

Management

The management of pseudo-SAH focuses on addressing the underlying cause:

Cerebral Edema: Osmotherapy, hyperventilation, and surgical decompression may be considered.

Intracranial Hypotension: Epidural blood patch and bed rest are common treatments.

Infectious Causes: Appropriate antibiotic or antiviral therapy is essential.

Post-Cardiac Arrest: Neuroprotective measures and supportive care are crucial.

Conclusion

Pseudo-SAH represents a diagnostic challenge due to its imaging resemblance to true SAH. Accurate differentiation is essential to avoid unnecessary interventions and to guide appropriate management. Advanced imaging techniques and careful clinical correlation are pivotal in distinguishing pseudo-SAH from true hemorrhage. Understanding the underlying etiologies and pathophysiology can aid clinicians in making informed decisions and improving patient outcomes.

References

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