CASE SERIES ASIAN JOURNAL OF MEDICAL SCIENCES

Untoward outcomes of successful chronic subdural hematoma evacuation, cortical blindness and brain stem stroke: A case series

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ABSTRACT

Chronic subdural hematoma (CSDH) is one of the most commonly encountered emergencies in neurosurgery practice. Burr hole with drainage of hematoma has been a time-tested standard treatment with good results and in itself a simple procedure to relieve patient clinical status. However, apparently simple surgery may at times have an unexpected surgical outcome of bilateral vision loss. All bilateral CSDHs should be treated diligently with early recognition of any visual deterioration in post-operative phase if any and due to possible interventions to be offered to the patients for prevention of permanent vision loss.

Key words: Chronic subdural hematoma; Vision loss; Cortical blindness; Burr hole

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INTRODUCTION

Chronic subdural hematoma (CSDH) is a commonly encountered emergency in neurosurgical practice that usually entails surgical intervention as hematoma evacuation though there may be different choices among techniques used including burr hole; twist drill, craniotomy, or nowadays endoscopic at some centers.[1]

The usual culprit is trivial head injury but other causes may be coagulopathy and others. Usually, outcomes are favorable with good recovery post-surgery. One side is affected usually but in about 1 in 5 cases there may be involvement of bilateral sides. Complete vision loss in a bilateral CSDH evacuation or unilateral stroke or vision deficits following unilateral CSDH evacuation are rare complications of evacuation of CSDH.[2]

Only few reports in literature[3-5] are there as posterior cerebral artery (PCA) territory stroke or vision loss following evacuation of CSDH. We here in this case series report three such cases with vision loss and brain stem stroke following evacuation of CSDH at our center with possible explanation in each case.

CASE SERIES

This was a retrospectively analyzed series of three patients operated at our institute from November 2021 to July

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2022. All of the patients were operated for CSDH as per standard protocol with burr hole and evacuation. All of them were later investigated with relevant radiological investigations to know the cause of vision loss or stroke following hematoma evacuation except one who was not willing to be admitted and further investigations.

Case 1

A 52-year-old male patient who was a known alcoholic with a history of fall on road about one and half month back came with a chief complaint of holocranial headache for a week followed by altered sensorium for the last 1 day. The patient was known case of hypertension and on medication. There was no other chronic illness. On neurological examination, the patient was conscious, drowsy, and disoriented, pupil was bilaterally reactive to light and equal in size. Non-contrast computed tomography (NCCT), head was suggestive of bilateral chronic subdural hematoma over cerebral convexity (Figure 1).

Post-surgery, the patient was improved in terms of sensorium and headache relief and discharged from the hospital on day 3 of surgery with an uneventful stay in the hospital. During the first follow-up on day 8 of surgery, the patient complained nothing but as per attenders, the patient had difficulty in reaching and holding things when passed on to him but the patient denied any visual deterioration. The patient was readmitted and investigated further with a repeat NCCT brain which suggested complete removal of hematoma but hypodensity in the bilateral occipital region (Figure 2).

An ophthalmology workup revealed PL negative vision in bilateral eyes with normal fundus findings blood investigations including lipid profile were normal.

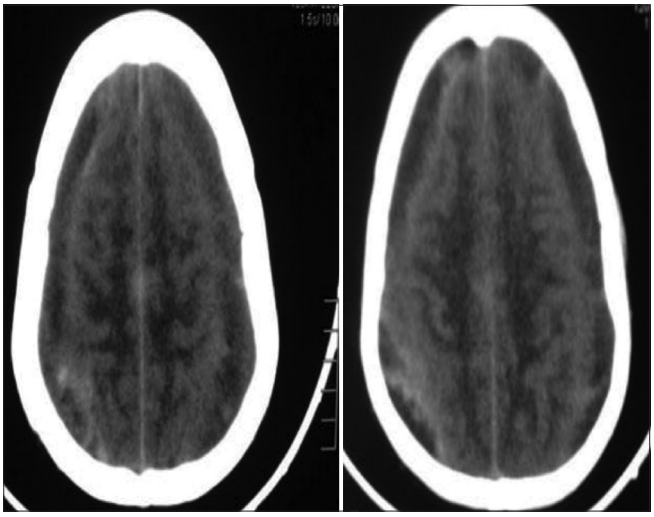

Figure 1: Preoperative non-contrast computed tomography head of case 1 showing bilateral subdural haeamtoma

Echocardiography was within normal limit. Patient was further worked up with MRI brain with magnetic resonance angiography (MRA) brain. MRI Brain revealed T2 and FLAIR hyperintensity in the region corresponding to computed tomography (CT) hypodensities but there was no corresponding restriction in diffusion-weighted imaging (Figures 3-5). MRA brain was suggestive no flow restriction in any of the vessels (Figure 6).

On retrospectively reviewing the case details of previous admission we noted an episode of increased blood pressure (BP) to the range of 180/100 mmHg during surgery which was managed with injectable labetalol and other anesthetic drugs. A diagnosis of PCA territory stroke/ posterior reversible encephalopathy syndrome (PRES) was suspected. Electroencephalography (EEG) was done which was reported to be normal. The patient was started on steroids with an anti-convulsant with good control of BP but there has been no improvement in vision.

