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# Diagnosis and management of chronic subdural haematoma: Our experience in Chittagong Medical College Hospital

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***Abstract****: SUBDURAL HAEMATOMA (SDH) is characterized by a collection of blood or fluid blood products in the space between the dura and arachnoid or pial layer in the brain. It can occur as a result from head injury or spontaneously or in various other pathologies. Chronic SDH are encapsulated blood collections within the dural border cell; develops 2 to 3 weeks after the inciting event or even later and often has a poor prognosis. A chronic subdural haematoma is predominantly a disease of elderly male usually above 70.*
*Imaging shows crescentic layer of fluid in the subdural space on a non-contrast computed tomography (CT) scan, best appreciated on sagittal or coronal reformats. Operative interventions, such as burr-hole craniostomy (BHC), and craniotomy are indicated if imaging implies compression (maximum fluid collection thickness >1 cm) or the patient is neurologically symptomatic. Because prompt diagnosis and treatment is essential to prevent disability or death and complications of CSDH, this study is intended to share our experience about the patients regarding their epidemiology, clinical assessment and diagnosis and surgical outcome in Chittagong Medical College Hospital(CMCH).*

***Key words****: Chronic subdural haematoma; Management; Outcome; Prognosis; Surgery*

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**Aims:** To prevent disability or death in chronic subdural haematoma cases where prompt diagnosis and effective treatment plays a key role.

**Methodology:**
STUDY DESIGN, TIME AND PLACE: A prospective study was done in Chittagong Medical College hospital during the period from Feb- 2017 to Feb- 2018 and 85 patients diagnosed as CSDH were admitted to dept. of Neurosurgery in Chittagong Medical College Hospital.

**Sampling Technique:** Non probability type of purposive sampling.
DATA COLLECTION INSTRUMENT: We collected data from the CSDH patients admitted in the neurosurgery department regarding their clinical manifestations and neuro-imaging findings, recorded those and analyzed. We observed prognosis following the surgical intervention, in terms of neurological outcome, morbidity, mortality, and recurrence.

**Data Presentation:** Data was presented in graphs and chart.

**Inclusion Criteria:** Patients presented with the clinical symptoms of CSDH alone were included in this study and confirmed by 1) fluid hematoma (usually >21 days after injury) identified at the time of surgery; and 2) Computerized tomography (CT) scan showed isodense to hypodense and mixed density hematomas with respect to the adjacent brain 3) any midline shift in CT scan of CSDH patients.

**Exclusion Criteria:** We excluded all patients who had been previously operated elsewhere or with incomplete medical records. Patients were also excluded if: 1) they had concomitant occurrences of other types of traumatic brain injury; 2) CSDH had resulted from complications of prior neurosurgical procedures, such as craniotomy or cerebrospinal fluid shunting; 3) CSHD patients receiving conservative treatment without surgery; 4) the images of the brain could not be assessed because of missing data or poor quality. Thus, only 85 patients were enrolled for this study.

## I. Introduction

Chronic subdural haematoma is an encapsulated collection of old blood, mostly or totally liquefied and located between the dura mater and arachnoid. It was first described by Virchow in 1857 as "pachymeningitis haemorrhagica interna". Later Trotter put forward the theory of trauma to the bridging veins as a cause of what he named "subdural haemorrhagic cyst". Since then trauma has been recognized as an important factor in the development of CSDH.

SDH may be acute, chronic, or subacute based on the timing of occurrence and brain imaging.

ACUTE - where the blood collects quickly after a head injury; symptoms can occur immediately or within hours. It generally occurs in younger adults, after a major trauma, often associated with structural brain injury.

SUBACUTE - where symptoms develop between 3-7 days after the injury.

CHRONIC - blood collects slowly after a head injury and symptoms can occur 2-3 weeks after the initial injury. Often occur in the elderly after a trivial trauma without any damage to the underlying brain.

