Spontaneous resolution of Radiologically Significant Acute SDH – Analysis of 32 cases at a Tertiary Centre in India.

AUTHORS

1st Author - Dr. Gian Chand, Assistant Professor , Department of Neurosurgery, AIMSS , Chamiana , HP (India) E-mail: giandr007@yahoo.com
2nd Author - Dr. Biplav Singh , Assistant Professor , Department of Neurosurgery , AIMSS , Chamiana, HP (India) E-mail: biplavsingh.1@gmail.com.
3rd Author –Dr.VineetTanwar , Assistant Professor , Department of Neurosurgery , AIMSS , Chamiana, HP (India) E-mail: drvineet837878@gmail.com
4th Author –Dr. Dig Vijay Singh Thakur , Assistant Professor , Department of Neurosurgery , AIMSS , Chamiana, HP (India) E-mail: diguthakur@gmail.com
5th Author- Dr. Vikram Singh , Senior Resident , Department of Neurosurgery , AIMSS , Chamiana, HP (India) E-mail: drvikram.singh@icloud.com
Corresponding Author –Dr. Rahul Gupta, Senior Resident, Department of Community Medicine and School of Public Health, PGIMER. E-mail: drrahulforpublichealth@gmail.com

ABSTRACT

Objective: Most of the cases of radiologically significant acute subdural haematoma (SDH) require surgical decompression in emergency, but some proportion of these cases with higher GCS may undergo spontaneous rapid resolution when managed conservatively under monitoring. The main objective of this retrospective study was to identify the predictive factors leading to spontaneous resolution of post-traumatic acute SDH.

Methods: Retrospective analysis of the data of the patients with acute SDH admitted in our institute within the period from July 2022 to December 2022 was done . Patients with radiologically significant acute SDH (clot thickness more than 10 mm and midline shift more than 5 mm), who were initially managed conservatively due to various reasons such as poor general condition, advanced age, family wishes not to operate and other factors, were identified. Patients were divided into resolution and non-resolution groups, and comparison was done on the basis of various factors such as age, sex, initial GCS, Pupillary reaction, clot thickness, midline shift and low density band on initial CT.

Results: A total of 32 patients fulfilled the study criteria, 24 in resolution group and 8 in non-resolution group. We found three most significant factors determining rapid resolution of acute SDH: high initial GCS, low initial midline shift and presence of low-density CSF band on initial CT.

Conclusion: We found in our analysis that among the patients with radiologically significant acute SDH, the patients with higher initial GCS, comparatively low midline shift and presence of low-density band on CT may be the candidates of initial conservative trial.

KEYWORDS Radiologically significant acute SDH, spontaneous resolution, conservative management.

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INTRODUCTION

Acute SDH is one among the most lethal complications of head injury following Road-traffic accidents or other injury mechanisms. It accounts for 10-20% of all head trauma patients carrying a very high mortality rate of 50 to 85%[1]. It most commonly involves fronto-temporo-parietal convexity; other sites may be inter-hemispheric fissure and middle cranial fossa[2]. Acute SDH requires an impact of higher magnitude as compared to Acute Extradural Haematoma (EDH) and it is much more dangerous than acute EDH. The main mechanism of Acute SDH is disruption of the bridging vessel due to acceleration-deceleration of the brain inside the cranial cavity due to head trauma. Other major mechanisms of Acute SDH are accumulations around parenchymal lacerations, bleeding from sinuses or arterial bleed. These patients are symptomatic due to Mass effect of hematoma, midline shift, injuries to brain parenchyma and cerebral edema[3,4]. It may be further lethally modified by subsequent phenomena such as focal and global cerebral ischemia beneath the hematoma, reactive hyperemia-hyperperfusion, coagulopathy, delayed hematomas etc. Because of the complex nature of its pathophysiology, its mortality remains quite high despite advancements in diagnostic modalities and treatment techniques[5,6]. A GCS score of 8 or less is seen in 35 to 80% patients of Acute SDH and a “talk and deteriorate '' have been described in 6% patients of Acute SDH[7,8].