Case 2

A 50-year-old male with a history of fall under alcohol influence followed by an episode of loss of consciousness followed by altered sensorium since one day. Patient also complained of on-and-off headache for a week. There was no history of chronic illness. On examination, the patient was unconscious, pupils were equally reactive, no eye-opening withdrawal to pain and he was intubated. Pre-operative Non-contrast CT (NCCT) head suggested right frontotemporo-parietal (FTP) chronic subdural heamatoma (CSDH) (Figure 7).

Post-surgery, the patient noted improvement in terms of neurological status. He was able to localize pain. Gradually, the patient was weaned off from ventilator and extubated on day 2, and was able to follow simple commands. Repeat NCCT brain on day 2 post surgery showed good evacuation of hematoma with resolution of mass effect (Figure 8). However, on postoperative day 4, patient developed neurological deterioration with dip in consciousness and respiratory difficulty followed by for re-intubation was done. Repeat NCCT brain post deterioration was showed PCA territory hypodensities with hyperdensity along right cerebral convexity as compared to previous day 2 scan (Figure 9).

The patient was started on symptomatic management with ventilator support and tracheostomized. Routine investigations including lipid profile were unremarkable. 2D echo was normal. Bilateral carotid Doppler suggested no compromise in bilateral carotid vessels.

MRI brain with MRA was done which revealed T2 hyperintensity in occipital region with diffusion restriction

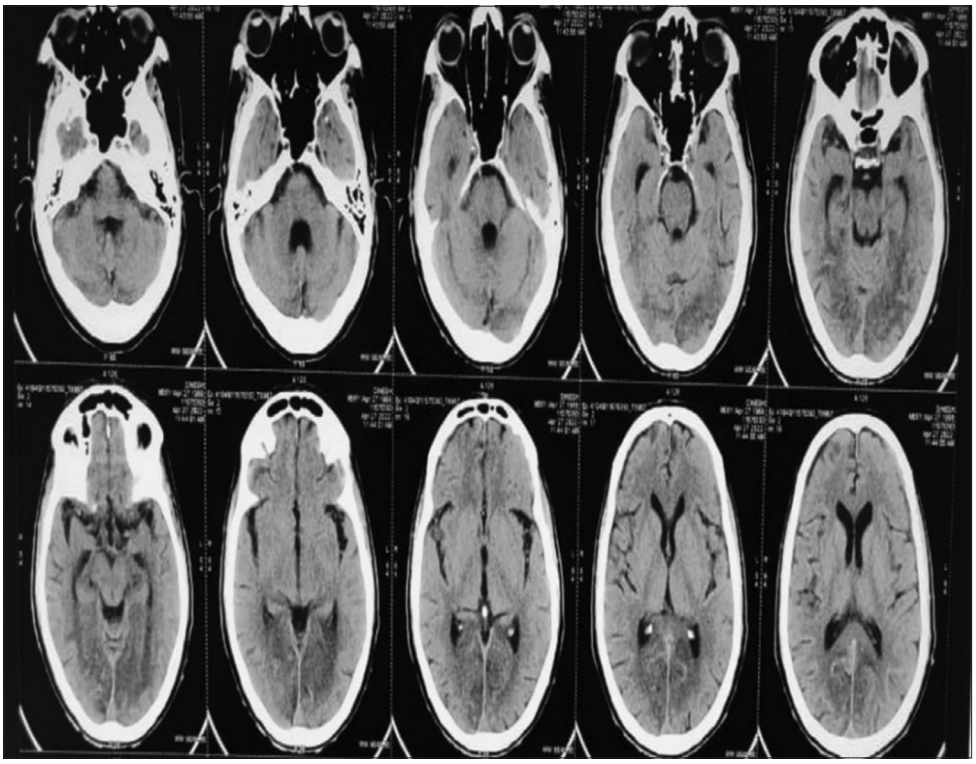

Figure 2: Computed tomography head was done on post-op day 8th which was suggestive of occipital hypo densities

