VOLUME - 13, ISSUE - 12, DECEMBER - 2024 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

Neurology

PSEUDO HYPOXIC BRAIN SWELLING: A POORLY DEFINED COMPLICATION POST-UNEVENTFUL BRAIN SURGERY – REPORT OF TWO CASES AND LITERATURE REVIEW

Dr. Vinit Suri* MBBS, MD Medicine, DM Neurology, Senior Consultant, Indraprastha Apollo Hospital, New Delhi *Corresponding Author

Dr. Kanika Suri MBBS, MD General Medicine, DM Neurology Resident, Mahatma Gandhi University of Medical Science & Technology

ABSTRACT Pseudohypoxic brain swelling, also known as postoperative intracranial hypotension is a rare and intriguing complication following routine elective spine or brain surgery. It is usually underdiagnosed and can be a fatal complication of seemingly uneventful neurosurgery. Diagnosis is usually confused with acute hypoxic injury in view of its clinical presentation, and imaging characteristics which are strikingly similar to anoxic brain injury. It manifests on CT brain as a hypodensity of the basal ganglia and thalami, and on MRI brain as oedema and restricted diffusion in the basal ganglia and thalami, similarly to hypoxic-ischemic brain injury. Functional outcomes vary from remarkable neurological recovery to death. We present two cases of unexpected clinical deterioration in patients with otherwise uneventful neurosurgery for subdural hematoma (SDH) and the postoperative MRI scans exhibited severe bilateral oedema in deep grey structures.

KEYWORDS : subdural hematoma, brain hypoxia, pseudohypoxia, brain edema

INTRODUCTION

Pseudohypoxic brain swelling (PHBS), also known as postoperative intracranial hypotension-associated venous congestion (PIHV), is a rare but potentially fatal complication following seemingly uncomplicated spine or brain surgeries. The condition is characterized by symptoms that mimic those of acute hypoxic brain injury, which can make diagnosis challenging. Clinically, patients may present with a range of neurological deficits, including headaches, cranial nerve palsies, new-onset seizures, alterations in the level of consciousness, brainstem dysfunction, vegetative states, or even death. The condition is particularly concerning because it often arises unexpectedly after otherwise uneventful surgical procedures, complicating the postoperative care process.

Van Roost et al. first described PHBS in 2003 in a study that reviewed 17 patients who postoperatively developed clinical features mimicking global cerebral hypoxia following the application of subgaleal suction drainage during cranial surgery[1]. Parpaley et al. in 2011 reported the first two cases of PHBS following spinal surgery with the application of epidural suction drainage[2]. Since then, relatively few cases have been reported in the literature[3,4], highlighting both the rarity of the condition and the need for increased awareness among clinicians to ensure prompt diagnosis and management.

Case Reports

Case 1:

A male presented in early 70s with fatigue and mild unsteadiness since 1 week prior to admission. He also had mild left frontal non throbbing headache since past 2 days followed by weakness of right upper and lower limb with non-fluent speech within 12 hours of admission. A history of fall a few weeks prior was noted. On examination, the patient was conscious but had expressive aphasia and right hemiparesis (power 4/5 in upper and lower limb), with pupils that were equal and reactive to light. A non-contrast computed tomography (NCCT) scan of the head (Fig 1a-c)revealed a large chronic subdural hematoma (SDH) along the left cerebral convexity, causing a midline shift of 13 mm.

The patient underwent a left fronto-temporoparietal craniotomy and evacuation of the SDH under general anesthesia, during which a subgaleal drain was placed. The surgery itself was uneventful. However, postoperatively, the patient did not regain consciousness, prompting an urgent NCCT scan, which showed no new bleeding. Despite this, the patient remained in a coma with a Glasgow Coma Scale (GCS) of E1VTM1 and developed generalized tonic-clonic seizures. Electroencephalography (EEG) revealed signs of non-reactive background rhythms of an encephalopathy with suspected left fronto temporal sharp wave discharges possibly epileptiform.

Magnetic resonance imaging (MRI) of the brain (Fig 2a-d) showed bilateral symmetrical T2 and FLAIR hyperintense areas with lentiform rim sign seen involving the basal ganglia and thalami without any restricted diffusion which was likely to be secondary to pseudo-hypoxic brain swelling (secondary to intracranial hypotension associated venous congestion). No vascular abnormalities were noted. The patient gradually improved with intensive care, including anticonvulsants and supportive measures, regaining consciousness and significant motor function over 6 weeks.

