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A review of the management of chronic subdura haematoma

Morgan E[1,2]
Adetunmbi B[3]
Ofuadarho C[2]
Poluyi E[4]

[1]Department of Surgery, Ambrose Alli Uiversity, Ekpoma, Nigeria
[2]Department of Surgery, Irrua Specialist Teaching Hospital, Irrua. Nigeria
[3]Department of Surgery, Federal Medical Centre, Abeokuta, Nigeria
[4]Neuroscience Division, University of Roehampton, London, United Kingdom

***For correspondence:**

Tel: +2348035724104
Email: morganeghosa@gmail.com

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Abstract

Chronic subdural haematoma (CSDH) commonly refereed as the great mimicker and it is a slow evolving blood collection located between the dura and the arachnoid layer which is surrounded by a capsule. It mainly affects elderly male individuals. Computerized tomography scan of the brain is the investigation of choice and surgery is the mainstay of treatment. Recurrence of CSDH is the most common complication and this is an important point in neurosurgical parlance and accounts for 10-20% of post-operative complications.

Keywords: Subdural, haematoma, surgery, recurrence

Introduction

Chronic subdural haematoma (CSDH) is a slow evolving encapsulated blood and its breakdown products collection in a potential space called the subdural space, which is anatomically located between the dura mater and the arachnoid [1]. Wepfer [2] first described this disease entity in 1657, not until In 1857 when Virchow [2] gave a detailed description on the pathophysiology of the haematoma, naming it pachymeningitis haemarrhagica interna.

Chronic subdural haematoma incidence have been noted to rise with increasing age. It is about 3.4 per 100,000 in those less than 65 years, while in those above 65 years, it is about 8.58 per 100,000. [1] Though, it is seen in younger age-group in Africa,[3] however, with slightly increasing elderly population in Nigeria, highest incidence is between 50-60year age-group comparable to the developed world with female to male ratio of 1:3[3].

Risk factors for CSDH has been widely studied ranging from advancing age due to global brain volume reduction and bridging veins increasing risk of tear, anticoagulants such as aspirin, alcohol ingestion, trauma seen in 30-50% of cases and epileptic among others. Generally, two theories have been propended in the pathophysiology of this disease. These are – the recurrent bleed from haematoma capsules and the osmotic theories, with the latter being more widely accepted.[2]

This pathology commonly known as “the neurological mimicker” [4], presenting a diagnostic challenge in neurosurgical practice as it is unusually suspected at presentation. Clinical presentation include headache, altered consciousness, seizures, aphasia. unusual neurological presentation without or without history of background trivial falls, thus requires a high index of suspicion prior to brain imaging.[2]

Cranial Computed Tomography Scan (CT-Scan) is investigative modality of choice the gold standard in diagnosing CSDH [5], defining it in terms of density as hypo-dense subdural collection, compared with the brain parenchyma, and clinically, it usually presents over 21 days after insult.[1]

Although both operative and non-operative options of treatment have been employed, surgery is the mainstay

of treatment thus improving neurological status and quality of life. Outcome post-operatively is good. [5,6] CSDH recurrence is an essential topic of discuss post-operatively and it is the most common complication following surgery accounting for 10- 20% of the post-operative complications [7,8]. Adeolu et al reported a value as high as 28.8%. [9]

Historical perspective

Several studies have been done as regards CSDH, Yet, full description of this entity still remains debatable. Johann Jacob Wepfer in 1657 pioneered the early description of this pathology [2,18]. Balzac in 1840 [19] in his monogram-Pierrette describes a posttraumatic chronic subdural haematoma, and the attendant operative intervention. However, this case report emanated at a time all pathology including CSDH were ascribed to be inflammatory in origin.

Virchow in 1857 [2] wrote extensively on the pathophysiology of the CSDH. He named the disease "pachymeningitis haemarrhagica interna". However, Cushing in 1925 [18] popularized the concept that "insignificant trivial trauma often forgotten by patient or from amnesia was a possible cause of CSDH" and this concept was generally accepted by the end of the twentieth century. The ancient practice of skull trephination was also noted historically [3] alluding to the fact that surgery might had been done for patients with CSDH in those periods. In the early 20th century, Putnam and Cushing [18], lay more emphasis on operative treatment for CSDH as mainstay of treatment, a fact which has not changed till date. With advances in neurosurgical imaging modalities, such as the pneumoencephalography, angiography as well as advent of Brain CT scan allowed the early diagnosis of CSDH.

