Pseudo-subarachnoid Hemorrhage in a Patient with Acute on Chronic Respiratory Failure

Sarbesh Tiwari, Pawan Kumar Garg, Preetham Patavardhan, Nishant Kumar Chauhan[1], Suryanarayanan Bhaskar[2], Pushpinder Singh Khera

Departments of Diagnostic and Interventional Radiology, [1]Pulmonary Medicine, [2]Neurosurgery, All India Institute of Medical Sciences, Jodhpur, Rajasthan, India

Case History

A 48-years-old non-smoker male, presented with complaints of gradually progressive headache for 20 days and recent onset confusion with Glasgow coma scale (GCS) score of 14/15. The patient had complains of breathlessness associated with cough for last 6 years. On examination, the patient was conscious, but irritable without any signs of meningeal irritation or focal neurological deficit. Patient was tachypneic with SpO_2 of 70% on room air. Corresponding arterial blood gas analysis revealed a pH of 7.25, PCO_2-74 mmHg, PO_2-35 mmHg and bicarbonate 31 mEq/L. Blood investigation revealed neutrophilic leucocytosis, polycythemia with hemoglobin level of 18.8 gm/dl and haematocrit of 59.8%.

The non-contrast CT (NCCT) head revealed diffuse cerebral edema with hyperdensities distributed along the sulcal spaces of bilateral cerebral hemispheres and the basal cisterns [Figure 1a and b]. Considering the possibility of subarachnoid hemorrhage, a CT angiogram of the brain was obtained, which did not reveal any aneurysm/vascular malformations [Figure 1c]. Correlating with the clinical details, diffuse cerebral edema, the absence of intraventricular hemorrhage and the absence of pathology on CT angiogram, a possibility of "Pseudo-SAH" was considered. MRI brain was done for further evaluation which showed diffuse cerebral and cerebellar edema [Figure 1d and e] with effacement of sulcal spaces causing transtentorial herniation and mild tonsillar descent. No FLAIR hyperintensities were noted within the sulcal spaces [Figure 1f and g]. The SWAN (susceptibility-weighted angiography) images revealed engorged and extensively blooming cortical and transcerebral veins along bilateral cerebral hemispheres [Figure 1h]. The MRI findings further reaffirmed the diagnosis of Pseudo-SAH. Diagnostic cerebral angiogram was deemed unnecessary and hence not performed. A CT chest was done for evaluation of breathlessness which showed mosaic attenuation in bilateral lung parenchyma with bronchiectasis in bilateral lower lung zones with predominant right sided involvement [Figure 2a]. Dilated pulmonary artery was noted suggestive of pulmonary hypertension [Figure 2b].

A diagnosis of bronchiolitis with bronchiectasis causing type 2 respiratory failure and hypercapnia induced diffuse cerebral edema was made. The patient was managed with intravenous antibiotics, systemic corticosteroids, inhaled bronchodilators, oxygen therapy and non-invasive ventilatory support for correction of hypercarbia. Two therapeutic phlebotomies were performed for secondary polycythemia. Gradually patient's sensorium improved with disappearance of headache over a period of 7 days. A follow-up NCCT head revealed complete clearance of sulcal hyperdensities with significant resolution of cerebral edema [Figure 2c and d].

Discussion

Pseudo-SAH is a radiological diagnosis based on the presence of increased attenuation along the subarachnoid spaces in the background of significant cerebral edema and the absence of true SAH. The most common cause of pseudo-SAH is hypoxic brain damage caused by cardiac arrest. Other common causes are basal meningitis, leptomeningeal carcinomatosis, secondary polycythemia, spontaneous intracranial hypotension, bilateral subdural hemorrhage, chronic hypoxemia, infarction, status epilepticus etc.[1]

The exact mechanism of development of pseudo-SAH remains uncertain. The most common postulated mechanism is diffuse cerebral edema causing compression of dural venous sinuses, resulting in stagnation and dilation of cortical veins. These cortical veins fill up the sulcal spaces replacing the hypodense CSF and giving raise to SAH like appearance.[2] The present case describes appearance of pseudo-SAH secondary to acute on chronic respiratory failure. Prolonged hypoxia can cause severe hypercapnia, which induces increase cerebral blood flow via pH-induced chemoregulatory cerebrovasodilation.[3]Hyperemia with increased vascular permeability from severe vasodilatation is responsible for cerebral edema.[4] Diffuse cerebral edema causes compression of dural venous sinuses resulting in stagnation and dilation of cortical veins.In addition, secondary polycythemia, which is associated with these conditions results in increased attenuation

Address for correspondence: Dr. Pawan Kumar Garg, Diagnostic and Interventional Radiology, All India Institute of Medical Sciences, Jodhpur 342005, Rajasthan, India.
E-mail: drgargpawan@gmail.com

