A CLASSIC CASE OF PSEUDO SUBARACHNOID HAEMORRHAGE (SAH) DUE TO SECONDARY POLYCYTHEMIA

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ABSTRACT Presentation of symptoms and clinical history always plays a significant role in differentiating true acute subarachnoid haemorrhage (SAH) from pseudo-SAH. We report a 25-year-old man presenting with recurrent headaches and attending a pulmonologist for recurrent chest infections. Non-Enhanced CT (NECT) of the brain was done, which showed extra-axial sulcal hyper density along cerebral arteries and hyperdense venous sinuses. MR imaging confirmed the linear hypointensity along vessels on susceptibility-weighted images (SWI), suggesting an increased concentration of paramagnetic deoxyhemoglobin within cerebral vessels; findings suggested polycythemia mimicking SAH. Pseudo SAH is a potential mimic of SAH. Awareness of various manifestations of pseudo-SAH is essential for proper treatment, avoiding unnecessary investigative modalities like angiographic studies, invasive procedures like Lumbar puncture, etc. Polycythemia presenting as pseudo subarachnoid haemorrhage is a rare differential. Clinical history and MR imaging help to differentiate SAH from pseudo-SAH.

KEYWORDS Pseudo subarachnoid haemorrhage, MRI-SWI sequence, Hypoxia, Haematocrit.

Introduction
Increased attenuation due to haemorrhage within basal cisterns and the subarachnoid spaces on computed tomography (CT) is the characteristic feature of acute subarachnoid haemorrhage (SAH). 1 Apparently, increased attenuation within the basal cisterns and subarachnoid spaces simulating actual subarachnoid haemorrhage is defined as pseudo subarachnoid haemorrhage (2). Several pseudo subarachnoid haemorrhage mimics are cerebral oedema, purulent meningitis, subdural hematoma (SDH), and leakage of intravenous contrast into subarachnoid space (SA space), status epilepticus, intracranial hypotension, intrathecal administration of contrast medium, and polycythemia. Presentation of symptoms and clinical history always plays a significant role in differentiating true SAH from pseudo-SAH.

In the diagnosis of SAH, Non-Enhanced CT is known to have a high sensitivity and an even higher specificity (3). The false-positive CT finding called pseudo-SAH is rare. We present a case of pseudo-SAH, which had its origin in chronic hypoxemia. The most common causes of polycythemia are myeloproliferative conditions (Polycythemia Vera); secondary causes include hypoxemic lung disease, Erythropoietin-producing malignancies, congenital cyanotic heart diseases, living at high altitude, or chronic exposure to carbon monoxide (4).

Case report
A 25-year young male presented to the clinic with recurrent headaches attending a pulmonologist for recurrent chest infections. NECT was advised. Imaging findings showed extra-axial sulcal and cisternal hyper density along the cerebral arteries (Fig 1) and hyper-dense venous sinuses (Fig 1). MRI was advised for further evaluation. MRI of the brain was done, which confirmed linear hypointensity along the vessels on SWI (Fig 2) images suggesting an increased concentration of paramagnetic deoxyhemoglobin within cerebral vessels and prominent cerebral veins. No evidence of any hemosiderin deposition in the sulcal and cisternal spaces. Imaging findings suggestive of polycythemia which mimicked SAH.
The patient gave a history of congenital cyanotic heart disease, i.e., Fallot’s tetralogy; however, he had not undergone any surgical correction. Instead, he underwent a haematological work-up, which showed increased haemoglobin of 24 g/dL, hematocrit of 75% and meant the corpuscular volume of 93f/L. Chronic hypoxia and polycythaemia secondary to cyanotic heart disease can mimic subarachnoid haemorrhage on imaging and thus needs to be considered a differential when evaluating such patients.