It has a peak incidence of 1.72/100000 per year, the incidence increasing steeply with advancing age up to 7.35/100000 per year in the age group 70-79.[1]

Morbidity and mortality associated with an SDH increases with age as well as exposure to anticoagulant and antiplatelet therapy. [2 3 4]

Several causes and predisposing factors associated with CSDH are cerebral atrophy, older age, male gender, head trauma, exposure to certain medications and seizure activity. Among them head injury is the commonest one and fall from height is the most common type of all traumatic brain injuries, including SDH. The incidence of falls peaks in both males and females between ages 75 and 79.[5]

The incidence of mild head trauma has been reported in up to 80% of patients with CSDH, although many have little or no memory of the inciting event.[6] Approximately 38% of patients with a CSDH have a history of only minor head trauma or can't recall any prior head trauma.[7] Systemic effects of anticoagulation and antiplatelet therapy are well known risk factors for CSDH. Approximately 75% of primary spontaneous or non-traumatic cases of CSDH are diagnosed in patients taking these medications. In population studies, patients on oral anticoagulation therapy are estimated to have a 4- to 15-fold increased risk for CSDH.[8] A SDH in the setting of anticoagulation can expand rapidly. Unless anticoagulation is quickly reversed, the expanding SDH can result in severe signs and symptoms and/or death.[5, 7]

## II. Pathophysiology

Subdural haemorrhages are believed to be due to stretching and tearing of bridging cortical veins as they cross the subdural space to drain into an adjacent dural sinus. These veins rupture due to shearing forces when there is a sudden change in the velocity of the head. The arachnoid may also be torn, creating a mixture of blood and CSF in the subdural space. In a chronic subdural collection, blood leaks from the veins slowly over time, or a fast hemorrhage is left to clear up on its own.

A day after the initial haemorrhage, the outer surface of the haematoma is covered by a thin layer of fibrin and fibroblasts. Migration and proliferation of the fibroblasts leads to formation of a membrane over the clot by the fourth day. The outer membrane progressively enlarges and the fibroblasts invade the haematoma and form a thin membrane during the next two weeks.[9] Liquefaction of the haematoma occurs due to the presence of phagocytes. Then the haematoma may either resorb spontaneously or slowly increase in size resulting in a CSDH.

Two major theories have been proposed to explain the growth of a CSDH—One is Osmotic theory which postulates that the liquefaction of the haematoma 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 semipermeable membrane (haematoma capsule).[10] However this theory was disproved by Weir, who demonstrated that the osmolality of the haematoma fluid was identical to that of blood and cerebrospinal fluid.[11]

Second one is widely accepted recurrent bleeding theory from the haematoma capsule. The haematoma capsule has been shown to have abnormal and dilated blood vessels, the source of haemorrhage.[12] This theory was supported by the study done by Ito et al.[13] Over time; a CSDH may further enlarge due to continued or recurrent venous bleeding, leading to serious complications such as increased intracranial pressure (ICP).[14] SDH may become lethal by increasing ICP. Compression of brain tissue results in cerebral ischemia, stroke, brain tissue shift, and brain herniation and ultimately death

A subdural hematoma is more common in older adults because of normal brain shrinkage that occurs with aging. This shrinkage stretches and weakens the bridging veins. These veins are more likely to break in older adults, even after a minor head injury.

Other risks include:

- Long-term heavy alcohol use
- Long-term use of aspirin, anti-inflammatory drugs such as ibuprofen, or blood thinning (anticoagulant) medicine such as warfarin
- Diseases that lead to reduced blood clotting

**Clinical manifestations**:

The exact symptoms that appear depend on the location and size of the hematoma. To assess symptoms, we classified the patients by age, over 65 years and below 65 years, in accordance with the definition of elderly population of the World Health Organization (WHO).[15]

Patients of CSDH usually presents with:

**Altered mental state**: The most common presentation in the elderly (50%–70%) is altered mental state.[16 17 18] Some of the patients presents with varying degrees of confusion, drowsiness, or coma. Some presents with focal neurological deficit. Hemiparesis was found in 58 % of cases in one series.[19] Weakness of the limbs is usually mild. Mostly the deficit is contralateral. Fluctuating neurological symptoms are uncommon and usually the symptoms start insidiously and progress gradually. [20]

**Headache**: The incidence of headache varies in different studies ranging from 14% to 80%.[21 22] It is less common in the elderly when compared with a younger patient.It is partly due to the large available intracranial space for the haematoma to accommodate before creating pressure on the adjacent brain. Another reason is the earlier onset of confusion, which attracts medical attention before the development of headache in the elderly.

