

Chronic subdural haematoma in adults – a tertiary centre experience

KEYWORDS

Acute Brain injury; Chronic Subdural Hematoma; Intracranial Hematoma.

Vijayakumaran Pillai	Meer M Chisthi
Associate Professor, Department of General Surgery,	Associate Professor, Department of General Surgery,
Government Medical College, Trivandrum, Kerala,	Government Medical College, Trivandrum, Kerala,
India. Corresponding Author	India.

ABSTRACT
Background: Chronic subdural hematoma is an encapsulated collection of altered blood, which is partly or totally liquefied.
The aim of this study was to analyse the clinical and demographic pattern of patients presenting with chronic subdural hematomas to a tertiary centre. Materials & Methods: This was a descriptive study, conducted on 50 patients admitted to the General surgical and Neuro-surgical wards of Government Medical College, Trivandrum, with diagnosed chronic subdural hematomas. From the data, the profile and pattern of these patients were analysed. Results: Majority of the patients presenting with chronic subdural hematoma are males in the age group between 60 to 80 years. Headache and hemiparesis are the commonest presenting symptoms in these patients. Most common mechanism of injury is fall followed by road traffic accidents. Conclusion: Chronic subdural hematoma is a disease predominantly affecting the elderly male population. Falls and motor accidents form the commonest mode of injury.

Introduction

Chronic Subdural Hematoma (Chronic SDH) is an encapsulated collection of old blood mostly or totally liquefied and located between the dura mater and arachnoid mater. It was first described by Virchow in 1857 as 'pachymeningitis haemorrhagica interna'. Later on Trotter put forward the theory of trauma to the bridging veins as a cause of what he named as 'subdural haemorrhagic cyst'. Since then trauma has been recognized as an important factor in the development of chronic SDH. The term chronic SDH applies to those haematomas that present over 20 days after injury. Haematomas are considered sub-acute when symptoms develop between 3 and 20 days after trauma and acute when the symptoms start earlier than 3 days. Those sub-acute hematomas presenting 3 to 4 days after injury have properties much common with acute SDH, while those presenting in the 3rd week are in their manner of presentation little different from chronic SDH.

The incidence of chronic SDH is estimated to be around 2 in 100000 a year $^{\rm 23.4.}$ The overwhelming majority of the patients are usually elderly or in late middle age. Trauma is supposedly an important factor in the development of chronic SDH. However a history of direct head injury is absent in as many as 30-50% of cases. Indirect trauma seems to be much more important as half of the patients have a history of fall but without hitting their head. In many situations the trauma is so trivial that it is often forgotten.

Chronic SDH is a clearly delineated fluid collection located between the dura and arachnoid mater. Long standing SDH is found enclosed within a capsule. The initiating factor in chronic SDH is either subdural bleeding or para infectious effusions. Virchow's hypothesis that chronic SDH's result from a generalized inflammatory disease of the dura mater is no longer accepted. He did not take into consideration the fact that the dura reacts entirely non specifically to blood, fibrin or fibrin degradation products with formation of a well vascularised hematoma capsule.

The exact etiology for the development of chronic SDH and especially its increase in size is still not fully understood. The lack of complete cellular organization and resorption of subdural blood or fibrin accumulation may be due to an already present latent coagulation disorder as well as excessive fibrinolytic activity in the cells of the 'neo' membrane. Eto et al 7 stressed the importance of local hyperfibrinolysis which causes liquefaction of the subdural blood clot as well as continuous hemorrhage from the sinusoidal vessels of the neo membrane in the development and enlargement of chronic SDH.

Two theories have been proposed to explain the growth of chronic SDH, namely the Osmotic theory and the Recurrent bleeding theory. Osmotic theory is based on the hypothesis that the liquefaction of the hematoma increases the protein content and oncotic pressure in the encapsulated fluid. This attracts fluid from the neighbouring vessels into the cavity due to osmotic pressure gradient across the semi permeable membrane^{5.} Recurrent bleeding from the hematoma capsule is the more widely accepted theory. The haematoma capsule has been shown to have abnormal and dilated blood vessels, as the source of hemorrhage. This theory was supported by the study done by Ito et al^{6,7.} They administered Cr⁵¹ labelled red cells intravenously 6 to 24 hours before the evacuation of hematoma and demonstrated that it contains 0.2 % - 28% of fresh blood. Also, increased fibrinolytic activity and coagulation abnormalities have been demonstrated within the chronic SDH, which may also play a part in the expansion of chronic SDH.