Case 2

The second case is a hypertensive male in his early 90s who presented after a fall a month prior, initially without neurological deficits. Routine investigations, however, revealed a subacute to chronic SDH of thickness 8.2 mm along the right fronto-parietal cerebral convexity. The patient was managed conservatively but later developed new-onset left-sided weakness and headache. Subsequent NCCT imaging showed an acute-on-chronic SDH with a midline shift of 2 mm to left. On examination, the patient was fully conscious with normal vital parameters. Motor examination revealed left hemiparesis (power 3/5 in left upper and 4/5 in left lower limb).

The patient underwent burr hole surgery and evacuation of the hematoma under general anesthesia. Postoperatively, the patient did not wake up and remained deeply comatose, with no response to pain. Multiple generalized seizures were observed and managed with anticonvulsants. An MRI with MR angiography (Fig 3 a-h) revealed findings consistent with PHBS, similar to the first case, showing bilateral symmetrical hyperintensities in the basal ganglia and thalami, without any signs of vascular thrombosis.

The differential diagnosis included anoxic brain injury, deep venous thrombosis, and metabolic-related brain edema. However, the lack of intraoperative hypoxia, normal vascular imaging, and absence of metabolic abnormalities helped narrow the diagnosis to PHBS. The patient required intensive care for 3 weeks but eventually made a full neurological recovery over 10 weeks.

DISCUSSION

The clinical presentation of PHBS often closely resembles that of anoxic brain injury, which can lead to misdiagnosis and delayed treatment. Imaging studies typically show symmetrical hypodensities on CT and hyperintensities on MRI in the basal ganglia and thalami[6]. The pathophysiology of PHBS is thought to involve acute loss of cerebrospinal fluid (CSF), which results in decreased intracranial pressure. This loss can cause a compensatory increase in cerebral blood volume, particularly in the venous system, due to the brain's attempt to maintain intracranial volume homeostasis as described by the Monro-Kellie doctrine.

When CSF volume decreases rapidly, venous blood volume increases to fill the resultant space, leading to venous congestion and subsequent vasogenic edema. This process can occur with or without detectable CSF leaks and is exacerbated by the use of subgaleal or epidural suction drains. Other contributing factors include intracranial decompression, rapid brain re-expansion, and impaired autoregulation.

Prompt recognition and appropriate management of PHBS are crucial to prevent permanent neurological damage. Early interventions should focus on minimizing further CSF loss and managing intracranial pressure. Positioning the patient in a Trendelenburg position, ensuring adequate hydration, and avoiding the use of subgaleal drains are essential initial steps. In severe cases, surgical interventions such as decompressive sub-occipital craniectomy may be necessary to alleviate intracranial pressure and prevent further brain injury[7]

CONCLUSION

PHBS is a rare but potentially life-threatening condition that can occur following spine or brain surgery. However, with timely recognition and appropriate management, including measures to control CSF leakage and intracranial pressure, outcomes can be significantly improved. Both cases presented here illustrate the importance of maintaining a high index of suspicion for PHBS in postoperative neurosurgical patients who exhibit unexplained neurological deterioration. Early intervention and aggressive management can lead to full recovery without long-term neurological deficits, underscoring the importance of awareness and prompt action in managing this rare complication.

Abbreviations

Pseudohypoxic brain swelling (PHBS)
postoperative intracranial hypotension-associated venous congestion (PIHV)
non-contrast computed tomography (NCCT)
subdural hematoma (SDH)
Glasgow Coma Scale (GCS)
Electroencephalography (EEG)
Magnetic resonance imaging (MRI)
cerebrospinal fluid (CSF)

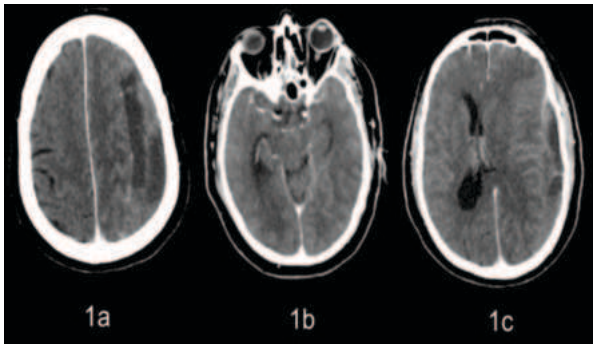

Figure I 1(a-c): Preop NCCT Head: a) Axial section showing Left frontoparietal acute on chronic SDH; b) and c) Axial section showing mass effect and Midline shift to Right.

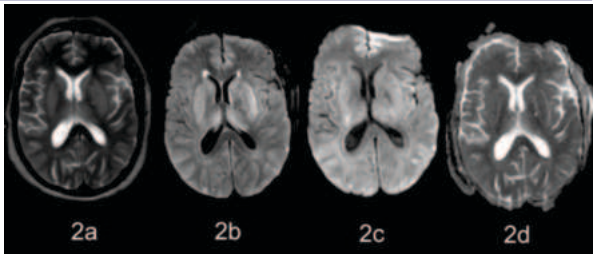

Fig 2 (a-d)Post OP MRI brain: 2.a) & b) Axial section showing bilateral symmetrical T2 and FLAIR hyperintense areas with lentiform rim sign seen involving the basal ganglia; 2. c) & d) Axial section showing no diffusion restriction on DWI and ADC drop in corresponding areas.