**Falls**: Interestingly falls have been reported to be a very common presenting symptom (74%) in a recent prospective study involving 43 elderly patients.[22]It is a well-known fact that recurrent fall is a significant risk factor for CSDH. Development of CSDH may lead to recurrent falls or increase the frequency of falls due to altered mental state, neurological deficits, and postural disturbances.

**Seizures**: Epilepsy is usually a rare presentation, even though it has been reported in up to 6% of cases as an initial symptom.[21] In patients with known epilepsy increasing frequency of seizures has been noted with the development of CSDH. Simple partial seizure has been reported as a sole manifestation of CSDH, and this could be easily mistaken for a transient ischaemic attack.[23] Seizures usually occur in the presence of a large haematoma associated with focal neurological deficit.

**Transient neurological deficit**: The incidence of CSDH presenting with TND varies from 1% to 12%.[24] The most common symptom is disturbance in language and the most frequent sign is hemiplegia or hemi sensory deficit. Sometimes CSDH patients may present atypically with the following symptoms:

**Isolated neurological deficits**: Patients presenting with vertigo, nystagmus, upward gaze palsy, and isolated oculomotor palsy due to CSDH have been reported.[25 26 27] Increased intracranial pressure causing uncal herniation and stretching of cranial nerves was thought to be the mechanism involved.

**Extrapyramidal syndrome**: CSDH causing parkinsonism is a well recognised phenomenon.[28] Reversible akinetic-rigid syndrome due to bilateral CSDH with complete resolution after surgery has also been reported.[29]

**Diagnosis of CSDH:**

The most important step in the diagnosis of CSDH is a high index of suspicion. It should be considered in any patient with or without a history of trauma presenting with (1) a change in mental status or worsening of pre-existent neurological or psychological illness, (2) focal neurological deficit, and (3) headache with or without focal neurological deficit.

Non contrast brain computed tomography (CT) is the initial imaging study of choice. In all cases, diagnosis was based on computed tomography (CT). Radiological measures of the CSDH, including the width of hematoma and midline shift were determined based on CT scans obtained before the operational procedure.

Magnetic resonance imaging doesn't routinely have a role in diagnosis of CSDH, but it may be performed when SDH is suspected but not demonstrated on CT scan.

Routine laboratory studies before surgery included a complete blood count, platelet count, international normalized ratio, prothrombin time and activated partial thromboplastin time, and biochemical investigations.

**Initial management**:

Commonly most of the elderly CSDH patients also have other chronic and complex comorbidities, so management requires collaboration among neurologists, neurosurgeons, nurses, rehabilitation specialists, and others such as a social worker and case manager, based on the patient's clinical status.[11 30] Immediate interventions are initiated to support the ABCs—airway, breathing, and circulation—optimize BP and hemodynamics, and normalize coagulation status. Any antiaggregant and AC therapy was temporarily discontinued upon admission and re-established no earlier than 4 weeks after the operation. Coagulopathy, if present, was corrected preoperatively by intravenous (IV) infusion of fresh frozen plasma, Vitamin K, or platelets.

After initial management when the patients became haemodynamically stable surgical intervention with Barr-hole craniostomy and a closed drainage system was done in all 85 cases.

## III. Observation And Results

85 patients of chronic subdural haematoma were treated in neurosurgery department of CMCH. Data shows that the sample population was composed of 68 males and 17 females (ratio 4:1).

**Table-1 Sex distribution in Study group (n=85)**

| Study group(n=85) | | |
|---|---|---|
| **Sex** | **No of cases** | **%** |
| **Male** | **68** | **80%** |
| **Female** | **17** | **20%** |
| **Total** | **85** | **100** |

Here male were mostly affected group ( 68, 80%). Because females are less exposed to exterior.

