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Paediatric traumatic chronic subdural hematoma – a very rare entity. Single institution study of 5 cases

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ABSTRACT

Objectives: To study the prevalence of Traumatic chronic subdural hematoma in children.

Material and methods: This is a prospective study conducted at a tertiary care centre at Gwalior, from November 2020 to November 2021. We encountered 5 patients admitted for signs of raised intracranial tension due to Head trauma, showing Chronic subdural Haematoma (CSDH) in plain CT Head or MRI brain. All patients had no history of loss of consciousness, no history of vomiting, no history of seizures, no history of nasal or ear bleed and GCS was 15/15. All blood investigation and coagulation profiles are normal. Frontal and parietal 2.5 cm trephine craniotomy with evacuation of subdural hematoma was done. Patients were followed up at 1 month and 6 months

Result: All children are male. Age ranging from 7 years to 14 years, Mean age of presentation was 10 years. All patients had a history of head trauma and mode of injury road traffic accidents (RTA) and Glasgow coma scale (GCS) at admission was 15 and duration of developed CSDH was 20 days to 42 days'(mean30.40 days). All patients had good outcomes and the average follow-up was 6.5 months. All patients went on Surgical intervention. and outcome assessed by Glasgow Outcome score. 3 out of 5 patients had brownish fluid as a collection in subdural space while the other 2 patients had fluid that has motor oil like consistency in subdural space. All the patients were subjected to a similar procedure. The postoperative course was uneventful. Mean hospital stay was 7 days. A postoperative scan was done on the 7th day.

Conclusion: Chronic Subdural Hematoma is a disease reported in infants and the elderly population. It is uncommon in the age group of 2-14 years. Most of the children present with symptoms of raised intracranial tension due to head injury, with no reported history of repeated head trauma, child abuse & coagulopathy. Traumatic Chronic subdural collection should be considered as one of the diagnoses as the prognosis of this disease is better. However, due to the scarcity of reports in the literature, established guidelines are not available.

Keywords
traumatic,
subdural hematoma,
paediatric

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INTRODUCTION

Subdural hematoma is the collection of blood on the cortical surface beneath the dura with bleeding from bridging veins or cortical arteries. The term Chronic subdural Haematoma (CSDH) is often applied to

these collections, although the content of the accumulation may vary from thin, watery fluid resembling cerebrospinal fluid to thick "motor oil" often associated with adult CSDH.

The pathophysiology and clinical outcomes of head trauma differ between children and adults.

Blood vessels are absent in the normal dura-arachnoid interface. Neo-vasculature is abundant, but just in the outer CSDH membrane.

Abnormal dilated sinusoids measuring as large as 1000 micrometer, with an incomplete basement membrane and attenuated endothelial cells, share the outer membrane with rapidly growing microcapillaries. Both vessel types are composed of endothelial cells with irregular surface because of numerous pseudopod-like structures extending into vascular lumen. Erythrocyte and platelets in various stages of degeneration are frequently found deposited in the perivascular space. These sinusoids contain gap junction as large as 8 micrometer, sufficient to allow leakage of plasma and even red blood cells into the hematoma cavity.

Inflammatory mediators present in CSDH fluid may potentiate chronic rebleeding of the fragile Neovasculature. Kallikrein, bradykinin, and platelet-activating factor (PAF) have all been identified at significant level in CSDH fluid.

These inflammatory mediators stimulate vasodilation, increase vascular permeability, prolong the clotting time, and release tissue plasminogen activator (t-PA) from endothelial cells. Other work has focused on disturbances of the prostaglandin system as possible components in the pathophysiology [1].

Eosinophil degranulation in the outer membrane may be the source of fibrinolytic factors and inflammatory mediators causing local coagulopathy and cell destruction in the CSDH [2]

The traumatic mass lesions such as subdural and epidural hematomas occur less frequently in children and when present, are associated with lower mortality.

Common manifestations of CSDH are altered mental state and focal neurological deficits [3]. Subdural hematomas (SDH) are associated with an increased morbidity and mortality and generally occur as chronic SDH among older patients [4].

The most frequent signs and symptoms like headache, alteration of consciousness, gait impairment and hemiparesis are also seen among other CNS diseases, so they have to be considered during evaluation. In the case of symptomatic patients with focal neurological deficits surgical intervention should be considered, where as in case of asymptomatic patients or patients with only slight headaches conservative treatment with clinical and radiological follow-up might be a possibility. Also after surgical intervention, the recurrence rate is between 5 and 33% [4].