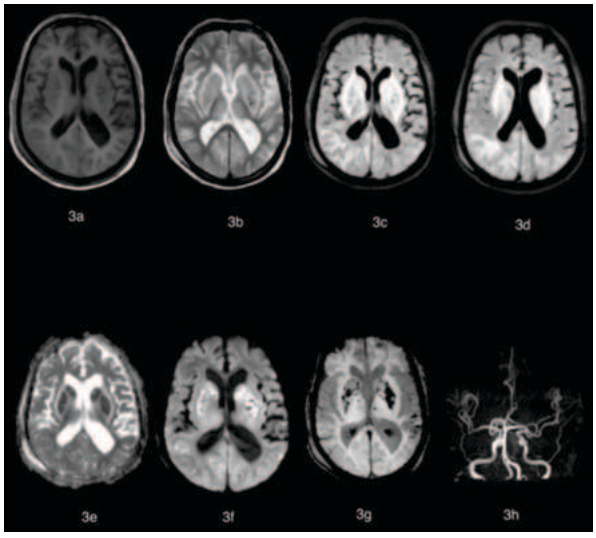

Fig 3 (a-h) MRI Brain with MR angiography brain a) Axial section showing normal intensity basal ganglia and thalamus; b) & c) T2 and FLAIR hyperintense areas with lentiform rim sign seen involving the basal ganglia and thalami d) FLAIR hyperintense areas in bilateral periventricular region e) & f) Areas of restricted diffusion on DWI and ADC drop involving the bilateral basal ganglia and thalami; g) SWI sequence showing Blooming in corresponding areas.; h) MR Angiography brain showing insignificant bilateral MCA atherosclerotic disease.

Acknowledgments: The authors thank the patient for showing patience and helping us improve care through the use of information.
Conflicts of Interest: There are no conflicts of interest.

Author Contribution

Conceptualization: Dr. Vinit suri
Methodology: Dr. Vinit suri
Software: Dr. Vinit suri
Validation: Dr. Vinit suri
Format analysis: Dr. Vinit suri
Investigation: Dr. Vinit suri, Dr. Kanika Suri
Resources: Dr. Vinit suri
Data curation: Dr. Kanika Suri
Project administration: Dr. Vinit suri
Funding acquisition: Dr. Vinit suri
Visualization: Dr. Vinit suri
Supervision: Dr. Vinit suri
Writing-original draft: Dr. Vinit suri, Dr. Kanika Suri
Writing- review and editing: Dr. Vinit suri, Dr. Kanika Suri

Ethical Approval Institutional Review Board approval is not required.
Declaration of Patient Consent: The authors certify that they have obtained all appropriate patient consent.
Financial Support and Sponsorship Nil.

REFERENCES

1. Van Roost D, Thees C, Brenke C, et al.: Pseudohypoxic brain swelling: a newly defined complication after uneventful brain surgery, probably related to suction drainage. Neurosurgery. 2003, 53:1315-26.
2. Parpaley Y, Urbach H, Kovacs A, et al.: Pseudohypoxic brain swelling (postoperative intracranial hypotension-associated venous congestion) after spinal surgery: report of 2 cases. Neurosurgery. 2011, 68:E277-83
3. Geran M, Carson C, Rabab E, et al , Pseudohypoxic brain swelling following elective lumbar laminectomy: A rare case report and review of literature Radiology Case Reports 19 (2024) 1351–1355 .
4. Dickinson J, Kroll D, Bentley J, et al. (April 09, 2018) Pseudohypoxic Brain Swelling After Elective Lumbar Spinal Surgery: Case Report. Cureus 10(4): e2454. DOI 10.7759/cureus.2454
5. Sarika J, Himanshu A, Kavita S (2023) Pseudo Hypoxic Brain Swelling - An Unanticipated Life-Threatening Complication in Neurosurgery – Report of Two Cases with Review of Literature. Int J Anesthetic Anesthesiol 10:164.
6. Hadizadeh DR, Kovács A, Tschampa H, Kristof R, Schramm J, et al. (2010) Postsurgical intracranial hypotension: Diagnostic and prognostic imaging findings. AJNR Am J Neuroradiol 31: 100-105.
7. Hou K, Zhu X, Zhang Y, Gao X, Suo S, et al. (2018) Early post□operative cerebrospinal fluid hypovolemia: Report of 7 cases. Exp Ther Med 15: 5119-5124.