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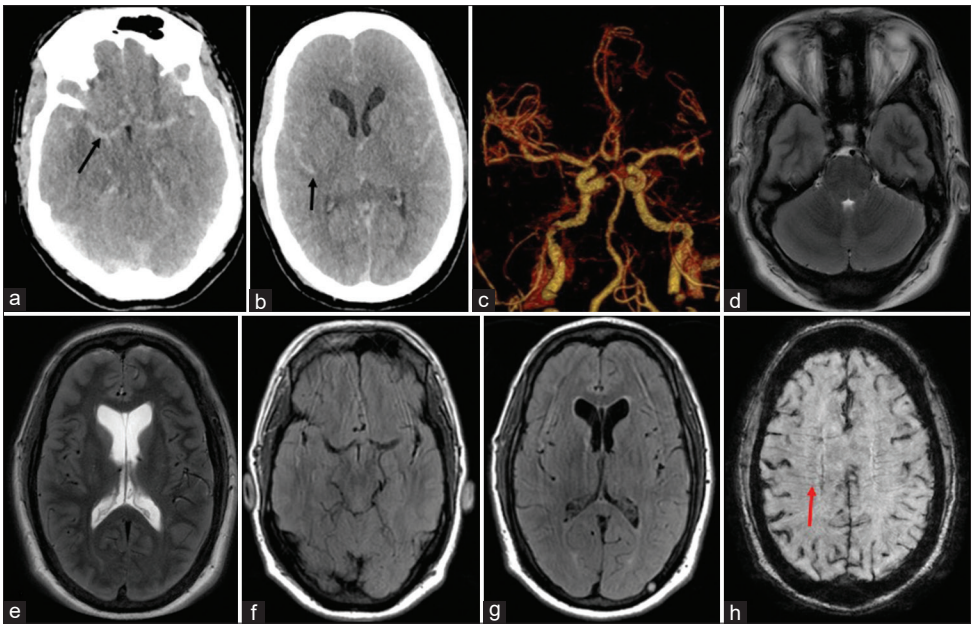

Figure 1: NCCT head images (a and b) shows hyperdensity along bilateral sylvian fissure and cortical sulcal spaces (black arrows) with diffuse cerebral edema. CT angiogram brain (c) does not reveal any aneurysm/vascular malformation. The axial T2WI images (d and e) shows diffuse cerebellar and cerebral edema with effacement of sulcal spaces. The FLAIR images (f and g) does not show any sulcal hyperintensity suggesting absence of subarachnoid hemorrhage. The Swan image (h) shows prominent veins along bilateral cerebral hemisphere and presence of trans-cerebral veins (red arrow)

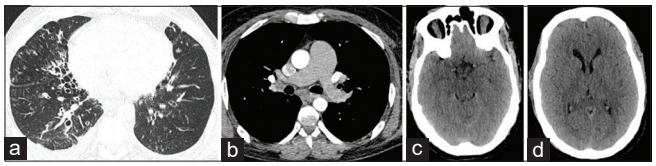

Figure 2: Axial (a) HRCT scan shows bronchiectatic changes are noted at bilateral lower lung zones (right > left). The axial CECT scan (b) shows dilated pulmonary artery suggesting pulmonary hypertension. The follow axial CT scan of brain at time of discharge (c and d) showed complete resolution of the sulcal hyperdensities with minimal reduction of cerebral edema

of intracranial vessels contributing to the pseudo-SAH appearance.[5]

Neuroimaging helps differentiate SAH from pseudo-SAH. On NCCT head, the attenuation values of true-SAH are observed to be higher than that of pseudo SAH. While the true-SAH measures 60-70 HU in attenuation, those of pseudo SAH are found to be 30-40 HU.[6,7]True aneurysmal SAH is often, but not invariably is associated with intraventricular hemorrhage, whereas in pseudo SAH no such changes are present.[7]On MRI, a combination of SWI and FLAIR images have higher detection rate for subarachnoid hemorrhage, particularly in the subacute and chronic phase. MRI brain, in the present case, demonstrated prominent cortical veins and transcerebral veins in bilateral cerebral hemisphere. This is secondary to increased oxygen extraction fraction and an increase in deoxyhemoglobin in the capillaries and veins. Further, absence of sulcal FLAIR hyperintensity and intraventricular hemorrhage reliably rules out SAH.

In conclusion, it is imperative to identify pseudo-SAH in emergency setting when NCCT brain shows diffuse SAH-like appearance. Whereas, true-SAH warrants imaging of the vasculature like CT angiography and DSA with further surgical/endovascular treatment, pseudo-SAH begs urgent medical attention to recognize the etiology and subsequent management.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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