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Cortical blindness and brain stem ischemia following burr hole evacuation of chronic subdural hematoma. A retrospective analysis of five cases

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Keywords
hematoma, chronic subdural, cortical blindness, spontaneous intracranial hypotension, brain stem infarction

ABSTRACT

Background: Chronic SDH is commonly performed in neurosurgery. The surgery usually has good surgical outcomes. There have been few cases reports reporting cortical blindness and brain stem stroke as a dreaded complication of the same.

Materials and methods: It is a retrospective, observational, descriptive type of study with analysis of patients operated for chronic SDH from March 2021 to March 2023 and reported an untoward outcome of vision loss or brainstem stroke following surgery. The cases have been reported with possible aetiology underlying the unfavourable outcome. The literature was reviewed for similar cases and a comparative analysis was done with possible hypotheses for the outcome.

Observation and results: An overall 5 such cases have been reported during this period. Two cases of bilateral and three cases of unilateral chronic SDH have been reported. Out of five, two cases did not have a preceding history of trauma probably spontaneous in nature. Possibility of PRES in one case, transtentorial herniation, thrombosis, and spontaneous intracranial hypotension with kinking of vessels have been proposed. Further studies are required to address this untoward complication following evacuation of chronic subdural hematoma.

Conclusions: A high index of suspicion, prompt recognition and management of reversible causes, slow decompression of hematoma especially in bilateral cases, and good hydration to prevent the possibility of thrombosis are key to preventing such complications. Also keeping the possibility in mind add on better counselling and prognostication of case in pre-surgery period.

INTRODUCTION

Chronic SDH is one of the most encountered emergencies in Neurosurgery practice. The chief management strategy involves surgical intervention although the type of surgery performed may vary

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with variability in approach. The incidence of disease is 1-5.3 cases per 100000 population, usually disease in unilateral but may involve bilateral side in one in five cases [1][12]. The possible etiology involves trauma most commonly, coagulopathies, antiplatelet use, intracranial hypotension (spontaneous or iatrogenic) etc. The usual prognosis after surgical evacuation is usually good. The common adverse outcomes described in literature includes rebleed, recurrence and requirement of redo surgery [8][11]. However there have been case reports in literature citing bilateral cortical blindness, neurological deterioration because of brain stem ischemia following successful evacuation of CSDH.

We here report a retrospective study of five such cases at our institute and discuss the possible basis of such an adverse outcome following optimal surgical management of this common entity.

Materials and methods

This is a retrospective, descriptive, observational study carried out at our tertiary care institute with analysis of patient's data who underwent surgery for chronic SDH and had an untoward outcome in form of vision loss or neurological deterioration with brain stem stroke following surgery; operated from March 2021 to May 2023.

All the patients were operated with similar technique involving two burr holes and hematoma evacuation depending on side involved with irrigation and putting subgaleal drain to reduce chances of recurrence. All patients were put on high flow oxygen for next 24 hours with being supine with head at 30 degrees propped up. Intravenous fluids continued till three days post surgery at 2000-2400 ml per day. Patients were discharged on day 4 as per clinical condition with repeat NCCT brain only if required according to patients' clinical status.

The data so observed were retrospectively analyzed and possible hypothesis for the etiology involved have been proposed. The literature was reviewed for similar case reports and series and our findings were compared with those previously reported in literature.

Observation and results

Representative Case 1

A 52-year-old alcoholic male patient with history of hypertension for last fifteen years taking on and off medications for the same, presented with chief complaints of severe holocranial headache for last seven days, altered sensorium for last 24 hours. There had been a history of road traffic accident one and a half month back. No history of any other comorbidity, not on any anti platelets or anti-coagulant drugs. Upon examination, pulse was 64/min, blood pressure was 160/98 mm Hg, patient was drowsy, pupils equally reacting to light. NCCT brain was suggestive of bilateral frontoparietal chronic SDH (figure 1a).