Chronic SDH may simulate dementia, stroke or even brain tumour with their clinical presentation. In the past the diagnosis was clinical and often required burr-holes for confirmation. With the advent of angiography earlier and now with the ubiquitous availability of CT and MRI scans, diagnosis and planning of treatment have become easy. With this background, this study was undertaken to assess the clinical and demographic pattern of patients presenting with chronic subdural hematoma in a tertiary level institution.

$Materials\,And\,Methods$

The study was done as a Descriptive study, at the General Surgery and Neurosurgery departments of Government Medical College, Trivandrum for 3 years, from March 2012 till March 2015. The objective of the study was to study the various clinical and demographic pattern of patients presenting with chronic subdural hematoma over the study period. The secondary objectives were to find the predisposing factors for chronic SDH and the surgical complications in these patients.

Institutional Review Committee and Ethics Committee clearance were obtained before data collection. Patients who were admitted with a radiologically documented chronic SDH, were included in the study. Only those patients with age between 12 - 80 years were included. The sample size was set at 50 based on values available from similar studies. After obtaining informed consent, patients were recruited into the study.

On admission, the patients' detailed history was noted. After recording general survey, patients were managed by the Neurosurgery department. At the time of admission, the patients underwent detailed clinical examination. The resultant data was entered into a prefixed proforma with specific reference to initial symptoms of headache, vomiting, behavior changes impairment of consciousness, seizures, and neurological deficits; past history of head injury and the cause of head injury, past history of other diseases like seizures, bleeding disorders, hyper tension, diabetes, previous intracranial operations etc. The CT scan findings were recorded in all patients.

Daily follow-up was done to continue the neurological monitoring. Daily assessment of temperature, pulse rate, blood pressure, respiratory rate, input and output was also done. Management included antiepileptics, antibiotics, sedatives and in all cases mechanical ventilation following neurosurgical procedures. All values are given as means and percentages as relevant.

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In this study of cases of chronic SDH, 50 patients were included in all. 84% of cases were between the ages of 40- 79 years, while 6% of cases were above the age of 79 years and 10% were below the age of 40 years [Table 1]. The oldest person was of 91 years. Incidence was much higher in males: 80%, and low in females: 20%.

Table 1. Age and Sex distribution of study cases

Age group	Male	Female	Total	Percentage
20-29	2	-	2	4
30-39	3	-	3	6
40-49	4	1	5	10
50-59	8	-	8	16
60-69	9	2	11	22
70-79	11	7	18	36
80-89	2	-	2	4
90-99	1	-	1	2
TOTAL	40	10	50	100
	80%	20%		

Majority of patients had more than one symptom [Table 2]. 64% of patients had head ache. 38% of patients had impaired consciousness, while 52% had hemiparesis. Vomiting was present in 16% of cases, seizures in 4%, memory loss in 8%, impairment of vision in 6%, history of recurrent fall in 4%, difficulty in walking in 14%, slurring of speech in 12%, vertigo in 6%, facial palsy in 6%, urinary incontinence in 6% and aphasia in 2% of cases.

Table 2. Analysis of main symptoms and signs of study patients

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Symptoms	Number of cases	Percentage
Head Ache	32	64
Vomiting	8	16
Behavioural changes	6	12
Impaired consciousness	19	38
Seizures	2	4
Hemi Paresis	26	52
Memory Loss	4	8
Impairment of vision	3	6
H/o recurrent fall	2	4
Difficulty in walking	7	14
Slurring of speech	6	12
Vertigo	3	6
Facial Palsy	3	6
Incontinence of Urine	3	6
Aphasia	1	2
Papilloedema	1	2

Out of 50 cases, 84% of cases had history of trauma to head. 30 patients had history of fall (60%), while 8% had history of hit to head,

while 16% had history of RTA. 12% cases had no history of trauma to head. 4% had history of anti coagulant therapy. 32 cases were admitted within the first week of first symptom(64%) [Table 3]. 17 cases were admitted within the first month but later than one week (34%), while 1 case was admitted after one month.