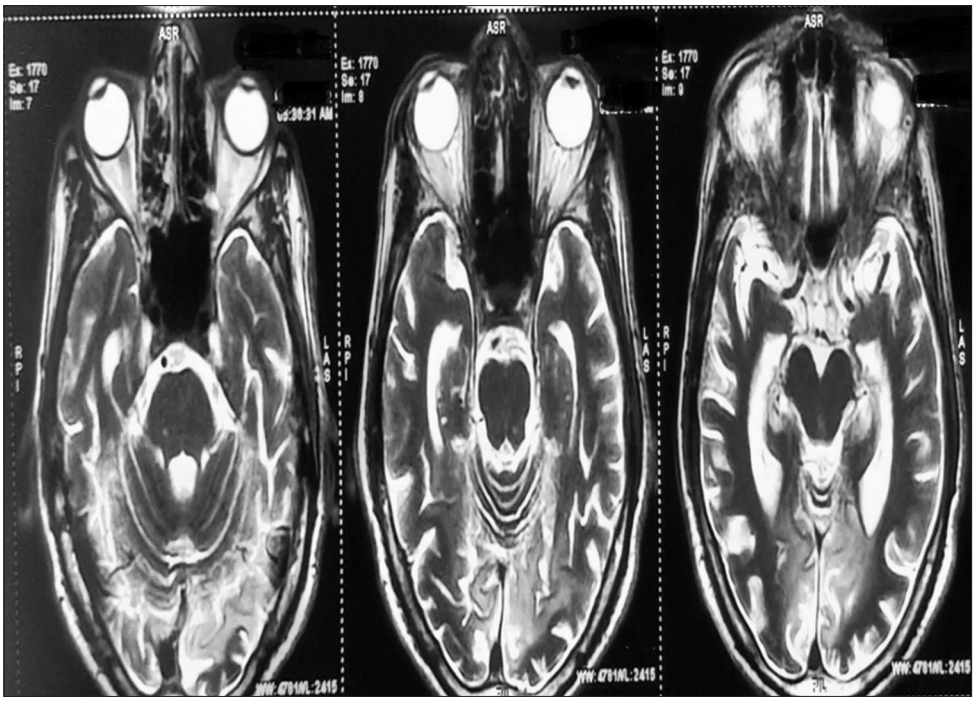

Figure 3: Magnetic resonance imaging brain shows T2 hyperintensities in bilateral occipital region

in PCA territory extending to brain stem suggestive of acute infarct along with subdural collection along right cerebral convexity (Figure 10). MRA brain vessels were suggestive of no luminal compromise of visualized vessels (Figure 11). A diagnosis of PCA territory stroke was made. In view of recollection in subdural space after resolution in previous scan; a suspicion of intracranial hypotension was made resulting in kinking of posterior circulation vessels. Although there was no sagging of brain seen in sagittal cuts (Figure 12a-c). Another possibility of some pre-existing

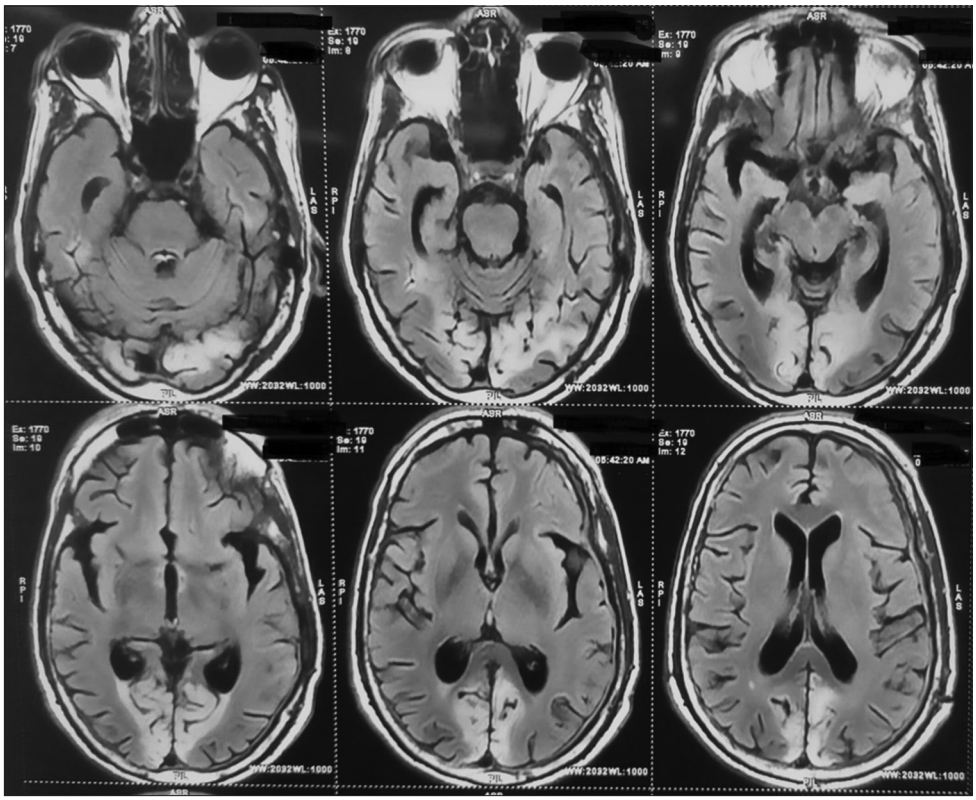

Figure 4: Magnetic resonance imaging brain shows T2 flair showing hyperintensities in B/L occipital region

