Original Research Article

DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20170625

Clinical study of acute subdural haematoma – a level I trauma care centre experience

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Received: 09 February 2017 Revised: 12 February 2017 Accepted: 15 February 2017

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ABSTRACT

Background: Acute subdural hematoma is the most common type of traumatic intra cranial hematoma accounting for 24% cases of severe head injuries and caries highest mortality. The aim of this study is to analyse the clinical spectrum, and to evaluate the postoperative outcome.

Methods: This is a prospective observational Study which included 150 patients admitted in King George hospital, Andhra Medical College, Visakhapatnam, India over the past two years (01^{st} August 2013 to 30^{th} August 2015) with head injury, diagnosed to have Traumatic subdural Hemorrhage. A detailed clinical history, physical examination, CT scan was performed in all patients. Patients who were subjected to surgery, post-operative out-come were compared. **Results:** The maximum patients suffering from SDH are in the age group of 20-40 years (63%) with male predominance (73%). The most common mode of injury is RTA (73.33%). 40% of cases presented to hospital with gcs <8. Pupillary reaction, hypotension, CT scan findings i.e. thickness of hematoma >10mm and midline shift of >5mm, Basal cisterns obliteration, post traumatic seizure and delay in interval between the surgery had greatly affected on outcome of patients. Out of 150 cases, surgical approach was considered in 120 patients while remaining 30 patients were managed conservatively.

Conclusions: Interval between injury to surgery with interval <4 hours having favourable outcome. Basal cistern obliteration, which is an indicator of increased intra cranial pressure is the single most important factor for unfavourable outcome. Hypotension and post traumatic seizures influence high mortality.

Keywords: Glasgow coma scale, Glasgow outcome, Scale subdural hematoma

INTRODUCTION

Acute subdural hematoma is the most common type of traumatic intra cranial hematoma accounting for 24% cases of severe head injuries and caries highest mortality. The mortality rates are seen to be ranging from 30% to 90%.¹⁻⁴

John abernethy, a pupil of hunter described extradural and subdural hematomas.⁵ Burrows and jacobson also contributed to understanding of these lesions.^{6,7}

Incidence

Acute subdural hematomas accounts for 24% of severe head injury patients and the incidence of severe head injury is 21 per 100000 people.^{8,9}

Pathophysiology

Acute subdural hematoma occurs in of 3 mechanisms.⁴

- Damage to surface cortical vessel.
- Bleeding from underlying parenchyma injury.

• Tearing of bridging veins from cortex to dural venous sinuses.

Arbitrarily divided into 3 stages. i.e. Acute SDH-1 to 3 days from injury; Sub acute SDH- 4 to 21 days of injury; Chronic SDH -after 3 weeks of injury.

Associated brain injury

The chief factor that makes an acute subdural hematoma a serious condition is the frequency with which associated brain damage occurs. Such brain damage varies from simple sub pial haemorrhage to extensive laceration of brain. Cerebral edema and brain stem distortion often complicates the picture.

Site of hematoma

The common sites for the acute subdural hematoma are the inferior frontal, the anterior temporal and the parietal regions. Fronto polar and sub frontal hematomas, hematomas in the middle cranial fossa, over the occipital pole and hematomas of posterior fossa and in the inter hemispheric regions have all been encountered.

Clinical picture

Alteration in conscious level, gradual worsening in the level of consciousness is the classical presentation. A fluctuation level of consciousness may also be seen. Increasing restlessness is an important sign. Some evidence of localization is present in about 70% of cases. In the rest the localizing signs do not occur either because of associated lesions or rapid development of brainstem signs.

The onset of pupillary changes and hemiparesis points to the side of the lesion. With the introduction and wide availability of cranial CT (Figure 1), early diagnosis and timely surgical intervention for SDH is an attainable gold standard.^{4,10}



Figure 1: CT scan showing sub dural hematoma.