Table 3. Interval between the first symptom and hospital admission

Duration	Number of cases	Percentage
Less than 1 week	32	64%
1 week to 1 month	17	34%
More than one month	1	2%

40 of the cases were unilateral and 10 cases were bilateral. 2 cases were complicated by re-bleeding. In the first case, patient's neurological status deteriorated in the post operative period. Evacuation of the haematoma was done by twist-drill and patient improved subsequently. The second complicated case was a known cardiac patient on anticoagulant drugs, whose neurological status deteriorated and was diagnosed as re bleeding. Re-operation was done through the same craniectomy wound. Patient improved neurologically. However, after some days, patient died due to myocardial infarction. Apart from this patient, 2 other deaths occurred. One patient was a known parkinsonism patient, who died few days after surgery due to respiratory failure. The third patient was a known case of chronic kidney disease, on haemodialysis, who died a few days after surgery of renal failure.

Discussion

The present study consisted of 50 cases of chronic SDH who were treated by surgery in the General Surgery and Neurosurgery Departments of our institution over 3 years. In the series, there is a male preponderance, which is seen in similar studies also. Majority of the patients were aged more than 70 years. Svien et als reported that over three quarters were over 50 years of age with an average age of 63. Cameron reported an average age of 56. The median age in other series has also been the late 50's or early 60's really age and is 7.4/100,000 per year for a population in the 70's. Whereas the incidence is only 0.13/100,000 per year for those in the 30's.

In our study, the commonest symptoms were headache and hemiparesis, which were the predominant symptoms in similar studies also. In the present study, 84% had past history of head injury. In a study by Foelholm, 71% of cases gave a history of head injury. Spontaneous chronic SDH without trauma has been reported to occur in young adults. Between 25 and 48 % of patients have no history of head injury. Even when remembered, the injury may be mild. All Many patients give history of chronic alcoholism. This may account for the failure to obtain definite history of head injury. Other precipitating factors are epilepsy, shunting procedures and coagulopathy ctc. Patients undergoing renal dialysis are more prone to develop chronic SDH presumably due to platelet dysfunction. Patients with adult polycystic disease may be predisposed to develop spontaneous SDH. As many as 24% of patients with chronic SDH are on warfarin or an anti-platelet drugs.

Majority of the cases were unilateral. One-fifth of the cases had a bilateral presentation. The mortality rate in our study was around 6%, which is comparable to other studies which reported mortality rates varying from 4% to 10%. In the present study, deaths were due to unrelated diseases like myocardial infarction, parkinsonism and renal failure, all of which occurred a few days after surgery.

Conclusions

To conclude, majority of the patients presenting with chronic subdural hematoma are elderly males. They are involved mainly by falls and road traffic accidents. Pre existing conditions like cardiac disease and renal failure can increase the mortality and morbidity in these patients. Health advice has to be given to the elderly population to take extra caution while moving around. Also, medical opinion has to be sought for as and when required so as to ensure optimal outcome.

References

- Adhiyaman V, Asghar M, Ganeshram KN, Bhowmick BK. Chronic subdural haematoma in the elderly. Postgrad Med J. 2002 Feb;78 (916):71-5.
- Foelholm R, Waltimo O. Epidemiology of chronic subdural haematoma. Acta Neurochir (Wien). 1975;32(3-4):247-50.
- Laudig GH, Browder J, Watsan RA. Subdural Haematoma- A study of 143 cases encountered during a 5-year period. Ann Surg. 113:170-191 (Feb) 1941.
- Weber G. [Chronic subdural hematoma] Schweiz Med Wochenschr. 1969:99:1483-1488.
- Gardner WJ. Traumatic subdural hematoma with particular reference to the latent interval. Arch Neurol Psychiatry. 1932;27:847–858.
- Ito H, Yamamoto S, Komai T, Mizukoshi H. Role of local hyperfibrinolysis in the etiology of chronic subdural hematoma. J Neurosurg. 1976;45:26–31
- Ito H, Yamamoto S, Saito K,et al. Quantitative estimation of hemorrhage in chronic subdural hematoma using the 51Cr erythrocyte labeling method. J Neurosurg 1987;66:862-4
- Svien HJ, Gelety JE. On the surgical Management of encapsulated subdural Haematoma. A comparison of the results of membranectomy and simple evacuation. J Neurosurg. 21, 172-177; 1964.
- Cameron MM. Chronic subdural haematoma: a review of 114 cases. J Neurol Neurosurg Psychiatry. 1978 Sep;41(9):834–839.
- Luxon LM, Harrison MJ. Chronic subdural haematoma. Q J Med. 1979 Jan;48 (189):43–53.
- McKissock W, Richardson A, Bloom WH. Subdural haematoma: a review of 389 cases. Lancet. 1960;275:1365–1369.