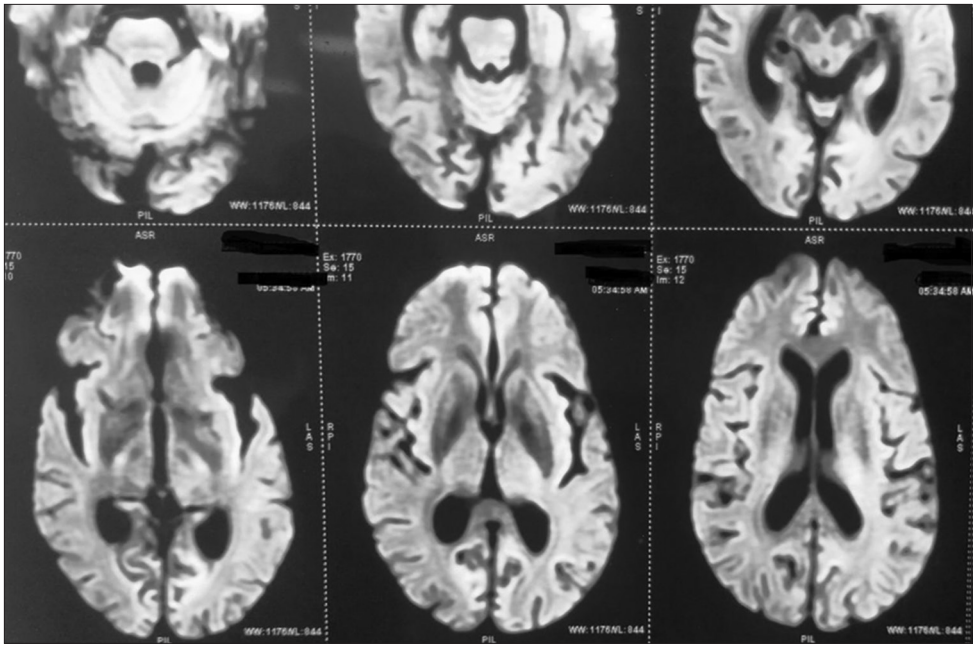

Figure 5: Diffusion weighted imaging showing no restriction

atherosclerosis was also kept in mind. The patient was unfortunately succumbed on day 9.

Case 3

A 42-year-old male patient known alcoholic with chief complain of sudden loss of consciousness since day of presentation with history of spontaneous subdural hematoma 15 days back and received conservative management for the same somewhere outside (Figure 13). No history of hypertension, diabetes mellitus and any other chronic illness. On examination, pupils were B/L reactive, eye opening was present on verbal command, speech was

confused and localizing pain was observed. NCCT Head on day of presentation was suggestive of right FTP CSDH with mass effect (Figure 14). The patient was operated in emergency with burr hole craniostomy and hematoma evacuation.

Post-surgery, patient improved in terms of improved sensorium. He was discharged with uneventful hospital stay on day 3 of surgery.

On follow-up in outpatient department on day 13 of surgery, patient complained of progressive vision loss in bilateral eyes. On examination there was normal pupillary reaction but the patient was not able to perceive direction of light. The patient was advised admission for further investigation and work up but patient was not willing for admission and gave negative consent for the same.

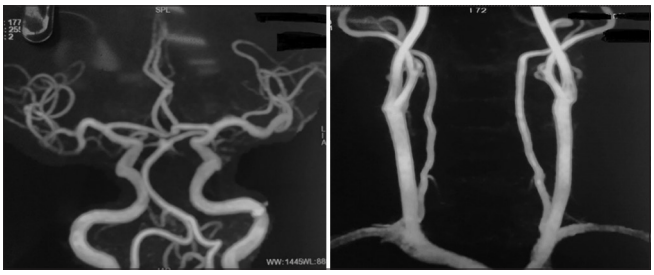

Figure 6: Magnetic resonance imaging showing no abnormal caliber (a and b)

Although no further investigations for this case could be done. We propose the vision loss to be due to spontaneous intracranial hypotension and kinking of posterior circulation vessels due to same in view of history of spontaneous subdural hematoma (SDH) and history of alcoholism.

DISCUSSION

CSDH is a common neurosurgical condition that is frequently encountered in neurosurgery practice. Its etiologies are dominated by traumatic head injury, and clinical presentations are various. The prognosis is generally good, but complications can be seen. Some of these complications can lead to the death of the patient.