Many factors influence prognosis. When symptoms of subdural hematoma appear slowly the prognosis is better.^{4,10,11} There is also a direct correlation between the

level of consciousness and postoperative mortality.^{10,12} With the onset of decerebrate rigidity the mortality increases rapidly.^{2,13}

METHODS

This is a prospective observational Study which included 150 patients admitted in King George hospital, Andhra medical college, Visakhapatnam over the past two years (01st August 2013 to 30th August 2015) with head injury, diagnosed to have Traumatic subdural Hemorrhage. All are analyzed with respect to age, sex, mode of injury, GCS at the time of presentation, post resuscitation GCS, pupillary anisocoria, focal neurological deficit. hemodynamic status, imaging findings of thickness of hematoma, location of hematoma, extent of hematoma either focal or diffuse, midline shift, simple SDH or complicated SDH, status of basal cisterns, interval between injury to surgery, GOS at the time of discharge and at six months. Results are also compared with published literature.One hundred and twenty patients underwent surgery.

Criteria for surgical intervention are - a) All cases of acute SDH with thickness of hematoma of >1cm, b) more than 5mm thickness with mid line shift of >5mm, c) GCS<8, d) pupillary dilatation and focal neurological deficit corresponding to the hematoma. Standard fronto-temporo-parietal craniotomy with question mark incision and flap was employed in all cases.

Dura was wide opened and hematoma evacuated. Brain swelling occurred in 50 cases. Anti oedema measures with mannitol and hyperventilation resulted in subsidence of brain swelling in 30 patients. Duraplasty was done with pericranium in 15 patients, and bone flap replaced in 5 patients (Figure 2). Glasgow outcome scale assessment was done at the time of discharge and again at 6 months. The objectives of present study are:

- To study clinical characteristics.
- Factors affecting the outcome.
- Surgical outcome.
- Comparative analysis with other series.

RESULTS

Total No. of Cases =150

One hundred and fifty cases of acute subdural hematoma out of 714 cases of severe head injury constituted 21% of severe head injury.

Age

Youngest patient was 8-year-old who fell down from second floor and oldest patient was 68 year old. 75% of patients were in second to fifth decade. The details of age incidences are shown in (Table 1).

Table 1: Age distribution.

Age	No. of patients	%
1-10	10	6.6
11-20	20	13.3
21-30	60	40.0
31-40	35	23.3
41-50	10	6.6
51-60	10	6.6
61-70	05	3.3
Total	150	

Sex incidence

This study showed that males are predominant as compared to females. Details are shown in (Table 2). Out of 150 cases, Males are 110 (73.33%) whereas 40 cases are Females (26.67%).

Table 2: Sex incidence.

Gender	No. of patients	%	
Male	110	73.33	
Female	40	26.67	

Mode of injury

Most common mode of head injury is road traffic accidents accounts for 73.33%.

Details are shown in (Table 3). Road traffic accidents are most common mode of injury in causing sub dural haemorrhage.

Table 3: Mode of injury.

Mode of injury	No. of patients	%
RTA	110	73.33
Fall from height	26	17.33
Assualts	14	9.33

Glasgow coma scale at presentation

Most of the patients gcs was <8 at the time of presenting to hospital, which indicates very bad prognosis. Details are described in (Table 4).

Nearly 40% patients are presenting to hospital with head injury of poor gcs, which shows poor indicator of recovery.

Table 4: Glasgow coma scale at time of
presentation to hospital.

Gcs	No. of patients	%
< 8	60	40.00
9-12	50	33.33
13-15	40	26.67

Post resuscitation glasgow coma scale

Approximately 7% of patients were improved in gcs after post resuscitation. Details shown in (Table 5).

Table 5: Post resuscitation glasgow coma scale.

Gcs	No. of patients	%
<8	50	33.33%
9-12	60	40.00%
13-15	40	26.67%

Pupillary abnormalities

Patients with normal size and reacting pupils the mortality is 10%, for those with unilateral dilated pupil the mortality risen to 24.43%, and it is 100% for bilateral dilated pupils (Table 6).