The cases of CSDH were found mostly among the age range from 61 to 80years. The mean age of patients with CSDH was 63, and 70.2% of them were over age 60yrs. The incidence of CSDH was expected to increase as the population continues to age.

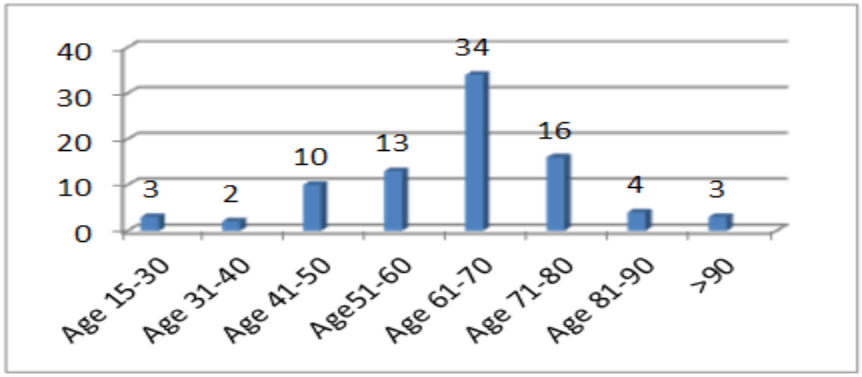


**Figure: 1** Age distribution in study group

Patients presented mostly with the symptoms of hemiparesis (65 cases), followed by headache (52 cases), language difficulty (36 cases), seizures (17 cases), confusion and coma in 26 and 9 cases respectively.

**Table -2 Clinical manifestation in Study group (n=85)**

| | **No** | **%** |
|---|---|---|
| Hemiparesis | **65** | **76.5%** |
| Headache | **52** | **61%** |
| Language difficulty | **36** | **42.3%** |
| Seizures | **17** | **20%** |
| Confusion | **26** | **30.5%** |
| Coma | **9** | **10.5%** |

The neurological status on admission was assessed according to the Glasgow Coma Scale and in our series we found 54 patients (63.5%) in grade between 13—15, 7 patients (8%) in between grade 11—12, 11 patients (13%) in between grade 8—10 and below grade 7 was 13 patients (15%).

| Group I GCS < 7 | | Group II GCS 8-10 | | Group III GCS 11-12 | | Group IV GCS 13-15 | |
|---|---|---|---|---|---|---|---|
| no | % | no | % | no | % | No | % |
| 13 | 15.2% | 11 | 12.9% | 7 | 8.2% | 54 | 63.5% |

**Table – 3 Shows the number and percentage of patients according to GCS level on admission.**

In our study group 33 patients (38.8%) did not give any history of trauma but 52 patients (61%) had a history of head trauma most of which were minor or moderate and most of them (42 patients) presented within 3weeks to 3 months after trauma.

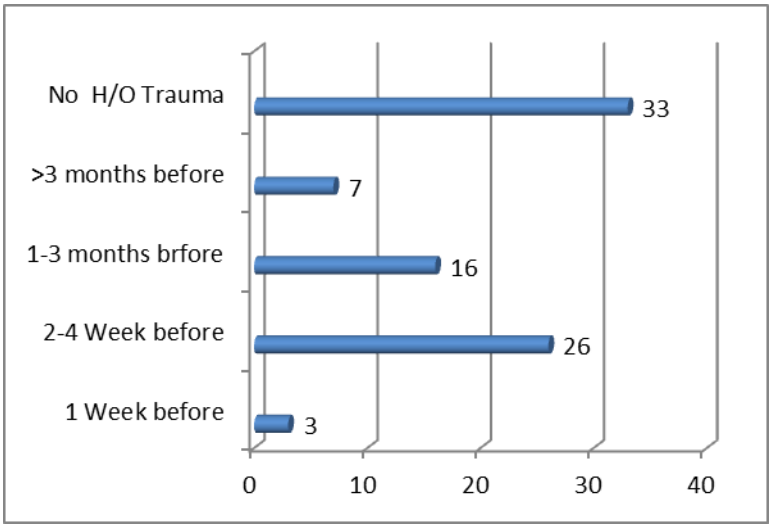


**Figure: 2** shows number of patients with history of trauma in relation to time of presentation

The CSDH was unilateral in 74 cases with left sided haematoma in 49 cases (66%) and right sided in 25 patients (34%). Haematoma was bilateral in the remaining 11 cases (12%) with left side preponderance.