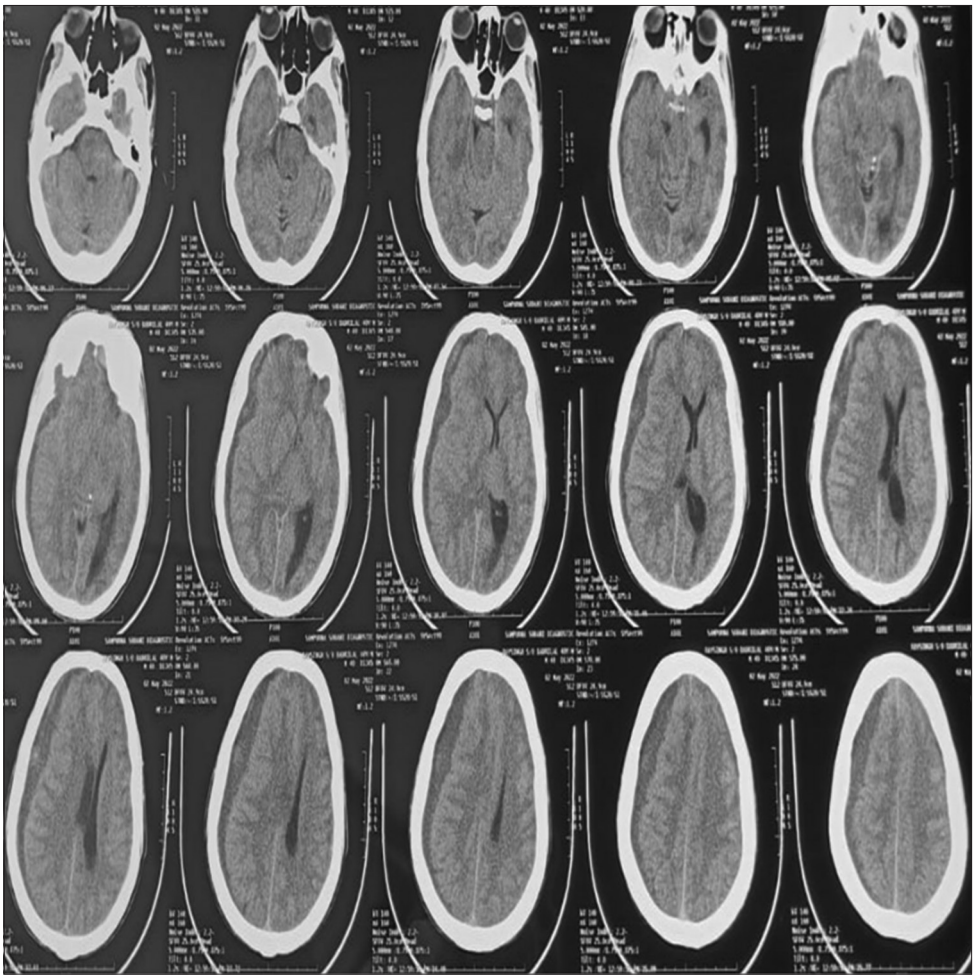

Figure 7: preoperative non-contrast computed tomography head suggestive of right fronto-temporo-parietal chronic subdural hematoma

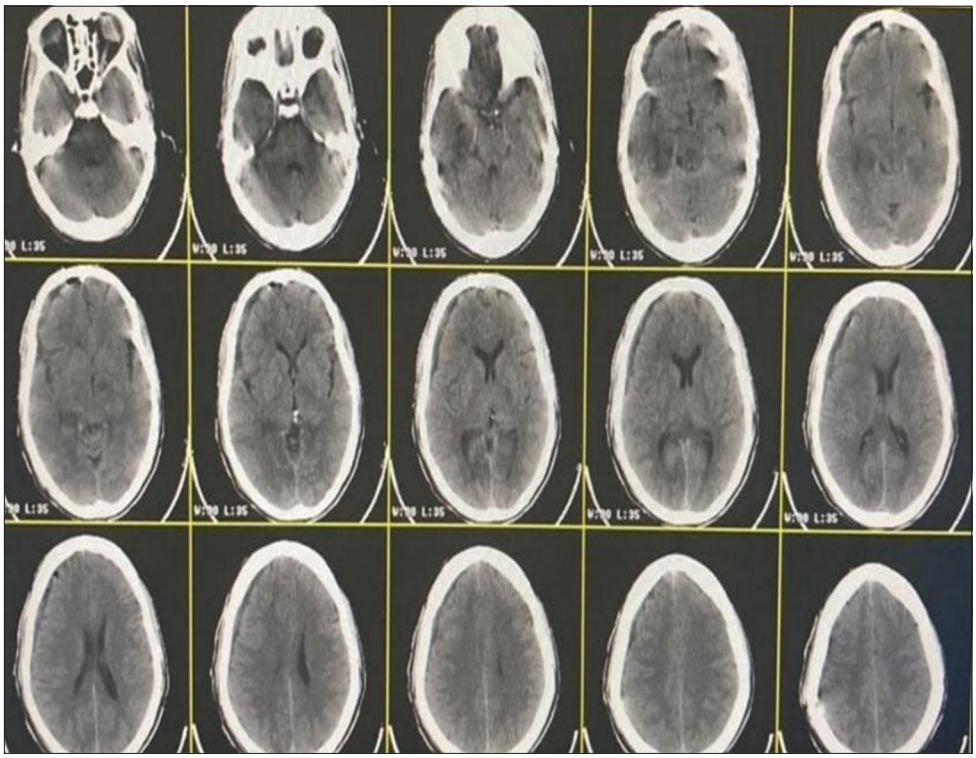

Figure 8: Repeat non-contrast computed tomography on POD 2 showing good evacuation of hematoma and resolution of mass effect hyperdensity along right convexity