CSDH are much more common in infants because of associated traumatic deliveries and frequently exist as a single entity. It is rare for chronic subdural fluid accumulations to occur after one year of age and they are more frequent during adolescence [5,6]. Specific traumatic events are usually unrecognized

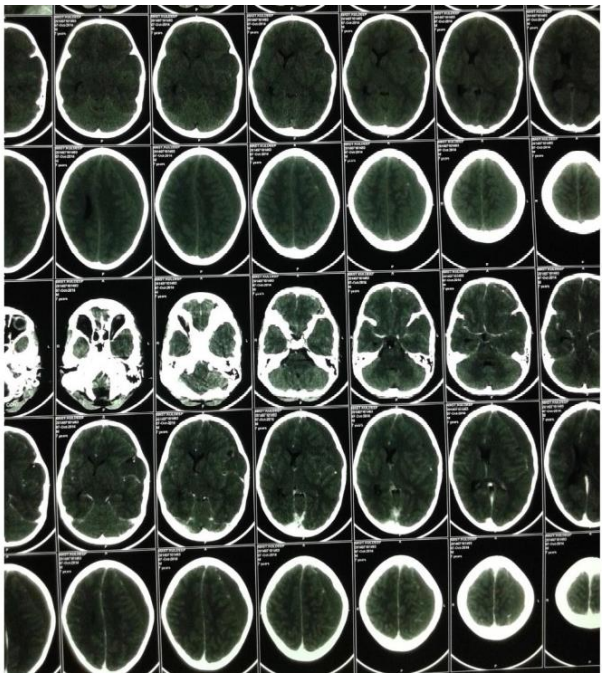

Figure 1. NCCT head revealed chronic subdural haematoma in Left Fronto-temporo-parietal convexity with midline shift toward right side with cisternal effacement.

Materials and methods

This is prospective study conducted at a tertiary care centre at Gwalior, from November 2020 to November 2021. We encountered 5 patients admitted for signs of raised intracranial tension who on radiological examination were found to have hemispheric subdural collection. All patients had No history of loss of consciousness, no history of vomiting, no history of seizures, no history of nasal or ear bleed and GCS was 15/15. All blood

investigation and coagulation profile normal. Frontal and parietal 2.5 cm trephine craniotomy with evacuation of subdural hematoma was done. Patients were followed up at 1 month and 6 months.

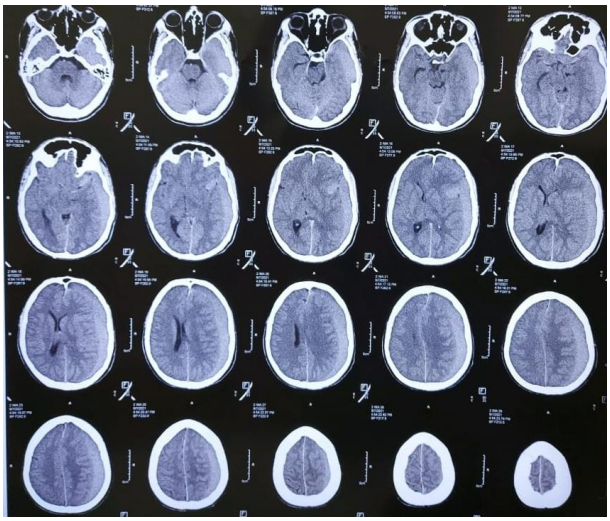

Figure 2. NCCT head revealed chronic subdural haematoma in Left Fronto-temporo-parietal convexity with midline shift toward right side with cisternal effacement.

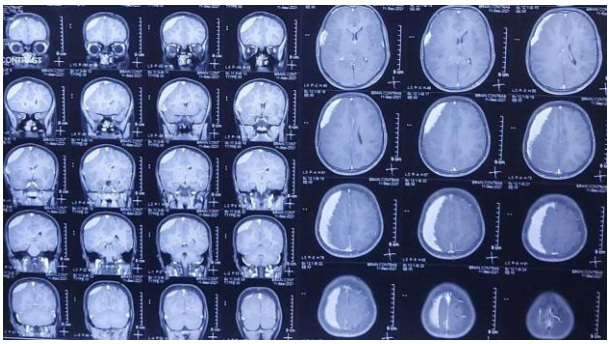

Figure 3. MRI Brain revealed chronic subdural haematoma in Right Fronto-temporo-parietal convexity with midline shift toward right side with cisternal effacement.