**Table-4 Shows number of CSDH patients with side determination on CT Scan in study group(n=85).**

| Name of investigation | Unilateral | | | | Bilateral | |
|---|---|---|---|---|---|---|
| | Unilateral -L | | Unilateral -R | | | |
| CT Scan of brain finding | 49 | 66% | 25 | 34% | 11 | 12% |

About 50% (43 cases) of the patients presented with comorbidity. Among them arterial hypertension and diabetes mellitus presented in 79% (34 cases) and 35% (15 cases) of patients respectively.

**Table – 5 Shows Comorbidities with % in study group. (n=85).**

| Name | No of patients | % |
|---|---|---|
| Diabetes | 15 | 35 |
| Hypertension | 34 | 79 |
| Cardiac disease | 12 | 39.53 |
| Respiratory disease | 5 | 11.62 |
| Renal disease | 1 | 4.65 |
| No Comorbidities | 42 | |

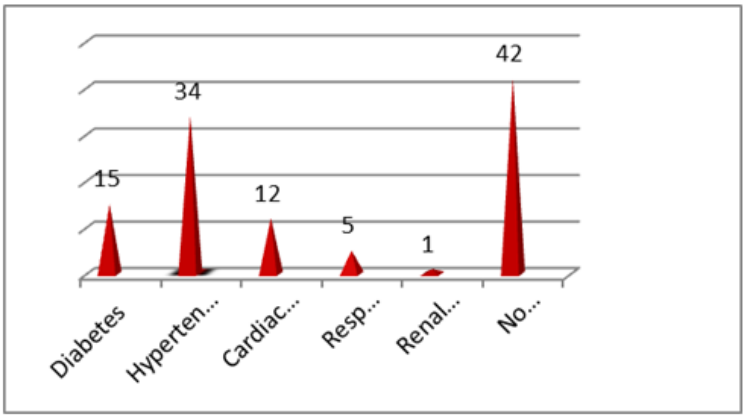


**Figure: 3** Comorbidities with % in study group

Mid line shifting ranges from 0.5cm up to 2.5cm was found in 72 cases out of 85, with varying degree of GCS level.

**Table-6 Shows CSDH patients with midline shift (CT scan) in study group (n=85).**

| 0-.05cm | .05-1.0cm | 1.1 - 1.5cm | 1.6-2cm | 2.1-2.5cm | > 2.50cm |
|---|---|---|---|---|---|
| 10 | 23 | 21 | 14 | 3 | 1 |

Postoperative complications occurred in 11patients (12.94% cases) and death occurs in 5 cases. (2 cases on table death and 3 cases after wards). Early results were estimated 30 days after surgery. Eighteen patients had an uneventful post-operative recovery and were discharged on 7th day without routine postoperative C.T. scans. Four patients had symptoms of increased intracranial pressure on the second or third post-operative day. C.T scan revealed cerebral edema. They were treated with 20% mannitol intravenously and made a complete recovery. Two patients had residual haematomas and required second evacuation via previous burr holes. They recovered completely. One patient acquired superficial wound infection on the 5th post-operative day, which responded to antibiotic therapy. Four patients developed generalized seizures and were treated with anticonvulsants. Minimum follow-up period was 3 months. Patients who returned for follow-up, were well. 28 patients out of 41, who had been discharged with hemiparesis or language difficulty recovered well within 3 months and few patients still had these symptoms but were independent in their everyday activities.

