CHAPTER 1

1.0 INTRODUTION

Chronic subdural haematoma is a well known entity since its first description by Johann Wepfer in 1656. Chronic subdural haematomas are collection of liquefied blood in the subdural space, consistency of which varies from thick dark oily to thin tea-coloured fluid, depending on the age of the haematoma. It is a relatively common condition seen in neurosurgical practice. European population studies have estimated an annual incidence of between 1-2 cases per 100 000 population per year (11). A higher incidence of 13.1 cases per 100 000 population per year was seen on the Japanese Island of Awaji (25). The mean age in large series generally ranges from 53-63 years, but it can occur in any age groups (10,11,14,29). Typically 70% to 80% of these patients are men. The occurrence of chronic subdural haematomas has been linked to a number of predisposing factors.

1.Trauma

In most series approximately 2/3 of patients have a history of trauma and usually of a minor nature (11,14). Chronic subdural haematomas have also been reported as a complications of birth trauma in neonates (43).

2. Non- Trauma

Therapeutic anticoagulation may play a factor in from 5% to 7% of patients presenting with chronic subdural haematoma (14,39). The risk doubled for each one-half unit increase in prothrombin time rates over two. Other medical condition leading to coagulopathic states may include renal dialysis, sepsis and hepatic failure .Intracranial hypotension is a less common cause of chronic subdural haematoma (18). Intracranial hypotension as a result of over drainage of cerebrospinal fluid leak may result in increased traction on bridging vein leading to haematoma.

3. Chronic alcoholism

Alcoholism is well known to be associated with the development of chronic subdural haematoma (39). Probable mechanism may be because of its strong association with cerebral atrophy, coagulopathy and repeated trauma.

4. Other associations

Chronic subdural haematoma may rarely complicate with arachnoid cysts, (32) vascular malformation (23) and tumors (29).

1.1 CLINICAL FEATURES

An understanding of the varied clinical presentation of chronic subdural haematoma is essential to stimulate clinical suspicion and prompt evaluation, so as to improve the clinical outcome before the occurrence of more severe neurological impairment.

The manifestation of clinical symptoms in individuals with chronic subdural haematoma parallels the gradual increase in intracranial pressure. Chronic subdural haematoma usually manifests initially with non-specific symptoms such as headaches or mental concentration disturbances, but when the volume of this chronic subdural haematoma increases the patient develop symptoms of increased intracranial pressure, progressive dementia or focal neurological deficits. Most of symptoms and signs associated with chronic subdural haematoma are non-specific and global.In patients with a preexisting neurological or psychological disease, symptoms are usually exacerbated. Clinical presentation typically falls into three groups: focal neurological deficit, features of raised intracranial pressure and alteration of cognitive functions.

Most common focal neurological symptoms include hemiparesis and difficulty in speaking. Occasionally, these findings may fluctuate, simulating transient Ischaemic attacks (30, 31). Pupillary inequality and 3rd nerve palsy are less common and sensory loss is rare. Other less

common symptoms includes ataxia, tremors and dystonia (31).

Headache is by far the most common manifestation of raised intracranial pressure and is also the most frequent isolated complaint. It may be a accompanied by nausea, vomiting, neck stiffness or visual obscuration. Evidence of papilloedema is present in one-third of cases.

Cognitive changes may range from mild attentional disturbance to delirium or dementia. Some of these subtle cognition changes include inattention, reduced concentration and slowed processing speed. More pronounced cognitive changes include disorientation, florid confusion and general intellectual impairment (9). Patients may also present with impairment of recent memory, language, abstract thinking and judgment as well as personality change (5).

Behavioral changes are frequently noted by family members which includes sleep-wake cycle disturbances, emotional outbursts, maniacal and depressive states, altered psychomotor activity, lethargy and giddiness.

Other clinical presentation includes seizures and coma. The presentations of chronic subdural haematoma in the elderly is clearly different from that seen in younger adult. In elderly common presentations are cognitive dysfunction and focal neurological deficit whereas in younger patients features of raised intracranial cranial pressure are common. The length of time between trauma and onset of symptoms increases with age.

The symptoms associated with chronic subdural haematoma can mimic other medical disorders such as dementia, stroke, transient ischemic attack, neoplasms and psychiatric illness. As chronic subdural haematoma is a diagnosis of exclusion. A careful history of the clinical symptoms is one of the most valuable means of accurate diagnosis.

1.2 PATHOGENESIS

In 1857 Virchow, showed that the chronic subdural haematoma was due to inflammation of the dura mater secondary to infection and he called it pachymeningitis hemorrhagia interna. Later

several studies demonstrated that this process was due to local inflammatory reaction of the dura mater in response to blood, fibrin or fibrin degradation products. The initial event in the formation of haematoma is the tearing of the bridging vein in the subdural space causing blood to collect with in the dural border cell layer. This is usually a traumatic event, but non-traumatic origins of subdural blood and fibrin accumulation are also known. It includes arteriovenous malformation, dual based tumors and infectious diseases. In addition to trauma and non-traumatic causes of subdural haematomas, various other factors may increase the vulnerability of the bridging veins to tear, which includes low intracranial pressure, cerebral atrophy and coagulopathies. Chronic hygroma has shown to be a precursor of chronic subdural haematoma (26, 34).