Results

All children are male. Age ranging from 7 years to 14 years, Mean age of presentation was 10 years. All patients had history of head trauma and mode of injury road traffic Accidents (RTA) and Glasgow coma scale (GCS) at admission was 15 and duration of developed CSDH was 20 days to 42 days'(mean30.40 days).. All patients had good outcome and average follow-up was 6.5 months. All patients went on Surgical intervention [Table-1] and outcome assessed by Glasgow Outcome score [Table-2].

Table 1. Demographic data, management and outcome of patients

Age (year)	sex	Mode of injury	GCS at the time of Admission	Management	Outcome measure by GOS
14	Male	Road traffic Accident (RTA)	15/15	Surgery	Good
11	Male	RTA	15/15	Surgery	Good
10	Male	RTA	15/15	Surgery	Good
8	Male	RTA	15/15	Surgery	Good
7	Male	RTA	15/15	Surgery	Good

Table 2. Glasgow Outcome Scale

GOS	Functional Status
5	Resumption of normal life; there may be minor neurological and Psychological deficit
4	Able to work in a shattered environment and travel by public transportation
3	Depend on daily support by reason of mental and physical disability or both
2	Unresponsive for weeks or month or until death
1	Death

Table 3. Description of Previous reported case of Traumatic Pediatric Chronic SDH

Author	Year	No. of case	Age/sex of patient	Description of patient
Osaka H et al.	1993	1	10 month/male	Gluteric Aciduria type-1
Narsinghani et.al	2002	1	5 year/Male	strikingly large calcified CSDH in a 5-year-old child with increased intracranial pressure and subfalcine herniation
Mori et al.	2002	12	All patients are paediatric age group	Chronic Subdural Haematoma with Arachnoid cyst
Acakapo satchivi et al.	2007	1	4 month/male	Macrocrania with Chronic Subdural Haematoma

Kumar et al.	2008	20	Range 1 month – 2year	Chronic Subdural Haematoma following minor head injury
Wang et al.	2010	1	1 year/male	Chronic Subdural Haematoma due to repeated minor Dodgeball head injury
Vivek kankane et.al.	2015	1	7 year/male	Chronic Subdural Haematoma due fall from bed 3 month ago

Discussion

Wang, et al. reported a rare case of CSDH in a 9-year-old child due to repeated minor dodgeball head injuries in 2010 [Table-3].

Although such a case has never been reported in sport. No altered mental state or focal neurological deficits were observed; the child presented with intermittent severe headache with nausea and vomiting. There was also no evidence of child abuse; however, the history of repeated minor head injuries during playing was significant [3].

Kumar et al. reported twenty cases of traumatic subdural empyema (SDEs) following minor head injury or unreported. In rare instances, SDH may indicate an underlying bleeding disorder, hematological malignancy or benign expansion of subarachnoid in 2008. The age of these children ranged from 1 month to 2 years. 70% children presented with subtle findings, 30% children presented with overt neurological signs and symptoms. Seizures were the most common mode of presentation.

Bilaterality and ventriculomegaly were more common in the subtle group, each with an incidence of 43%. 35% cases required operative management of traumatic SDEs. Recurrence was seen in 10% cases who had been conservatively managed previously. Only one child showed conversion of traumatic subdural hygroma to CSDH on conservative management [7]

Narsinghani et al. reported a case the dramatic presentation of a strikingly large calcified CSDH in a 5-year-old child with increased intracranial pressure and subfalcine herniation in 2002 [5].

Mori et al. reported that 12 patients with CSDH and arachnoids cyst were significantly younger than the patients with CSDH without arachnoid cyst in 2002. The most frequent symptom was headache followed by vomiting in the patients with arachnoid cyst, while gait disturbance and hemiparesis predominated in patients without arachnoid cyst. CSDH formation may be preceded by subdural hygroma caused by the rupture of arachnoid cyst [8].

In 2007, Acakapo satchivi et al. reported an unusual case of cortical herniation into a CSDH. A 4-month-old boy with a history of Macrocrania and very large bilateral chronic SDHs underwent subduro-peritoneal shunt treatment shortly after presentation.

To the authors' knowledge, this is only the fourth report in the medical literature of cortical herniation through a chronic subdural membrane and the first in which successful treatment with a good outcome is described [9].

CSDH in infant after abusive head trauma is a serious form of child abuse that can lead to severe neuropsychological sequelae or death in infants [10].

Osaka H, et al. reported a 10-month-old male with glutaric aciduria type-1 (GA-1) in 1993. This patient showed frequent partial motor seizures, irritability, and involuntary movements, including oral dyskinesia at the age of 3 months. On admission, magnetic resonance (MR) scanning revealed a chronic subdural hematoma and widening of the bilateral insular cisterns. Urine organic acid analysis showed marked excretion of glutaric acid, 3-hydroxy glutaric acid and glutaconic acid, suggesting GA-1 [11].