**Table-7 Shows Operative outcome (n-85)**

| Discharged after 7days with uneventful recovery | | Discharged within 7 to 10 days with mild hemiparesis and mild language difficulty | | Complications developed and discharged after recovery | | | | Death | | Recovery | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Number of cases | % | Number of cases | % | Increased ICP | Residual haematomas | Wound infection | Seizures | On table | After one month | Recovery after 1 month | Recovery after 3 month |
| 18 | 21.1 | 41 | 48.2 | 4 | 2 | 1 | 4 | 2 | 3 | 57 | 77 |

## IV. Discussion

Acute and chronic SDH are common findings in patients who have sustained severe to mild closed head injuries.[31] The mortality of chronic subdural haematoma has significantly reduced in the recent years, mainly due to improvement in diagnostic techniques, which allows earlier recognition of the intracranial lesion and appropriate neurosurgical treatment.[2 32]

Chronic subdural haematoma is predominantly a disease of the elderly, but not infrequent in the young[32,33,34,35] Data collected from the United States cencus bureau show that the incidence of SDH almost doubles from ages 65 to 73 and continues to increase in people over age 80. Incidence of SDH is highest in the fifth through seventh decades of life.[2] In our study group among 85 patients, the cases of CSDH were mostly in between the age range from 61 to 80years and not over 80 years . Where life expectancy in our country is 72

years on an average this finding is quite consistent . The mean age of patients with CSDH was 63, and highest incidence (70.2%) of them were over age 60yrs which correlates to the study of others.[2] Nearly similar results was found in cases of review studies[3 4 5].

In all series men were more commonly affected than women, corroborating our findings.

Minor to moderate trauma history was found among 52 patients (61%) in my series, and 33 patients ( 38.82%) did not give any history of trauma. Rovlias A et al also cited the similar result in their study with 986 cases of chronic subdural haematoma showing that 51% of their patient (530 cases) had a history of trauma, most of which were minor to moderate. [36]

Adhiyaman et al found that although trauma is an important factor in the development of CSDH but a history of head injury (direct trauma) is absent in about 30%–50% of the cases.[7] Indirect trauma seems to be more important. About half of the patients have a history of fall but without hitting their head on the ground.[8 9] Again approximately 38% of patients with a CSDH have a history of only minor head trauma or can't recall any prior head trauma. [37] .

Elderly people usually suffer from other concomitant diseases which complicates the diagnosis and hence timely proper management of CSDHs hence can impair both its prognosis and surgical outcome. In fact, death and recurrences are sometimes influenced more by the patients poor preoperative clinical status or complication caused by concomitant diseases than by complications or failure of surgical treatment. Comorbidities which we found in our series were arterial hypertension in 79 %( 34 cases) and diabetes mellitus in 33% (15 cases). Associated systemic disease like hypertension, diabetes, and ischemic heart disease etc were diagnosed in 43 patients (41%) by J.Gurunathan M ch[10]

The neurological status on admission was recorded according to Glasgow Coma Scale and most of the patients 54 out of 85 ( 63.5%) were in grade between 13—15,and below grade 7 was found 13 cases (15%).). The Glasgow Coma Scale on admission ranged from 4 to 15 with an average of 12, and after surgery it ranged from 7 to 15 with an average of 14.

Although altered mental state is the most common presenting features found by many authors,[20 38 39] we found mostly hemiparesis in 65 cases (76%), followed by headache in 52 cases (61%), language difficulty (36 cases),seizures (17 cases), confusion and coma in 26 and 9 cases respectively. In a developing country like ours where everyday poverty leads the elderly people to be treated as a burden on the family, late presentation with hemiparesis is not very unlikely for them. More over lack of awareness about the good health status and communications problem along with the absence of good neurosurgical infrastructures discourages our poor elderly to present early with an altered mental state as like those presented early in the developed country.

Along with the advancing age and previous history of head injury, other predisposing factors included anticoagulant or anti aggregant therapy, alcohol abuse, and coagulopathy. But fortunately in our cultural practice and religious background 'alcohol abuse' may not play a causal role for CSDH. On the other hand our poor people are not literate enough to be health conscious and take anti platelet or anticoagulation therapy routinely for a longer period of time. Both of this may have a beneficiary effect on our post-operative outcome. Whereas the literature of different authors show symptomatic recurrence of CSDH after surgery ranges between 3% to 27% [40 41] or from 8% to 37% [42] we found post-operative recurrence only in three cases (3.52%).