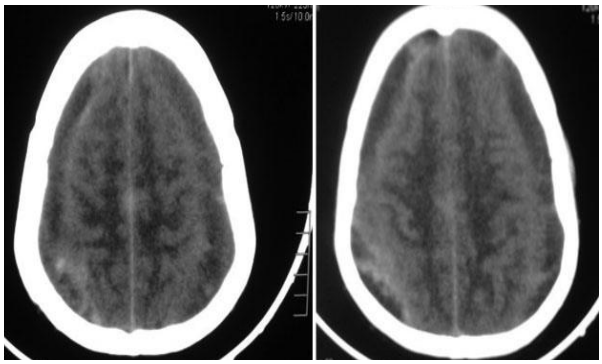

Figure 1a. Bilateral frontoparietal Chronic SDH.

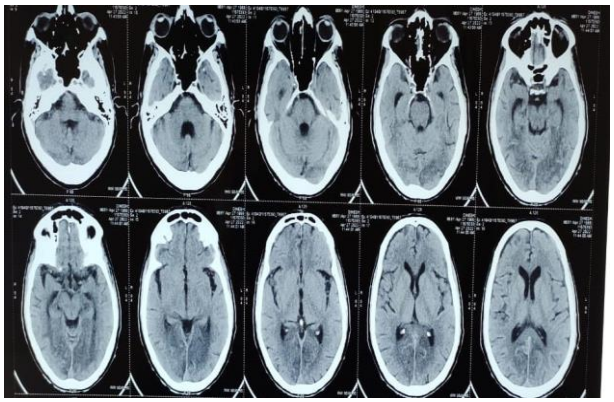

Figure 1b. Post surgery bilateral occipital hypodensities.

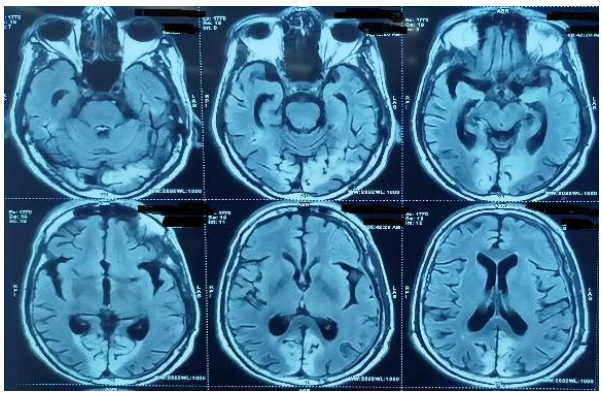

Figure 1c. MRI brain suggestive of T2 FLAIR hyperintensities but no corresponding diffusion restriction.

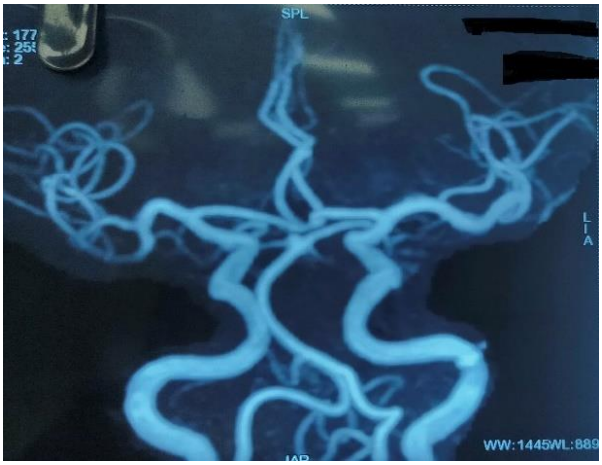

Figure 1d. MRA brain suggestive of no vessel cut off or compromise.

The patient underwent surgery as per protocol and symptomatically improved. Post operative management was also according to our standard practice. Patient was discharged on day 4 of surgery. Upon follow up in OPD on day 8 of surgery, patient was happy with improved symptoms but his relatives complained of difficulty in reaching to and holding the things when passed on to the patient. Although patient denied any diminution of vision.

Patient was readmitted and an NCCT brain was done which revealed good removal of hematoma but hypodensity in bilateral occipital region (Figure 1b). Ophthalmology work up revealed normal fundus findings but no perception of light in both eyes. An MRI brain with MRA brain was done which was suggestive of T2 FLAIR hyperintensities located corresponding to those in NCCT but there was no diffusion restriction and normal ADC findings (Figure 1c). MRA brain suggestive of no vessel compromise (Figure 1d). Blood investigations including lipid profile were normal and 2 D echo was also normal.