Table 6: Pupillary abnormalities.

Pupils	No. of patients	Mortality	%
Normal pupils	50	5	10
Unilateral dilated pupil	90	22	24.45
Bilateral dilated pupil	10	10	100

Focal neurological deficit in mortality

Patients with no neurological deficit the mortality is 12.5% and with neurological deficit it is 28.57% (Table 7).

Table 7: Focal neurological deficit.

Neurological deficit	No. of patients	Mortality	%
No neurological deficit	80	10	12.5
Contralateral hemiparesis	70	20	28.57

Hemodynamic status

Patients with hypotension (systolic BP <90 mm hg), the mortality is 35% and for those with BP >90 mm hg the mortality is 21% (Table 8).

Table 8: Hemodynamic status.

Вр	No of patients	Mortality	%	P value
Systolic bp <90mmhg	40	10	35	< 0.005
Systolic bp >90mmhg	110	24	21.81	<0.09

CT scan thickness of hematoma

When the thickness of hematoma is <5mm the mortality is 5%, when the thickness is 5-10 mm the mortality is 22.5% and when the thickness is >10mm the mortality is 30% (Table 9).

Table 9: CT scan thickness of hematoma.

Thickness of hematoma	No of patients	Mortality	%	P value
<5mm	20	01	5	< 0.01
5-10mm	80	18	22.5	< 0.005
>10mm	50	15	30	< 0.001

Location of hematoma vs mortality

When the hematoma is located in the frontal region the mortality is 13.33%, when it is located in temporal fossa the mortality is 26.67%, when it is in fronto-temparo parietal region the mortality is 25%, and it is 20% when it is located in the posterior fossa (Table 10).

Table 10: Location of hematoma vs mortality.

Location	No. of patients	Mortality	%	P value
Frontal	30	04	13.33	< 0.001
Temporal	30	08	26.67	< 0.05
Fronto temporo Parietal	80	20	25	<0.05
Posterior fossa	10	02	20	< 0.05

Extent of hematoma

Details shown in Table 11.

Table 11: Extent of hematoma.

Extent of hematoma	No of patient	Mortality	%	P value
Focal	70	14	20	< 0.05
Diffuse	80	20	25	< 0.01

Diffuse hematoma shows high mortality.

Midline shift

Patients with midline shift <5mm the mortality is 16%, with midline shift >5mm the mortality is 26% (Table 12).

Table 12: Midline shift.

Midline shift	No. of patients	Mortality	%age	P value
<5mm	50	08	16	< 0.05
>5mm	100	26	26	< 0.01

Associated intra cranial injury

Details shown in Table 13.

Table 13: Associated intra cranial injury.

Associated Injury	No of patients	Mortality	%	P value
Simple sdh	30	04	13.33	< 0.005
Sdh with edh	20	04	20	< 0.05
Sdh with contusion	30	06	20	< 0.05
Sdh with sah	70	20	28.57	< 0.005

Table 14: Glasgow outcome scale at 1 week.

Grade	No. of patients	%age
1	34	22.67
2	05	3.33
3	15	10
4	46	30.67
5	50	33.33

Nearly 70% of patients were improved within one week of management.

Status of basal cisterns

When the basal cisterns are patent the mortality is 17.27%, and when they are obliterated the mortality is 37.5%.

Interval between injury to surgery

When the interval between the injury to surgery is <4 hours the mortality is 13.33%, when interval is between 5-12 hours the mortality is 35%, and when the interval is >12 hours the mortality is 60%.

Seizures - outcome

When post traumatic seizures are present the mortality is 35%, and when they are not present the mortality is 18.18%.

Table 15: Glasgow outcome scale at 6 months.