Once the haematoma is formed, dural border cells proliferate and forms a pseudo membrane. This membrane contains activated inflammatory cells and immature vessels. Activated inflammatory cells may play a role in the angiogenesis, increased vascular permeability, hypercoagulative activity, hyperfibrinolytic activity . The reason for the development of a chronic subdural haematoma and especially its expansion , are still not fully understood. Repeated micro-haemorrhages from the fragile sinusoidal vessels in the outer membrane were found to be the source of haemorrhage. H. Murakami et al (16) thought that vessel damage was caused by transmitted brain pulsation, change in head position and head injury as a cause for repeated haemorrhages. Excessive activation of both the coagulative and fibrinolytic system, (17,19,40) high expression of tissue-type plasminogen activator in haematoma's,(42,48) presence of thrombomodulin (16) have been proposed as a possible explanation for the failure of haematoma

to coagulate . Interleukin-6, Interleukin-8, (3) vascular endothelial growth permeability factor (46) may be involved in the pathogenesis of chronic subdural haematoma. Currently, chronic subdural

haematoma is considered a chronic self- perpetuating inflammatory process that involves the dura mater (1,39,40,46).

1.3 DIAGNOSIS

If chronic subdural haematoma is suspected, CT Brain is the usual method of diagnosis. This demonstrates a uni-loculated extra axial hypodense, isodense or mixed density collection which may extends over the hemisphere. Haematoma may be multi-loculated with high density septum running between the inner and outer membrane of the haematoma cavity. MRI is beneficial for differentiating fluid collections containing blood breakdown products from effusion, however, it is more time consuming, less readily available, more expensive and has little obvious diagnostic advantage for most cases of chronic subdural haematoma. It is useful in bilateral isodense haematomas. In infants with open fontanella, ultrasound examination can be used to demonstrate the subdural haematoma.

1.4 TREATMENT

The need for treatment is based on the clinical signs and symptoms. Asymptomatic haematomas without mass effect on CT scan can be observed, as some of these haematomas can resolve spontaneously(33). However, these haematomas also have the potential to enlarge and these patients do require close observation. Medical treatment of symptomatic chronic subdural haematoma with high dose of mannitol has been reported with 70% success(41). Because of the incidence of electrolyte imbalance and renal failure, this treatment method was not accepted.

The standard treatment of chronic subdural haematoma in symptomatic patient is surgical evacuation. Despite the general agreement concerning the indication of operative treatment, the optimal surgical treatment of chronic subdural haematoma is still controversial. In infants subdural tap by insertion of a needle into the lateral aspect of the fontanel was proposed(43). Subdural peritoneal shunts, subdural subgaleal shunts has been advocated in persistent collections.

In adults various surgical methods were recommended. Most author recommends(10,27.48) burr hole drainage with closed system drainage. Three studies compared burr hole drainage with and without closed drainage system and found less recurrence rate with closed drainage system(28,48). Twist drill drainage offers an alternate method to the treatment of chronic subdural haematoma (6,37,42). Both these procedure offers least invasive and comparable morbidity and mortality. Craniotomy with removal of the membrane was once thought to be required in all cases, but now it is reserved in multi-loculated heamatoma, presence of solid component and recurrence(35). Craniotomy has least risk of recurrence but with higher complications. Sambasivan et al (38) had remarkable experience with subtemporal craniotomy with 0.5% mortality and low recurrence. Combining each approach with the use of intra operative irrigation or the use of drainage provides variety of treatment options. Aoki (3) reported filling of subdural space with 100% oxygen after haematoma drainage, while kitakami (21)used carbon dioxide to reduce the incidence of recurrence.

Several newer treatment options have also been discussed in the literature. Yoshimoto et al advocated a small craniotomy at the superior lateral angle of the forehead just beneath the hair-line (50). Helling et al and others have advocated endoscopic treatment of chronic subdural haematomas especially in septated lesions (8, 15). It is evident that the goal of treatment is

minimally invasive while at the same time preventing recurrences.

1.5 PROGNOSIS

Clinical outcome is based on mortality, morbidity and recurrences. Recent studies have shown that mortality to be between 1.5 % to 8 % (6,22,26). Van Haven burg, et al (45) defined morbidity as Glasgow outcome scale score of 2 to 3 at discharge. Morbidity ranges from 6 to 25 % (2,10,26,42). In a study Mc Kissock, et al (30) and later Kotwica et al (22) demonstrated poor outcome with increased age above 60 years. This was related to presence of co-morbid condition and increase use of anti-thrombotic medication. Although early studies by Diamond, et al (7) and mattle et al

(25) found an increase in mortality in chronic subdural haematoma patients treated with anti-coagulant recent studies has discounted it (20,49).There are no definite data in the literature that shows any difference in prognosis between trauma and non traumatic cases of chronic subdural haematomas. Van Haven burg et al (45) found a very significant correlation between good pre-operative grading and good Glasgow outcome score. Longer duration of symptoms and treatment is significantly associated with mortality(36). Likewise early diagnosis and surgical intervention has a positive effect on outcome (28). The haematoma size, midline shift and bilateral haematoma on the CT image by itself has no prognostic importance (37,44).

Different surgeons use different operations in different situations, some prognostic trends are associated with the various treatments. Craniotomy is associated with greatest mortality up to 30% (22, 14). Burr hole drainage and twist drill drainage are associated with the lowest.

The recurrence rate varies substantially between studies and treatment methods. It usually varies from 0% to 33% (27,48). Symptoms of recurrent cerebral compression due to chronic subdural

haematoma usually develop in 1 to 7 days (most often in 2 – 3 days) after surgery. Prolonged failure to improve the symptoms may indicate haematoma recurrence.