Upon meticulous review of previous admission record, an episode of hypertension to the range of 190/110 mm Hg during surgery was noted which was managed with intravenous labetalol and other anesthetic agents. We presumptively suspected a diagnosis of PCA territory stroke or posterior reversible encephalopathy syndrome (PRESS). An EEG was done but it was also normal recording. Patient was started with steroids with strict blood pressure syndrome and an anti-convulsant but there was no improvement in patient vision. In follow up visits, patient was later aware of his vision loss.

Representative Case 2

A 48-year-old chronic alcoholic male with no known comorbidity and history of fall one month back under alcohol influence presented to emergency room with history of headache on and off for last one week and history of loss of consciousness and altered sensorium for last 12 hours.

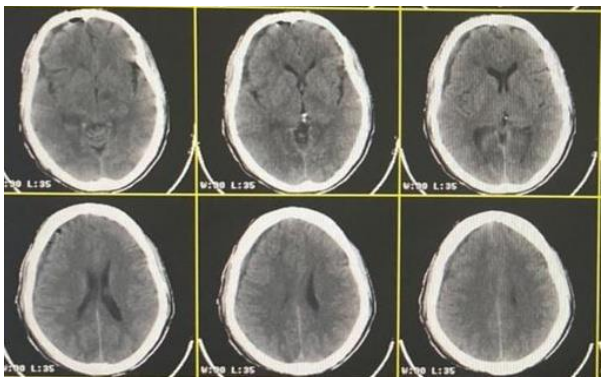

Figure 2a. Post Op day 2 NCCT Brain showing resolution of mass effect.

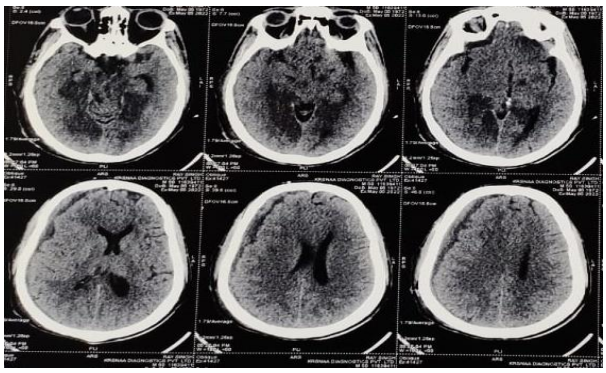

Figure 2b. NCCT on day 5 after deterioration; hypodensities in B/L PCA territory.

Upon examination vitals were stable, patient was unconscious with a Glasgow coma score of 5

(E1VetM4) with NCCT brain suggestive of right fronto temporoparietal chronic subdural hematoma with mass effect. Patient underwent burr hole and hematoma evacuation surgery wit irrigation and subgaleal drain as per standard protocol. Post surgery phase, patient improved in symptoms, weaned off from mechanical ventilator and extubated on day 2 of surgery with following simple commands. A check NCCT brain on day 2 (Figure 2 a) suggestive of resolution of mass effect and satisfactory removal of hematoma. On day 5 of surgery, there was dip in consciousness again with need to reintubate the patient. A NCCT brain post deterioration, suggestive of hypodensities in PCA territory involving brain stem and an increase in hyperdensity along right frontotemporoparietal convexity as compared to day 2 post surgery scan (Figure 2 b). An MRI brain with MRA brain and neck vessels, suggestive of T2 FLAIR hyperintensity with diffusion restriction in PCA territory involving brain stem suggestive of acute infarct (Figure 2 c). Other routine investigations and 2 D echo was normal.

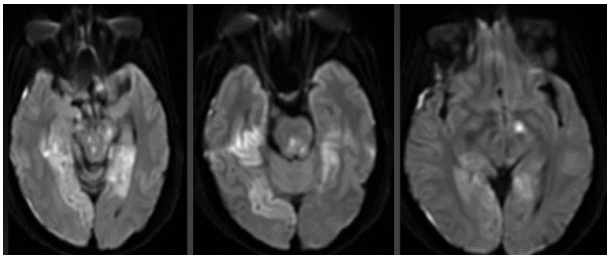

Figure 2c. Diffusion restriction along B/L PCA territory.