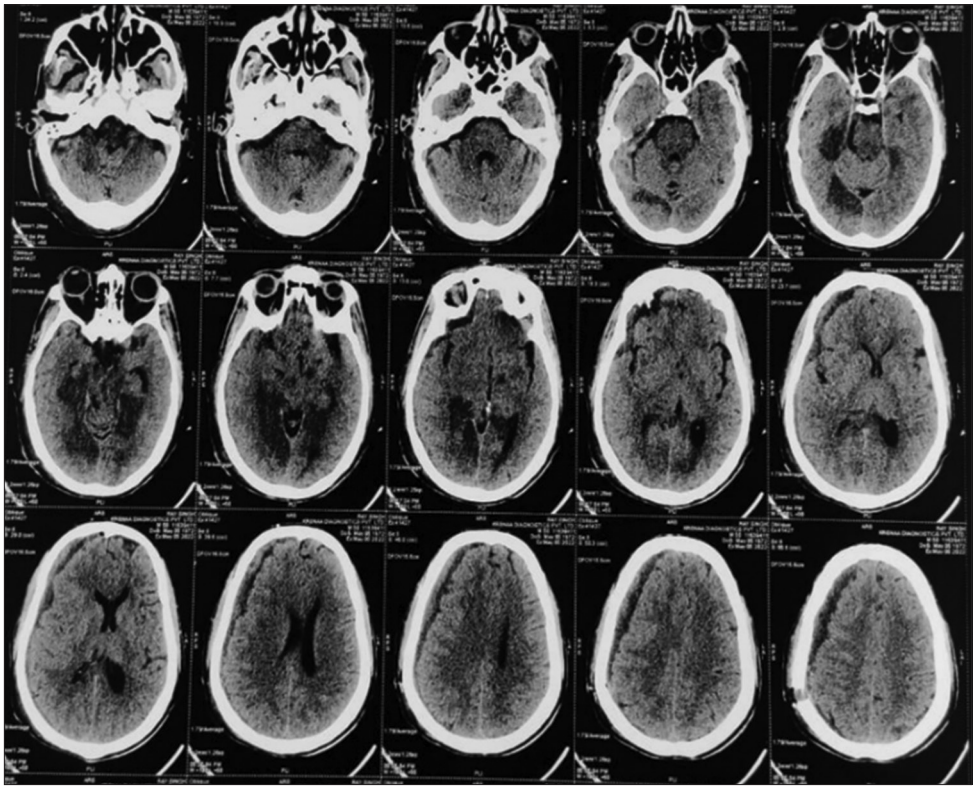

Figure 9: Repeat non-contrast computed tomography brain post deterioration showed posterior cerebral artery territory hypodensities with hyperdensity along right cerebral convexity

Cortical blindness is a rare neurologic condition characterized by loss of vision in the presence of an intact anterior pathway. It is usually binocular with preserved pupillary reflexes.[6] It occurs due to ischemia of the occipital

cortex due to a local event such as embolism or hemorrhage or, more commonly, due to global hypoperfusion.

Cortical blindness occurring following chronic subdural evacuation in the absence of any other associated risk factor is an extremely rare presentation. There are only a few cases which reported blindness following bilateral chronic subdural evacuation. The pathophysiology behind this rare complication remains to be understood.

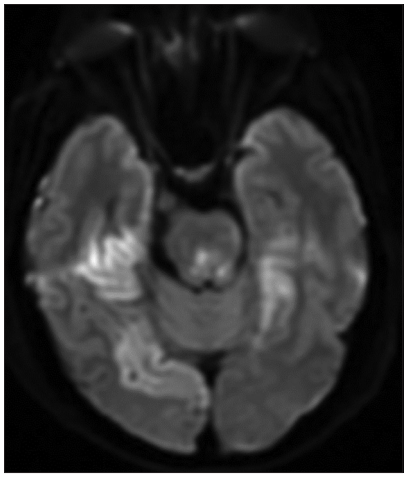

Figure 10: Diffusion weighted magnetic resonance imaging brain imaging showing restricted diffusion in B/L posterior cerebral artery territor

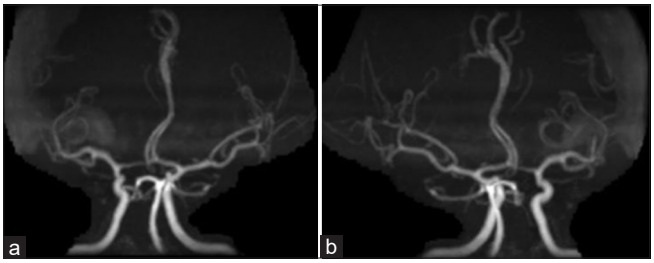

Figure 11: (a and b) Magnetic resonance imaging of brain vessels suggestive no luminal compromise

In 1980, Kaene published the first case report, detailing seven cases of persistent sight loss that resulted from tentorial herniation decompression. The reasons were postoperative infarction, bilateral CSDH, bilateral subdural empyema, traumatic intracerebral hematoma, and unilateral subdural hematoma in three patients. The occipital lobe polar ischemia and infarction that resulted from the kinking of the bilateral posterior cerebral arteries during the decompression may have been caused by a descending transtentorial herniation, according to the authors' conjecture. Three of these patients later had optic atrophy, a sign that posterior circulation compression and anterior visual pathway impairment had occurred.[4]

The second case of a 51-year-old patient who experienced bilateral blindness after a traumatic cerebrospinal fluid decompression has been documented by Russegger et al. The patient had bilateral optic atrophy, and the authors hypothesized that the optic atrophy and ensuing amaurosis were brought on by a breakdown in the altered vasoregulation of the optic nerve that happened during the intracranial pressure drop during the decompression.[7]