Guidelines given by Bullock MR et al[9] (2006) state that any acute SDH with a thickness greater than 10mm or a midline shift greater than 5mm on computerized scan should be surgically evacuated regardless of the patient’s Glasgow coma scale (Radiologically significant acute SDH). Whereas those with Acute SDH thickness less than 10mm or midline shift less than 5mm should undergo surgical evacuation if GCS drop 2 or more point from injury to admission, and/or pupils are asymmetric or fixed and dilated and/or ICP is more than 20 mm of hg. Regarding timing of surgery, “Four hour rule” was given by Seelig JM et al[10] (1981) in a series of 81 patients with Acute SDH, according to which patients operated within four hours of injuries had 30% mortality as compared to 90% mortality if surgery was delayed more than four hours.

In this study , we analyzed the data of the patients with radiologically significant Acute SDH with minimal, mild and moderate head injury. We reviewed the data of patients with radiologically significant acute SDH over the period of six months, who were managed conservatively due to various reasons and analyzed their outcome.

MATERIALS AND METHODS

We retrospectively analyzed the records of the patients from July 2022 to December 2022 presenting to our institute , suffering from head injury with Acute SDH.

Inclusion criteria were:

1. Age 18 years and above
2. Closed head injury patients.
3. Isolated head injury patients.
4. Patients reporting within 24 hours of head injury.
5. No patients with severe coagulopathy (platelets <100000, INR>1.4 or aPTT>50).
6. CT Head findings suggestive of Acute SDH thickness more than 10 mm and midline shift more than 5 mm with no associated contusions, SAH, cerebral edema and impending herniation.
7. Patients with GCS 10 or more.
8. No pupillary asymmetry on initial presentation.

Patients with radiologically significant acute SDH (Acute SDH clot thickness more than 10mm and midline shift more than 5 mm) identified,

who were initially managed conservatively due to various reasons such as poor general condition, advanced age, family wishes not to operate and other factors. A total of 40 patients were found having radiologically significant acute SDH fulfilling above mentioned inclusion criteria, which were being managed conservatively. We defined the spontaneous resolution of Acute SDH with the following criteria: 1. Neurological improvement of at least 1 point GCS in the first 24 hours and by 2 points in 48 hours. 2. Reduction of Acute SDH clot thickness more than 5mm within 96 hours of initial CT.

Patient data collected include age, sex, GCS, Pupillary response & vitals at presentation, hemoglobin, platelet count, INR, any use of anticoagulants or antiplatelets before head injury, clot thickness and midline shift on initial CT. These patients were conservatively managed on IV fluids, antiepileptics, osmotic agents, analgesics, antiulcer drugs, antiemetics, sedation and intermittent oxygen inhalation. Vitals, GCS, Pupillary response and intake-output recorded every 6 hours. Daily RFTs and serum electrolytes were sent. Non-contrast CT brain was repeated after 48 and 96 hours. Patients which showed signs of deterioration on conservative management were immediately operated upon.

OBSERVATIONS AND RESULTS

A total number of 1950 patients of head injury were admitted between July to December 2022, out of which 212 patients suffered from Acute SDH. Out of 212 patients, 10 patients died prior to any surgical intervention and 62 patients were managed surgically immediately after admission. And 140 patients were kept on conservative treatment after an initial survey in the emergency department. Out of 140 patients, 100 patients were having small thin SDH and only 40 patients were having radiological significant Acute SDH (Clot thickness more than 10mm and/or midline shift more than 5mm). Another 4 patients excluded from study, 3 below 18 years, and one with severe coagulopathy. 4 more patients were excluded from the study whose all CT head records could not be traced. Finally a total of 32 patients were included in our study (Table 1).

Acute SDH was present in 10.87% of total patients admitted with head injury. About 4.72% patients of acute SDH died prior to any surgical intervention, whereas about 30% of total Acute SDH patients required immediate surgical intervention. 47.17% of acute SDH patients had small thin SDH without any significant midline shift and were managed conservatively. Another 18.86% of acute SDH patients, although having radiological significant Acute SDH, were being managed conservatively, either because of poor general condition, advanced age or family wishes not to operate and other patients with mild symptoms and signs some of which were operated later on deterioration. After excluding 8 patients not fulfilling our study criteria, 32 patients were included in our study analysis. Statistical analysis was done by using MEDCALC easy-to-use-statistical software.