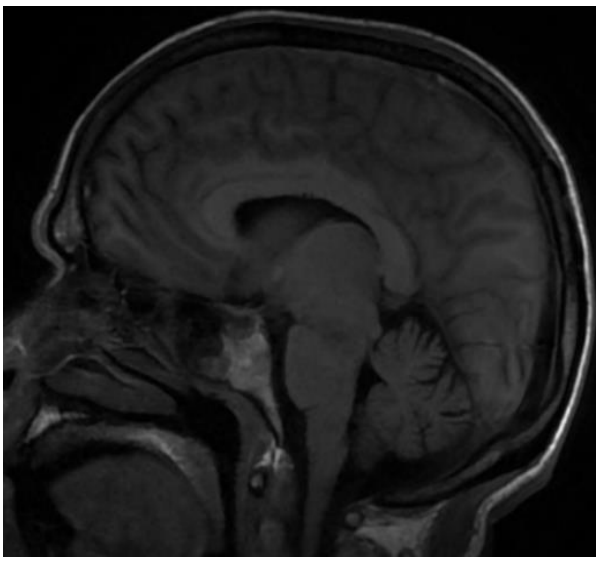

Figure 3. No sagging of brain in coronal cuts.

Representative case 3

A seventy-year-old male with known case of Alzheimer's disease and known diabetic for last 10 years, presented to us in altered sensorium for last 48 hours with history of repeated falls. Upon examination, vitals were stable, pupils bilateral reacting, GCS of 6 (E2VetM4). Blood sugar levels were 154mg%. NCCT brain suggestive of bilateral frontoparietal chronic SDH with no mass effect on either side. Patient was taken to OR and underwent surgery under standard protocol as per our practice. Patient improved in post surgery phase on day 1. There was spontaneous eye opening, with patient localizing pain and on oxygen support. Patient was planned for extubation but patient deteriorated same evening and repeat NCCT brain was suggestive dense hypodensities in B/L occipital lobes and brain stem. Patient was put on mechanical ventilator and succumbed on day 4. MRI brain could not be done in view of unstable clinical status of patient.

Representative case 4

A 45-year-old known alcoholic male with no history of any previous trauma admitted with complaints of sudden loss of consciousness for last 8 hours. The patient had a history of being conservatively managed for a spontaneous right frontotemporoparietal subdural hematoma at outside hospital one month back. NCCT on admission showed a chronic SDH with mass effect and ventricular effacement. Patient underwent surgical evacuation as per our protocol. Patient improved in post-surgery phase well. Patient was discharged on day 5 of surgery. Patient reported in follow up visit again on day 13 post surgery with complains of vision loss in bilateral eye. MRI brain was suggestive of bilateral occipital lobe acute infarcts. Ophthalmology examination was essentially normal apart from no perception of light in both eyes.

Representative case 5

A 50-year-old male with no preceding history of trauma but history of chronic alcoholism presented with inability to speak and right-side weakness for last 15 days. NCCT brain was suggestive of left Frontoparietal chronic SDH; underwent surgery and improvement in post surgery period with uneventful immediate post-surgery period. On day 4 patient reported diminution of vision in both eyes, patient

fundus was normal, pupils were reacting to light but patient was not able to follow light on examination. Patient was advised further admission stay and further work up but patient and his relatives were not willing for same and gave negative consent for same. The patient went on discharge against medical advice.

Discussion

The usual complications described in literature following CSDH surgery include seizures, infection, pneumocephalus, recurrence and rebleeding [8] [11] [12]. There have been few case reports only discussing possibility of bilateral PCA territory stroke leading to acute neurological deterioration of vision following surgery of chronic SDH. According to Balasubramanian et al in 2017 [1] only four cases had been reported so far having blindness following chronic subdural hematoma evacuation. Table 1 enlists details of cases reported so far having vision loss or bilateral PCA territory stroke following chronic SDH evacuation with possible explanation of findings in all the cases as per the respective authors of reports.