New pathophysiologic aspect might have an impact on conservative treatment in future. In particular, detection of the angiogenic cytokines responsible for development of the well-known leaky vessels within the outer membrane of a hematoma might offer new and promising targets to be blocked by pharmacologic agents.

Recently, it was shown that the angiogenic properties of angiotensin converting enzyme inhibitors could reduce the rate of recurrence in CSDH, as well as level of vascular endothelial growth factor within the hematoma [12,13].

Subsequent studies have identified lower levels of all coagulation factors in CSDH fluid than plasma. Factor II, V, VII, VIII, and X are disproportionately depleted. These findings reflected a phase of accelerated fibrinolytic activity after the rapid and defective clot formation. The end result is a milieu of anticoagulant protein (chiefly FDPs) and depleted coagulation factors. Other author have suggested that the PAF derived from lysis of red blood cells may stimulate the synthesis and release of t- PA, as well as induce chemotaxis of inflammatory cells to the CSDH fluid and elevated plasma level of PAF in patient with CSDH versus healthy volunteers. The latter observation may suggest a systemic predilection to the development of CSDH.

Conclusions

Chronic Subdural Hematoma is disease reported in infants and elderly population. It is uncommon in the age group of 2-14 years. Most of the children present with symptoms of raised intracranial tension due to head injury, with no reported history of repeated head trauma, child abuse & coagulopathy. Chronic subdural collection should be considered as one of the diagnoses as prognosis of this disease is better. However due to scarcity of reports in literature, established guidelines are not available.

References

1. Hirashima Y, Endo S, Kato R, Ohmori T, Nagahori T, et al. (1994) Plateletactivating factor and development of chronic subdural haematoma. Acta Neurochir 129: 20-25.
2. Muller W, Firsching R (1990) Significance of eosinophilic granulocyte in chronic subdural hematomas. Neurosurge rev13: 305-308.
3. Wang HK, Chen HJ, Lu K, Liliang PC, Liang CL et al. (2010) A pediatric chronic subdural hematoma after dodgeball head injury. Pediatr Emerg Care. 26: 667-668. 4.
4. Gautschi OP, Gallay MN, Kress TT, Korte WC, Hildebrandt G (2010) Chronic subdural hematoma -assessment and management. Praxis (Bern 1994).
5. Narsinghani U, Burson G, Schmidt MB, Anand KS (2002) Case of the month. Symptomatic chronic calcified subdural hematoma in a 5-year-old child. J Ark Med Soc 98: 300-303.
6. Per H, Gümüş H, Tucer B, Akgün H, Kurtsoy A, Kumandaş S (2006) Calcified chronic subdural hematoma mimicking calvarial mass: a case report. Brain Dev 28: 607.
7. Kumar R, Singhal N, Mahapatra AK (2008) Traumatic subdural effusions in children following minor head injury. Childs Nerv Syst. 24: 1391-1396.
8. Mori K, Yamamoto T, Horinaka N, Maeda M (2002) Arachnoid cyst is a risk factor for chronic subdural hematoma in juveniles: twelve cases of chronic subdural hematoma associated with arachnoid cyst. J Neurotrauma 19: 1017-1027.
9. Acakpo-Satchivi L, Luerssen T (2007) Brain herniation through an internal subdural membrane: a rare complication seen with chronic subdural hematomas in children. Case report. J Neurosurg 107: 485-488.
10. Wuerfel nee Tysiak E, Petersen D, Gottschalk S, Gerling I, Gliemroth J, et al. (2012) Progression of chronic subdural haematomas in an infant boy after abusive head trauma. Eur J Paediatr Neurol 16:736-739.
11. Osaka H, Kimura S, Nezu A, Yamazaki S, Saitoh K, et al. (1993) Chronic subdural hematoma, as an initial manifestation of glutaric aciduria type-1. Brain Dev 15: 125-127.
12. Weighel R, Hohenstein A, Schlickum L, Weiss C, Schilling L (2007) Angiotensin converting enzyme inhibition for arterial hypertension reduces the risk of recurrence in patients with chronic subdural hematoma probably by an angiogenic mechanism. Neurosurgery 61: 788-792
13. Kankane Vivek, Jaiswal Gaurav and Gupta Tarun, A Pediatric Traumatic Chronic Subdural Hematoma: Rare Case Report and Review of Literature. J Spine Neurosurg 2015, 4:3.