Symptom evolution have also changed to headache and focal neurological deficit from mental apathy and coma in the extreme form as earlier described. Further advances in training and knowledge of neurosurgery saw a period where procedure became less invasive and minimal with less morbidity and mortality and haematoma removal still remaining the main goal of surgery. The place of closed drainage system in operative procedure has been noted to aid brain re-expansion of the brain post-operatively.

Epidemiology

It is a relatively common pathology that present to the neurosurgical unit. The annual incidence is about five per 100,000 per population but higher in those above 65 years, and noticed to rise to about 58 per 100,000 per annum. However, delays in diagnosis and treatment have been noted in our environment due to paucity of neuroimaging tools and scarcity of trained neurosurgeons [10,11].

Worldwide, an increase in incidence over time have been noticed. In Helsinki, it is about 1.7 cases per 100,000 people, during mid-half of last century [12]. while, in Japan, it is about 13.1 cases per 100,000 people from 1986 to 1988 [13], but in 2005, it rose to 20.6 cases per 100,000 persons. [14] and higher in the very old groups. On those over Eighty years, who comprise 1/3rd of the population with this pathology, it is 127.1 cases/100,000 people. [15] Incidence rate in the United State of America (USA) is also increasing according to Balser et al, at 79.4/100,000 persons.[14]

The increase life expectancy in the developed world has also brought this pathology to the front burner. In the Japanese population, with a higher life expectancy compared to elsewhere, coupled with the current medical trends with patient on anticoagulant, and/or anti-platelet therapy and haemodialysis has results in the rise increase incidence in the elderly with M:F=3:1, mean age was 71.2±12.8years and total incidence of CSDH of 20.6/100,000 per year. 76.5% in the age group of 70-79 years. [15]

Report from Africa study suggest CSDH were seen in younger population with an average age of 49.66 [16] but studies by Mezue et al [17] in Enugu Nigeria shows the highest peak in the 50-60 years of age with M:F= 3:1 which is comparable to results from studies done by Hode et al [16] but noted to be on the increasing trend.

Aetiology/risk factors

Age: The elderly patients are more susceptible to present with CSDH particularly from trivial forgotten trauma. Widespread brain volume reduction with associated increase risk of bridging vein shearing and tearing seen in aging population and younger age group who are chronic alcoholics are predisposed to having CSDH [2,20]. With advancing age, the cerebral volume reduces, with corresponding increase in the cranio-cerebral space from the usual 6% to 11%. This results in shearing effect of the bridging veins. The resultant increase craniocerebral space makes these veins vulnerable to trauma due to frequent intracranial content movement.[2]

Gender: It is widely acknowledged that males are more affected compared to females, The real reason for this have not been stated in known studies conducted.[1,3,10] It may probably be due to the increasing risk of exposure of male to trauma and oestrogen in female having protective effects on the capillaries.[21]

Trauma: A study by Sousa et al[1] in Brazil determining the origin of the CSDH in 778 patients, 497 patients

(63.9%) had known trauma while origin remained unclear in 281 patients (36.1%), which is similar to results obtained by Bankole et al [10] in Lagos Nigeria where 63.5% had history of trauma. Falls was identified the commonest cause of CSDH in patients aged 65 years or older seen in 154 cases of cases.[1] This is unlike in Nigeria where motor vehicular-motorcycle accident is the most frequent origin of brain trauma.[10] this is more were more frequent in younger patients which may be most likely due to progression of previous acute subdural hematoma (ASDH) missed or managed non-operatively.

Post Neurosurgical Procedure: CSDH has been observed to result from neurosurgical interventions, such aneurysmal clipping surgeries,[22] superficial temporal artery to middle cerebral arterial bypass surgery such as superficial to middle cerebral artery, or surgery for arachnoid cysts,[23] placement of lumbar drain for control of raised intracranial pressure in patient with idiopathic intracranial hypertension, and for post-operative cerebrospinal fistula,[23] or over-drainage post-operatively in ventriculo-peritoneal shunting or endoscopic third ventriculostomy.[24-26] These may probably suggest that intracranial interventions that breach cerebrospinal fluid (CSF) space or results intracranial hypotension from most likely CSF fistula may result in CSDH post-operatively.[25]

Also in the elderly with brain atrophy, there is enlarged subarachnoid space and shearing of the bridging veins, with associated tearing of the arachnoid, leakage of bloody CSF into the subdural space after mild head injury or a craniospinal operation that produces over drainage of CSF from the subarachnoid space or the ventricular system can result in CSDH.[26] Diminished elastance of the brain coupled with reduced capacity for brain re-expansion also may be implicated. This is commonly seen in patients with old infarct resulting in localized cerebral atrophy, or in those with a global cerebral involution. Factors such as subdural air post-operatively could affect brain re-expansion after a craniospinal surgery and should be avoided [27].