Grade	No of patients	%age
1	37	24.67
2	02	1.33
3	10	6.67
4	51	34
5	50	33.33

Glasgow outcome scale at 1 week

Details are shown in Table 14.

Glasgow outcome scale at 6 months

Details shown in Table 15.

Comparative study

Mortality in our series is 22.64%, details are shown in (Table 16).

Table 16: Comparative study.

Author	Sdh/total patients	% sdh of total	Mortality	%
Stone et al	128/712	18	59	46
Haselberger et al	111/567	21	57	51.35
Marshall et al	159/746	21	50	31.44
Gennerelli et al	319/1107	29	61	19.12
Present study	150/714	21	34	22.67

DISCUSSION

Morbidity and mortality after an acute sub dural hematoma are the highest of all traumatic mass lesions.¹⁻⁴ This poor outcome results largely from associated parenchymal injuries and subsequent intracranial hypertension.²⁻⁴ Approximately 50% of the patients have associated lesions.^{1.3,4} In our series we found 68% of acute SDH patients are having associated lesions. Majority of them are due to two-wheeler accidents most of them are not wearing helmets. We found a decreasing trend towards severe head injury with the enforcement of compulsory helmet wearing. Prognosis is worse in patients with pupillary abnormalities than those without. In our study, we found 10% mortality in patients with normal size reacting pupil, 24% with unilateral dilated pupils and 100% with dilated fixed pupils.

Kristianson et al found 100% mortality when pupils are dilated and fixed, 19% when they are unequal and 14% when pupils are normal.^{13,14}

Other significant factors associated with poor outcome are interval between injury to surgery, hypoxia, hypotension, post traumatic seizures, focal neurological deficit, obliteration of basal cisterns, thickness of hematoma, and midline shift. Traumatic coma data bank cohort study showed hypoxia in 46% patients and mortality in 40% of patients.¹⁵ In present study we found hypoxia in 40% and mortality in 33.33%

Hypotension is even more ominous predicting factor and the same study found it in 35% of patients and mortality in 60%. We found hypoxia in 25% of patients and mortality in 35% of patients.¹⁵ Obliteration of basal cisterns was found in 26.66% of patients and mortality in

37.5%, whereas without basal cistern obliteration the mortality is only 17.27%. Traumatic coma data bank also showed the similar type of results.¹⁵ Seelig and associates found that the time from injury to operative intervention is the critical determinant. The mortality if operated within 4 hours is 30% after it rose to 90%.¹⁶ Present study also confirmed this.

Wilberger and coworkers challenged this concept and they found that there is improved functional outcome if operated within 4 hours but this difference did not reached statistical difference.¹⁷ Gennereli et al found that interval between injury to surgery, hypotension, and basal cistern obliteration are the three important factors which have significant impact on outcome. Our study also found these three factors together have significant impact to the extent of 90% mortality.¹⁸

Heissler et al found thickness of hematoma, midline shift are having significant impact on outcome. They found hematoma thickness of >10mm, with midline shift >5mm are having 30% increased mortality than those without.¹⁹ Our study showed similar trend with mortality reaching 30%.

Sakas, Bullock, Teasdale GM fond that fixed dilated pupils with basal cistern obliteration, >3 hours duration of dilated pupils is having 100% mortality. We also found 100% mortality with fixed dilated pupils irrespective of above factors.²⁰

Yanaka K, kamazeki et al fond patients with GCS <8, Pupillary inequality, volume of hematoma >30ml, and midline shift >5mm, are having mortality to the range of 40%. We found it to the range of 30%.²⁰ The overall mortality in our series is 22.67%. In the study published by Stone et al it was 46%, in Haselbergers series it was 51.35%, in Marshall at al series it was 31.44% and in Gennerelli series it was 19.12%.^{18,19,21}

Jamieson, reviewing autopsy material of extradural and subdural hematomas found a disturbing trend towards inoperable lesions, and concluded that surgery can't hope to repair those lesions and advised accident prevention would be the chief means of preventing such lesions.^{2,3}

As situation, has not changed even after three decades of Jamisons observations his advice continues to be the most relevant even now.^{2,3}

CONCLUSION

We found that, the following factors are having significant influence on the outcome of acute sub dural hematoma in our study.