Spontaneous resolution of acute SDH was seen in 24 (75%) patients of our study group and eight (25%) patients deteriorated on conservative management, some of which operated later after their family members gave consent for surgery. There was no statistically significant difference in age and sex between Resolution and Non-resolution groups due to our study inclusion criteria. All of the patients in the Non-resolution group were male and 75% males in the resolution group. There was a statistically significant difference in the initial GCS of the two groups with P-value of 0.02. Higher GCS at initial presentation was likely an

indicator of early resolution as compared to the lower GCS. Pupillary asymmetry was noted in 7 (87.5%) cases of non-resolution group as compared to one (4.17%) in resolution group with p-value of <0.0001, highly suggestive of non-resolution in patients with pupillary asymmetry. There was no statistically significant difference in clot thickness in the two groups. There was a highly statistically significant difference in midline shift between two groups with p-value of 0.01. Higher midline shift was found to be associated with non-likelihood of resolution of acute SDH. Low density band around the SDH as an indicator of CSF in acute SDH was seen in 19 (79.17%) in resolution group as compared to 2 (25%) in non-resolution group with p-value of 0.006, which is highly specific for possibility of spontaneous resolution.

DISCUSSION AND REVIEW OF LITERATURE

Acute subdural hematoma with thickness more than 10mm and midline shift more than 5mm always warrants surgical evacuation regardless of GCS as per standard guidelines till date. But there are many case reports and institutional data showing spontaneous resolution of acute subdural hematoma more than 10mm thickness and 5 mm midline shift on conservative management, which happens between few hours to few days after head injury[11,12].

There are two possible mechanisms regarding spontaneous resolution of acute subdural hematoma: "CSF washout effect" through arachnoid tears[13] and "compression and redistribution" of hematoma[2]. In the first mechanism some cases of acute subdural hematoma are usually associated with arachnoid tears. These tears allow flow of CSF into subdural space which dilutes the hematoma and allows reabsorption back into subarachnoid space. In the second mechanism, increased ICP and cerebral swelling results in compression and redistribution of hematoma in inter hemispheric and cerebellar tentorium subdural spaces. And also accompanying dural tears and skull fractures may drain hematoma into inter-diploic and subgaleal spaces.

Some radiological signs have been suggested for likelihood of resolution of acute subdural hematoma. Presence of low density layer between hematoma and inner plate of skull is suggestive of CSF in subdural space is a favorable sign supporting our hypothesis of "CSF washout effect" through arachnoid tears[14] (Fig A1). Other signs may show beaking into sulcus or cistern.

Follow up CT scan of our patient at 72 hours demonstrated significant reduction in thickness of acute subdural hematoma (Fig C). Redistribution of blood to the cerebellar tentorium on the same side on follow up CT scan supports the hypothesis of "compression and redistribution"(Fig B).

Another theory of spinal migration of intracranial SDH was given. Bortolotti et al[15] first described it in a case of a 23 year old female of traumatic subdural hematoma with no spinal trauma findings. Most cases of migrating SDH to spinal canal in literature involve spontaneous SDH, although cases of traumatic SDH also have been reported. Li et al[16] reported a case of cranial SDH migrating into the spinal canal following a trauma in a 26 year old male. Kundra et al[17] gave another theory of spontaneous resolution and extra cranial redistribution of acute SDH. They stated that acute SDH gives rise to scalp hematoma by passing through dural tear or calvarial fracture with direct pressure on the soft tissue, thus exhibiting redistribution. Wu et al[18] stated that acute SDH volume less than 30 ml and distributed near the sylvian fissure and in the fronto-temporal or temporo-parietal resulted in the spontaneous resolution of acute SDH.

Cerebral atrophy[19,20] is also one of the factors which may facilitate accommodation and intracranial redistribution of acute SDH. Absence of cerebral contusions obstructing outflow of CSF and elasticity of brain[21] are also optimal requirements for rapid resolution.

CONCLUSION

All the patients of acute subdural hematoma, even with significant thickness and midline shift may not require craniotomy for evacuation of hematoma, depending upon their initial clinical presentation and further progression. We found in our analysis that among the patients with radiologically significant acute SDH, the patients with higher initial GCS, comparatively low midline shift and presence of low-density band on CT may be the candidates of initial conservative trial. It may avoid some unnecessary craniotomies.