Kudo et al., reported the third and fourth case reports and described two patients who had bilateral chronic subdural evacuation and afterwards developed an occipital infarction and blindness. At admission, both patients had a low Glasgow coma scale, and their CT scans showed interpeduncular and ambient cistern compression. The severe aftereffects have been ascribed by the authors to central transtentorial herniation-induced occipital infarction.[5]

Balasubramanian et al., (2017) reported bilateral CSDH with cortical blindness following evacuation. They postulated the reason as preexisting chronic ischemia has led to the decrease in brain volume, thereby giving space for the collection of the chronic subdural hemorrhage, and subsequent surgical evacuation has caused the worsening of the ischemia resulting in PCA infarction.[2]

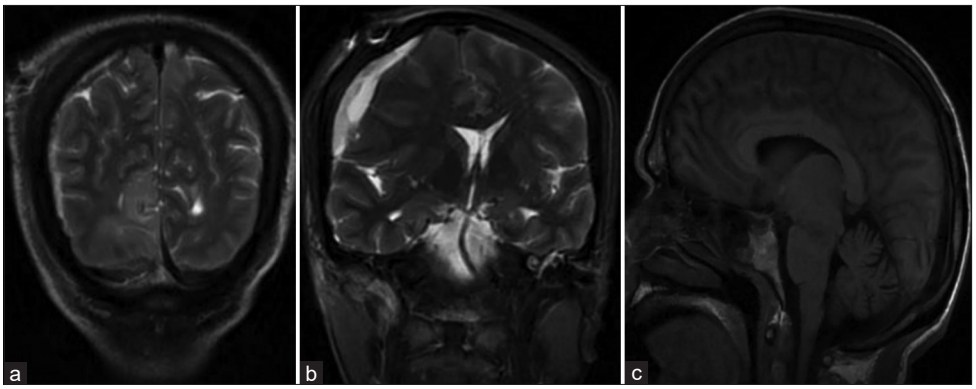

Figure 12: (a) T2 images showing right occipital hyperintensities (b) T2 images showing right subdural collection, (c) T1 sagittal image showing no brain sagging

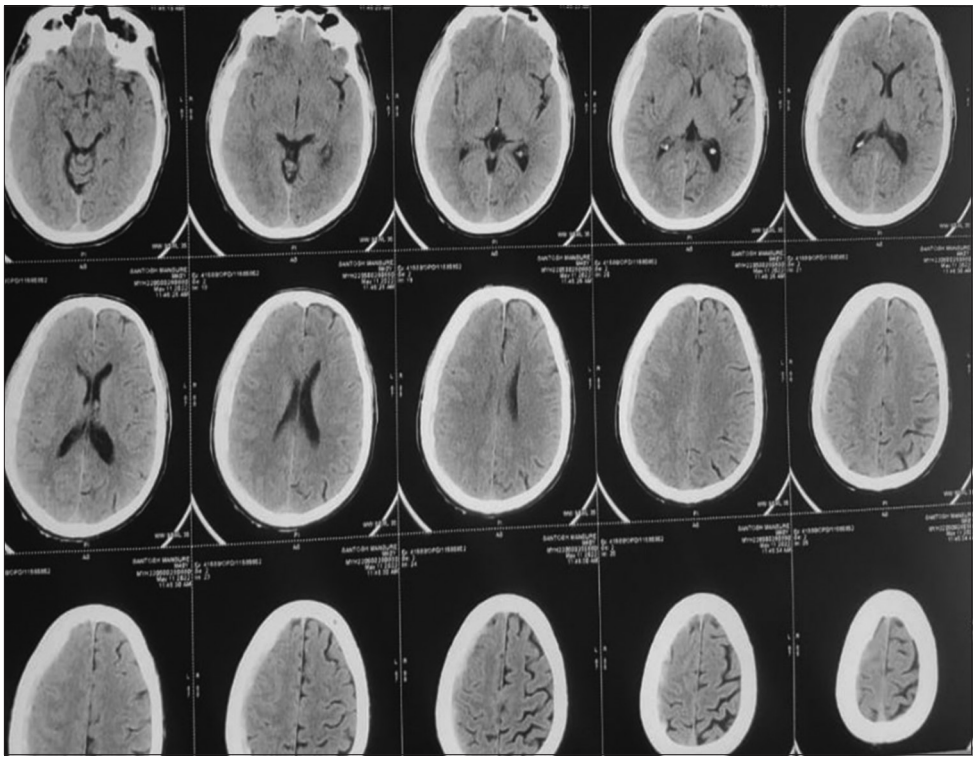

Figure 13: Initial non-contrast computed tomography with spontaneous right fronto-temporo-parietal subdural hematom