- Interval between injury to surgery with interval <4 hours having favorable outcome.
- Basal cistern obliteration, which is an indirect indicator of increased intra cranial pressure is the

single most important factor responsible for unfavorable outcome.

- Hypotension is another factor which is having significant influence on the outcome after surgery for acute subdural hematoma.
- Post traumatic seizures influence the mortality to the extent of 35% whereas without seizures the mortality is 19% only.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

- Ramamurthi B. Acute subdural haematoma. In : Handbook of clinical neurology : Injuries of the brain and skull. Vinken PJ, Bruyn GM (eds). North Holland Publishing Company Amsterdam. 1976;275.
- 2. Bagchi A. An introduction of head injuries; Oxford University Press, Calcutta. 1980;58-9.
- Jamieson KG, Yelland JD. surgically treated traumatic subdural hematomas J Neurosurg. 1972;37:137.
- 4. Kalyana Raman S, Ramamurthi B subdural hematoma. Neurol. 1970;18:18.
- 5. Abernethy J. surgical observations on injuries of head Dobson. 1811;2.
- 6. Burrows G. Lond Med Gaz. 1835;16:710.
- 7. Jacobson HH. Inter hemispherically situated hematoma. Case report. Acta radiol. 1995;43:23.
- Kristiansen K, Tandon PN. Diagnosis and surgical treatment of severe cranio cerebral injuries. J Oslo City Hosp (Supl.). 1960;10:107-213.
- 9. Miller SD, Statham PF. Surgical management of traumatic intracranial haematomas. In: Operative Neurosurgical Techniques. Schmidek HH, Sweet

WH (eds.) W.B. Saunders Company, Philadelphia. 1995;73-80.

- 10. Pospiech J, Kalff R, Herwegen H. Prognostic factors in acute traumatic epi and subdural haematoma. Aktuelle Traumatol. 1993;23:1-6.
- 11. Gutam MB, Moulton RJ, Sullivan I. Risk factors predicting operable intracranial haematomas in head injury. J Neurosurg. 1992;77:9-14.
- 12. Rao D, Subramaniam MV, Reddy MVR. Mortality in head injuries. Neurol. 1967;15:1.
- 13. Kristianson K, Tandon PN. Diagnosis and treatment of severe oedema. 2001.
- 14. Tandon PN. Management of head injury: Fads, fashions and facts. Neurol India. 1986;34:1-30.
- 15. Seelig J, Becker DP, Miller JD. Traumatic acute sub dural hematoma. N Engl J. Med. 1981;304:1511-8.
- 16. Marshall. The outcome of severe closed head injury. J.neurosurg, 75, s.J. Neurosurg. 1991;75, s28-36.
- 17. Willberger J, Harris E. Acute subdural hematoma J Neurosurgery. 1991;74:212-8.
- Gennereli T. A Influence of type of lesion on outcome from severe head injury.J.Neurosurg. 1982;56:26-32.
- 19. Stone JL. Acute subdural hematoma :progress in definition, clinical pathology and therapy Surg Neurol. 1983;19:216-31.
- Vigouroux RP, Guillermain P. Post-traumatic hemisphere contusion and laceration. In: Progress in neurological surgery. Krayenbuhl H, Maspco PE, Sweet WH (eds). 1981;10:49-163.
- 21. Hasselberger K. Prognosis after acute sub dural and extra dural hemorrhage. Acta neurochir. 1988;90:111-6.

Cite this article as: Prahaladu P, Prasad KS, Rajasekhar B, Reddy KS. Clinical study of acute subdural haematoma – a level I trauma care centre experience. Int J Res Med Sci 2017;5:857-62.