CONFLICT OF INTERESTS

The authors declare that they have no conflict of interests.

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Table 1

	Resolution Group (n=24)	Non-resolution Group (n=8)	P-value
Age(years)	42.917±11.85	40.75±15.173	0.6790
Male Sex	18(75%)	8(100%)	0.2842
Initial GCS	12.04±0.999	11±1.195	0.0213
Pupillary asymmetry	1(4.17%)	7(87.5%)	<0.0001
SDH thickness (mm)	10.787±0.489	10.825±0.620	0.8598

Mid-line shift (mm)	7.667±1.384	9.125±1.026	0.0106
CSF in subdural space (Low density band)	19(79.17%)	2(25%)	0.006

Figure Legends: Fig(A1) NCCT Head showing Acute subdural hematoma along right cerebral hemisphere of 11.2mm thickness . Low-density band (black arrow Fig A1) of CSF can be clearly seen surrounding subdural hematoma. Fig(A2) showing 7.3mm midline shift . Fig(B) showing redistribution of subdural hematoma around the tentorium (black arrows). Fig(C) showing almost complete resolution of acute subdural hematoma on the third day.

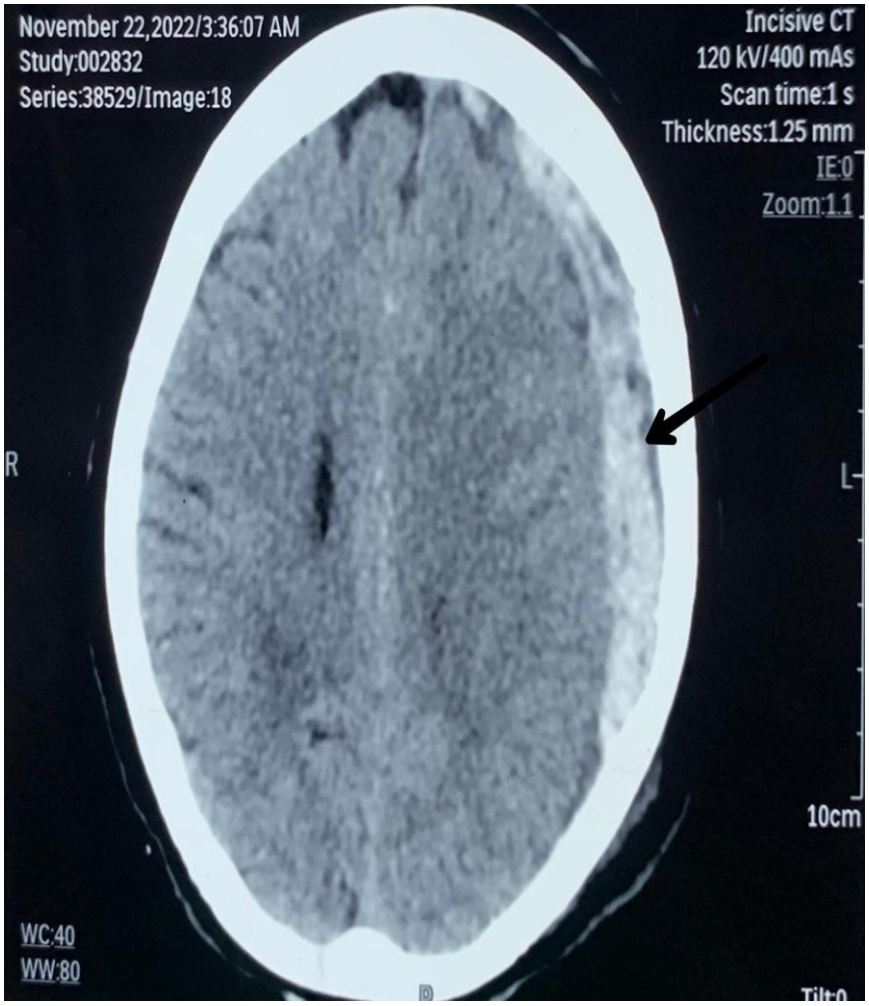

Fig A1

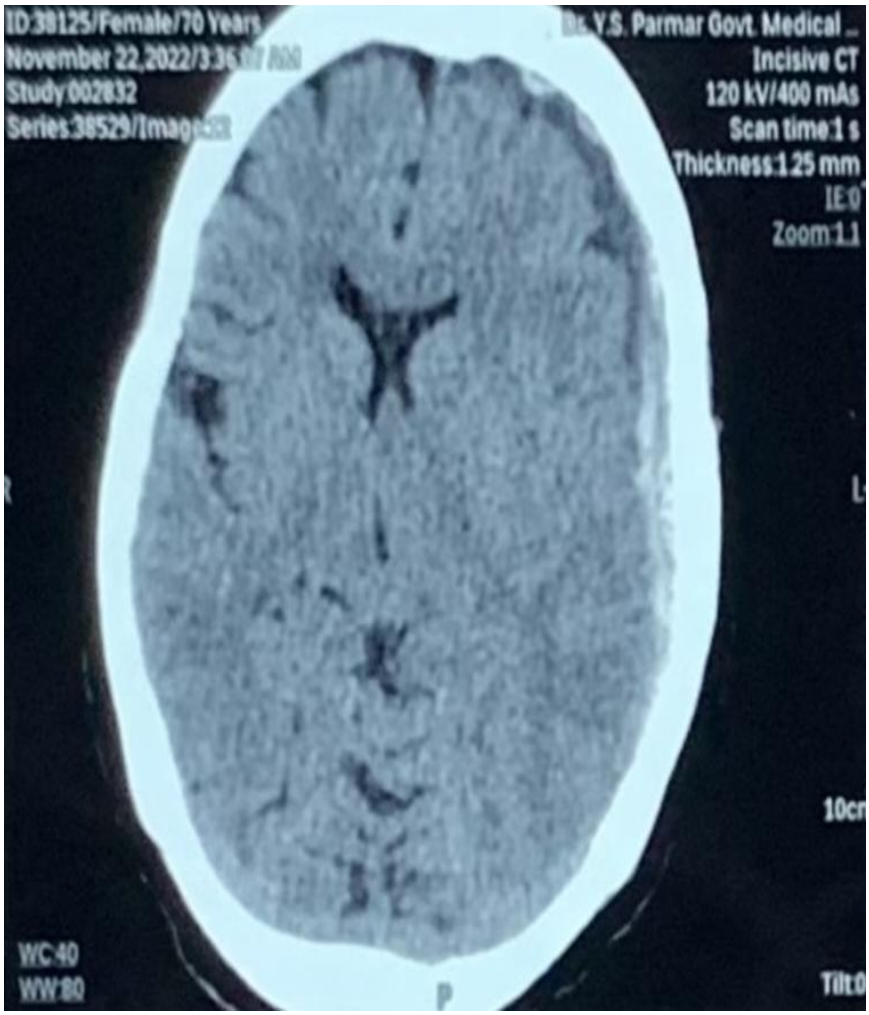
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Fig A2