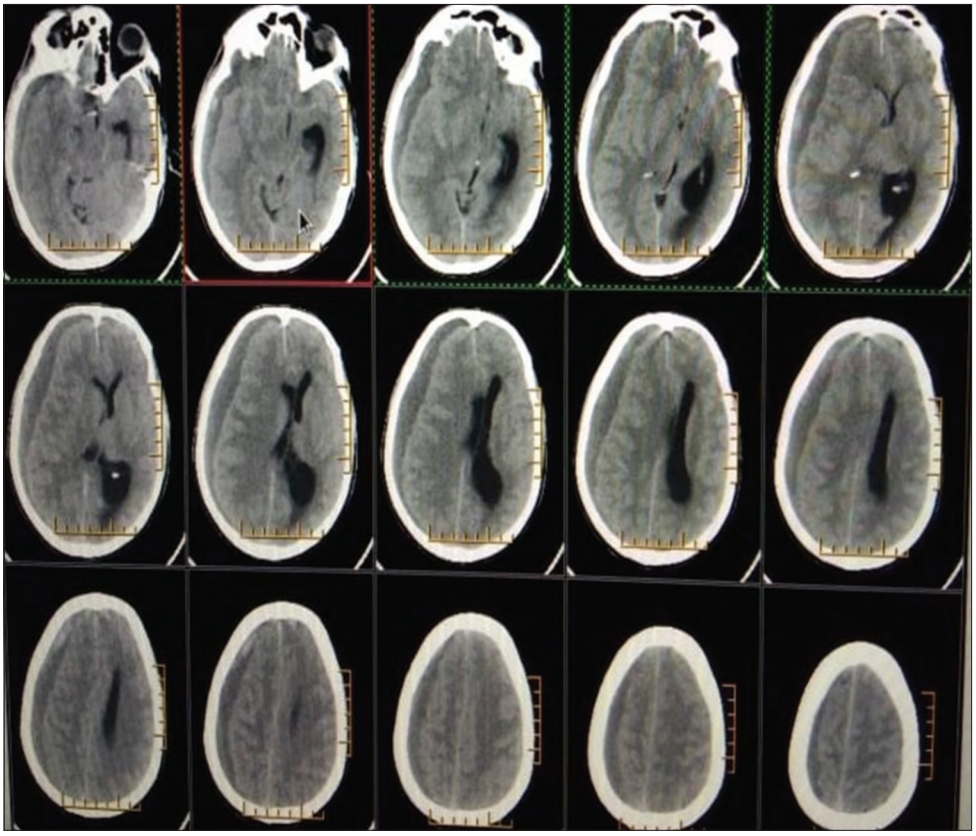

Figure 14: Non-contrast computed tomography on day of presentation with right fronto-temporo-parietal chronic subdural hematoma with mass effect

Adam et al., (2018) reported Anton Babinsky syndrome in one patient following evacuation of bilateral CSDH with bilateral occipital infarct but no possible cause of infarct has been mentioned.[8]

Finally, Imoumby et al., in 2021, reported bilateral CSDH with brainstem stroke following evacuation. They postulated the reason as Spontaneous intracranial hypotension with brain stem sagging and kinking of posterior circulation vessels or thrombosis in view of old age of patient despite no associated comorbidity apart from old age.[9]

The possible mechanisms responsible for PCA territory infarct following CSDH evacuation and resulting blindness or brain stem stroke are many including transtentorial herniation at presentation due to raised increased intracranial pressure, thrombosis of posterior circulation vessels due to various causes, spontaneous intracranial hypotension etc.

However, in our series of case reports, we postulate it possibly due to:

- Transtentorial herniation particularly in case 2 who presented in unconscious state but day 2 NCCT scan contradicts this possibility
- Thromboembolism of posterior circulation vessels may be the possibility but normal angiogram, lipid profile, 2 D echo precludes this possibility
- Spontaneous intracranial hypotension may be the cause specially in case 2 and case 3 in view of appearance of new hyperdensity in case 2 on day 5 scans and spontaneous SDH in case 3, history of headache in both cases, deterioration after initial improvement in case 2. No sagging on MRI goes against this possibility and
- PRES may be one of the possibilities in first case in view of MRI findings, history of hypertension but normal EEG, no response to steroids is not in favor of this possibility.

Limitations of the study

The limitation of the present study was small sample size. Also, the study did not exclude patients with other associated injuries in addition to brain injury.

CONCLUSION

CSDH is a frequently encountered problem in neurosurgical practice. CSDHs are viewed as "common lesions that are easily treated with a minimal morbidity and mortality," as Gelabert-Gonzalez et al., have noted. However, bilateral CSDH should be handled more carefully because it is a distinct entity with a different etiology and dysfunctional cerebrovascular autoregulatory mechanisms. For bilateral CSDH, early and simultaneous decompression is advised to avert fast deterioration and neurologic consequences. Blindness after bilateral chronic subdural hemorrhage is an extremely uncommon consequence that serves as further evidence that bilateral chronic subdural hemorrhage is not a benign condition.

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ETHICAL COMMITTEE APPROVAL

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Authors Contribution:
RRS- Definition of intellectual content, literature survey, prepared first draft of manuscript, implementation of study protocol, data collection, data analysis, manuscript preparation and submission of article**, PP-** Concept, design, clinical protocol, manuscript preparation, editing and revision; **RG-** Review manuscript, preparation of tables and graphs, coordination and manuscript revision, **CA-** Review manuscript, preparation of tables and graphs, coordination and manuscript revision.

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