Spontaneous: Chronic anticoagulation therapy have been identified to increase the chances to developing CSDH. Although, this is poorly understood, it has been opined that those without symptomatic who have microbleeds with this evolving to formation of significant CSDH.[28] Anticoagulants are routinely used in a large number of the elderly population with pre-existing co-morbid condition, may lead to an increase risk of CSDH with values as high as 42.5 times.[28] and research have shown that patients on anticoagulant represent due to high chances of recurrent CSDH.[29]

Pathophysiology

The anatomy of meninges is of great value, in which there exists a potential anatomical space between dura and arachnoid known as the subdural space. Subdural haematomas often result from the blood effused into this subdural space. Histologically, under high power electron microscope, the meninges were shown to revealed absent of the subdural space bur revealed the innermost border of the dura mater (the dura border layer) and the outermost border of the arachnoid (its barrier layer) which are tightly packed and attached to each other. these two membranes are tightly attached to each other.[30]

It is widely accepted that CSDH arise from the initial trauma resulting in the shearing of the bridging veins which leads to bleeding into this space. Twenty-four hours after the bleeding into the subdural space. a thin fibrin and fibroblast layer cover the outer surface of the haematoma. By the fourth day, the fibroblast proliferates and migrate resulting in the formation of haematoma membrane over the clot. However, the outer membrane enlarges steadily and the proliferating fibroblasts invade the cavity of the haematoma forming an inner membrane over a period of two weeks.[31] The proliferation of the phagocytic cells allow the haematoma liquefy. The haematoma may spontaneously resorb or gradually increase in size [31].

Theories of CSDH

Two main theories have been proposed to explain the pathophysiology of chronic subdural haematoma. These are - the osmotic theory and the recurrent bleeding from the capsule of the haematoma, though each still controversial.

Theory of Recurrent Bleed: The initial thought was the theory of recurrent bleeds from studies done by Friede et al[30] which was later challenged by Gardner after performing experiment in dogs using semipermeable membranes that separate CSF from the blood, He propanded an osmotic theorem: he stated that the arachnoid membrane is semipermeable and the cerebrospinal fluid is drawn into the hemorrhagic cyst by the osmotic gradient of the plasma proteins.

Osmotic Theory: This was based on the hypothesis that the liquefied haematoma increases the plasma protein component and the resultant oncotic pressure of the fluid in the haematoma capsule. This cause the movement of fluid from the surrounding vessels into the haematoma cavity due to osmotic pressure gradient across the semipermeable membrane [32].

The theory was further substantiated by Weir [33] who challenged the osmotic theory. He further revealed that the haematoma fluid osmolality was identical to that of blood and CSF.[33] Recurrent bleed from the capsule is the well documented and accepted theory. This capsule has been shown to have abnormal dilated vessels, often

refered as the origin of the haematoma.[34] Ito et al [35] study support this theory. 51Cr-labelled red cells was administered intravenously 6-24 hours by the researchers before the haematoma was evacuated by the neurosurgeon. It showed that 0.2%–28% of the haematoma was fresh blood. Studies have also shown that there is an increase assay of abnormal coagulation profile and fibrinolysis during the evolution CSDH.[36].

There may be intracranial hypertension. The reduced brain volume and absent of tamponing effect often result gradual increase in CSDH. The quality of the collection may vary between fresh clot to alters blood and watery depending on the duration and how recurrent the haematoma.

Classification

Classification of CSDH can be clinical and radiological. Clinical classification is based on duration of haematoma (< 3days referred to acute, >3-21days referred to as subacute and > 21days referred to as chronic subdural haematoma) and clinical presentation which has had several classification system CSDH should be differentiated from acute subdural haematoma. Acute subdural haematomas often follows trauma in young individual with associated underlying cortical injury. Radiologically, it is based cranial computed tomography (CT) scan as CSDH is confirmed as a hypodense extra-axial crescentic collection subdural haematoma.it usually presents 21 days after trauma.[1]

Markwalder in 1981[37] classify CSDH based on findings from clinical evaluation as follows:

- Grade 0: asymptomatic
- Grade 1: alert, oriented, mild symptoms (such as headache)
- Grade 2: drowsy or disoriented, variable neurological deficits (hemiparesis)
- Grade 3: stupor but responds to stimuli, severe focal signs (hemiplegia)
- Grade 4: coma (GCS<8) without motor response to painful stimulus, flexes/extends to pain

Grades 0-2 have a favourable outcome and Grades 3 and 4 have an unfavourable outcome. This help to prognosticate and counselled appropriately.