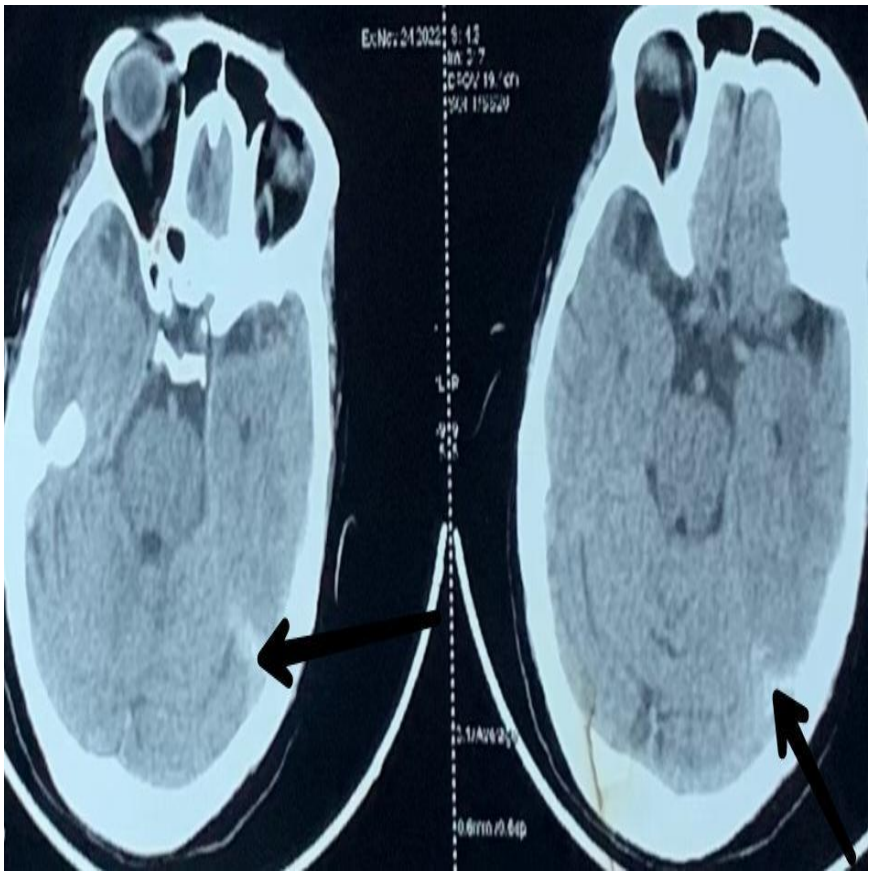

Fig B

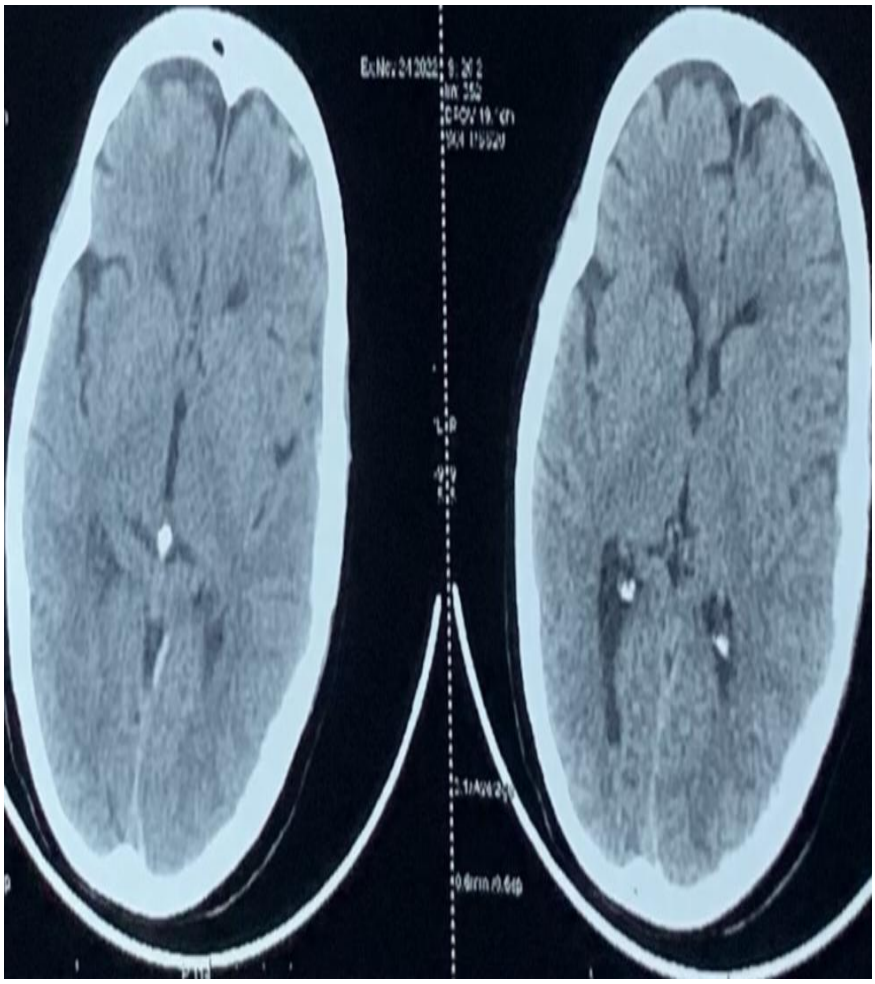

Fig C