Table 1. Cases of vision loss or brain stem stroke reported in literature following chronic SDH evacuation with possible mechanism for the same.

S.No.	Case Report/Series	Case Details	Outcome	Possible explanations as per the author
1)	Kaene [6]	Three cases; 2 unilateral 1 bilateral	Bilateral vision loss; anterior pathways as well as occipital cortex both were affected	Trans tentorial herniation
2)	Russeger [9]	One case, Unilateral	Bilateral blindness	Altered vasoregulation around optic nerve due to sudden drop in intracranial pressure during decompression
3)	Kudo etal [7]	Two cases, unilateral	Bilateral occipital lobe infarct	Central trans tentorial herniation
4)	Balasubramanian etal [1]	Single case, bilateral	Bilateral blindness	Preexisting chronic ischemia led to chronic SDH; Evacuation led to worsening of ischemia leading to PCA infarction
5)	D. Adam etal [2]	Single case, Bilateral	Anton Babinski syndrome	No possible explanation mentioned
6)	Imoumby etal [4]	Single case, bilateral	Brain stem stroke	Spontaneous intracranial hypotension following evacuation leading to brain sagging and kinking of bilateral PCA arteries. Another possibility of thrombosis was also postulated as a cause of PCA infarct.

To the best of our knowledge, current series is the only reported series of multiple cases reporting bilateral PCA territory stroke following surgery for chronic subdural hematoma. In our series we propose following possible explanation of blindness or brain stem stroke following successful evacuation of hematoma:

1) Tran tentorial herniation, especially in case 2 and case 3 of our series looking at poor GCS at arrival and rapid deterioration. Absence of any PCA territory hypodensity in pre surgery and

immediate post surgery scan (case 2) goes against this possibility.

2) Thromboembolic phenomenon in posterior circulation may be one of possible cause leading to bilateral PCA infarcts. Again, normal lipid profile, no atherosclerosis in neck vessels in angiogram and unremarkable 2 D echo goes against this hypothesis. Although in case 3, looking at old age and as patient died in post surgery phase, we could not get a complete work up in view of unstable condition of patient; thrombotic phenomenon may a likely event.
3) Spontaneous intracranial hypotension may also be one of the plausible causes. In case 2 there has been a deterioration after decompression noted after initial post-surgery CT, case 3 old age with Alzheimer's, case 4 and case 5 being cases of spontaneous subdural hematomas more point toward possibility of intracranial hypotension leading to kinking of posterior circulation vessels against tent and resulting ischemia. Absence of any brain sagging in any of the MRI picture in coronal cuts (figure 3) contradicts this theory.
4) Posterior reversible encephalopathy syndrome (PRES) may be one of the explanations in our case 1. Patient was a known case of hypertension with an episode of hypertension noted during surgery, MRI findings support this hypothesis. Normal EEG recordings and no improvement with steroids disagree this.

Like case 1 reported in this series, J manual et al [5] have reported a case of cortical blindness arising in a patient of posterior reversible encephalopathy syndrome. Although as the cause was identified at the same time, prompt management in that case resulted in improved vision in the patient which was not the case in our patient.

We have reported this series highlighting the undesired and unforeseen complications of what is considered a simple and commonly encountered neurosurgery emergency. The possible explanations for the cause; highlights the importance of slow gradual decompression to reduce chances of sudden intracranial hypotension, maintaining a good hydration perioperatively to reduce chances of any thromboembolism, prompt diagnosis and management of reversible causes like PRES and a high index of suspicion for such unlikely complications in selected cases which might help in better pre operative counselling and case prognostication.

At the same time, we emphasize here for a large scale multicentric study for reporting of these untoward complications and enlightening of underlying mechanisms.

Conclusions

Chronic subdural hematoma evacuation is a routinely performed procedure. The high frequency of cases and simpler techniques at time underestimates the complications associated. There should be consideration of all precautions to prevent such complications and the possibility should be discussed in pre operative counselling and prognostication. More studies are warranted to address these complications further.

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