Discussion

Pseudo SAH is a potential imaging pitfall, which mimics true SAH. Several SAH mimics include diffuse cerebral oedema due to various causes, purulent meningitis, large areas of cerebral infarction, SDH, leakage of intravenous contrast into subarachnoid space, status epilepticus, intracranial hypotension, intrathecally administered contrast medium, and polycythaemia (1). In all these cases, there is no acute intracranial haemorrhage. This case report mainly focussed on the mimic of SAH in hypoxemia-induced polycythaemia and a brief discussion on other causes.

The patient gave a history of cyanotic heart disease, i.e., Fallot’s tetralogy; however, he had not undergone any surgical correction. Instead, he underwent a haematological work-up, which showed increased haemoglobin of 24 g/dL, hematocrit of 75%, and meant the corpuscular volume of 93f/L. Chronic hypoxia and polycythaemia secondary to cyanotic heart disease can mimic subarachnoid haemorrhage on imaging and thus needs to be considered a differential when evaluating such patients.

Polycythaemia (PC): Patients with polycythemia secondary to chronic hypoxemia due to complex cardiopulmonary situation with a significant intermixture of the blood of systemic and pulmonary circulations also show generalized proliferation and dilation of the intracranial veins to maintain adequate cerebral oxygen supply. Nevertheless, increased attenuation of venous sinuses is typically observed in cerebral venous thrombosis (CVT).

The use of Hounsfield unit (HU) measurements to discriminate between pseudo-SAH and SAH does not provide any favourable information in the NECT brain as pseudo-SAH in our case is due to an increased hematocrit level of 75%.

MR imaging with Susceptibility Weighted Imaging (SWI) is sensitive to different oxygen statuses. SWAN, a T2* weighted multiple echo gradient-echo sequence that differs from SWI using only magnitude information, also has that quality.3 SWI sequence shows marked hypointense signal depiction of cerebral and subarachnoid veins due to hypoxemia.

MR venography/CT venography helps differentiate between Cortical Vein Thrombosis (CVT) patients with polycythemia with hyperdense venous sinuses.4 Chest radiographs also provide a clue to cyanotic heart diseases chronic obstructive airway disease conditions. On clinical examination of the patient, central cyanosis and clubbing of fingers can be appreciated, favouring pseudo-SAH diagnosis.

Cerebral oedema and a large area of Cerebral infarction: Several aetiologies could result in diffuse brain oedema. The cerebral cortex becomes displaced into CSF space, veins become congested and decreased attenuation of brain parenchyma due to oedema increases “contrast” between parenchyma and cistern. Effaced ventricular system and poor grey-white matter differentiation may imply this situation.

Purulent meningitis: Infectious meningitis, especially tuberculous meningitis, has been reported to show measure increased CT attenuation over basal cistern leak of proteinaceous material due to blood barrier disruption and purulent material in SA space leads to increased attenuation. HU unit measurements of 35-40 HU (<50HU) were presumed to indicate pseudo-SAH in patients with purulent meningitis and post-resuscitation encephalopathy.

Clinical history, CSF analysis, and enhancement of based cistern in post-contrast CT are clues for diagnosis.

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

Contrast extravasation and after myelography: Reduced contrast clearance in renal failure, overdose contrast usage in angiographic studies and intrathecal injection of contrast media in myelographic studies shows contrast extravasation in brain and contrast distribution in subarachnoid spaces, respectively mimics SAH appearance. The majority of patients recover without any neurological deficits.

Other conditions like intracranial hypotension status epilepticus also mimic SAH in the NECT brain due to tight basal cisterns, secondary sagging of brain parenchyma, focal gyral swelling, respectively.

Conclusion

Hyper attenuation of the sulcal and basal cisterns on NECT in most cases represent SAH. However, in non-traumatic patients who presented with chronic variable symptoms, history of cardiopulmonary resuscitation, status epilepticus, previous history of invasive procedures like angiographies and myelography, one...
should be aware of pseudo-SAH. Recurrent headaches and history of our patient’s cyanotic heart disease fulfilled the criteria of chronic tension headache, secondary to chronic hypoxemia.

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**Conflict of interest**

There are no conflicts of interest to declare by any of the authors of this study.

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