Other than the recurrence, complications which we found in our series was bedsore in 1 case, Intraventricular haemorrhage in 1 case, Extradural haematoma (left) in 1 case and abscess formation in one case.

The morbidity and mortality in CSDH varies widely in the literature. However the outcome is good in patients who undergo neurosurgical intervention where the morbidity and mortality after surgery is around 16% and 6.5% respectively. [43]

Although currently there are no specific guidelines to show the indications for surgery in CSDH, all symptomatic individuals should be considered as potential surgical candidates. [44] Surgical evacuation is recommended in cases of any SDH confirmed by radiologic imaging with maximal thickness of greater than or equal to 1 cm, or greater than or equal to 0.5 cm of brain tissue midline shift. So we did surgery in our study group having mid line shift ranges from 0.5cm to 2.5cm regardless of their GCS level to achieve a good result.

Surgery what we did was simple barr hole with drainage in all 85 cases because general consensus is that patients with signs and symptoms that can be attributed to radiologically confirmed CSDH should be treated surgically.[45]

The basic assumption is that CSDH should be removed by a simple means. Convincing evidence has accumulated that burr holes technique is a safe, time-saving, and rational treatment which can be performed for elderly patients or those with multiple medical problems using local anaesthesia and is usually able to achieve favourable results without complications.[46 47 48 49 50]

Mortality in our series we found 5 cases (5.88%), 2 patient were in table death and in 3 cases after one month. We were mainly interested to see the overall outcome of patients with CSDH based principally on the

evolution of the clinical course and all our patients had a postoperative follow- up for up to one month. Mortality might be high if we could follow up the patients for longer period.

Main disadvantage of our study was proper follow up of the patients. If we could followed up all our patients maintaining our protocol schedule the morbidity and mortality for the cases might show more accurate result. Variations in the assessment of neurological status was another account for the observed differences in results. The period of evaluation of neurological outcomes varies and follow up information was difficult to obtain. Despite these miscellaneous limitations, this study provides useful preliminary information to identify CSDH patients, which can optimize the timing of diagnosis and establish therapeutic strategies and open door for further research.

## V. Summary

CSDH is a collection of liquefied blood between the dura and arachnoid layer of the brain thought to result from injury to bridging veins crossing the subdural space. Head injury is a common risk factor and in a study of 1000 patients, 61.7 % recalled a recent one.[51]

It affects mainly elderly patients. As the world population becomes progressively older, the overall incidence is increasing. Advancing age with associated brain atrophy and prophylactically used blood thinner and anticoagulants makes the elderly vulnerable to develop CSDH. Various presentations which occasionally mimic stroke or rapidly progressive dementia sometimes mislead the clinicians to reach the diagnosis early. At the same time associated comorbidities of older people impair both its prognosis and surgical outcome. With the advent of computed tomography radiological diagnosis of CSDH becomes reliable. Treatment is also improved dramatically in recent years because of this diagnostic tool and simple (burr hole) surgical technique ultimately reducing the morbidity and mortality of these cases.

## VI. Conclusion

Chronic subdural haematoma (CSDH) is one of the most common clinical entities in daily neurosurgical practice which carries a most favourable prognosis when diagnosed accurately and treated adequately.

Early identification of reliable prognostic factors is also of great importance for CSDH patients to achieve a good result.

Treatment of CSDH is improved dramatically in recent years only because of advances in diagnostic tools and surgical techniques. Thus the burr hole evacuation, irrigation, and subsequent closed drainage technique has become a simple treatment able to achieve good results with minimal complications.

Elderly people, people with a bleeding disorder and who take blood thinners are more likely to develop CSDH after infliction of a relatively minor head trauma. So all measures should be taken to prevent head injury and falls in elderly to reduce the overall chronic Subdural Hematoma cases and thus reducing the total morbidity and mortality associated with CSDH.

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