Other scores frequently used for follow-up evaluation of CSDH patients include, the Glasgow Outcome Score [38], and the modified Rankin scale (mRS) [39].

Nakaguchi and colleagues in 2001 [40] classify the haematoma based on its internal architecture.

- Homogeneous haematoma was defined as haematoma exhibiting homogeneous high-density.
- Laminar type haematoma was defined as a subtype of the homogeneous type with a thin high-density layer along the inner membrane.
- Separated type haematoma was defined as a haematoma containing two components with different densities with a clear boundary between them; that is, a lower density component located above a higher density component.
- Trabecular type haematoma was defined as a haematoma with heterogeneous contents and a high-density septum running between the inner and outer membrane on a low-density to isodense background.

This classification system helps to predict the rate of recurrence. As recurrence rate is less with homogenous compared to trabecular type which has the highest rate of recurrence.[8]

Clinical features

CSDH more often mimic a wide variety of intracranial illnesses. It may be asymptomatic or symptomatic [4]. The clinical presentation depends on the age and condition of the patient, the site, size, and rate of growth of the haematoma [42] this presentation are non-specific and it can he headache which is often most common symptom. Others include, seizure, focal neurological deficit and few with impaired level of consciousness. There may be background history of trivial trauma most especially in those >65 years, use of anticoagulant, chronic alcoholism [1,10,21,43,44]. The site of CSDH may modifies the presentation. Haematoma at the skull base or in the posterior cranial fossa are uncommon. the interhemispheric collection has been noted in many patients with marked weakness of the lower limbs compared to upper limbs [44] some maybe bilateral CSDH and present with bladder and bowel paresis co-existing with para-or quadriparesis mimicking cervical cord compression[44,45] Shields et al [45] opined that congestion and kinking of the superior rolandic veins compromising venous drainage and causing localised decreased cortical function may likely explained the para-quadriparesis. A positive correlation between the size of the CSDH and those with neurological symptoms was found by Aronson et al [46]. Similar relationship was seen in same study with fatality. There was also some correlation between the volume of bleeding and the percentage of fatality.

Headaches in chronic subdural haematoma have been characterized differently. It could insidious, localized or generalized, few occasions it could headache characteristic if intracranial hypertension. Mental disturbances at the onset maybe of poor alertness, followed by confusion and impaired memory which can progressively to dementia in the absence of treatment.[47] visual manifestations in patients with CSDH varies. Mitsumoto et al [48] reported anisocoria in

24% of their cases, homonymous hemianopsia in 22%, papilloedema in 12%, lesion of the third cranial nerve in 5%, of the sixth cranial nerve in 3%, However, Brihaye's series reported ipsilateral anisocoria in two patients. These clinical manifestations, ipsilateral to the side of the haematoma, are due to the lateral compression of the midbrain resulting in the contralateral half being pressed and contused against the rigid edge of the tentorium cerebellum (Kernohan's notch phenomenon). Seizures can be generalized or focal, either before or after drainage of chronic subdural haematoma [41-43,47]

Bankole et al [10] reported clinical features similar to earlier studies, However, this distribution is different from what Ohaegbulam [44] observed in his study at Enugu Nigeria. He reported that hemiparesis/hemiplegia occurred in 85 (64.3%) cases as the most frequent complaint, impaired consciousness at the time of diagnosis in 75 (56.8%) cases while headache which occurred in 55 (41.7%) cases is the 3rd most common symptom. His study, though different from others, is similar to review done by Sambasivan [21] on 670 cases of CSDH, where 29.1% of patients presented with hemiplegia which was the highest. These haematomas may be of very long duration, sometimes up to 35 years in the case reported by Debois and Lombaert [49] and this is often seen in less than 18 years of age,

Investigation

Cranial Computed Tomography Scan (CT-Scan) is the investigation of choice in patient with CSDH.[5] It appears as a crescentic shaped hypodense collection in the subdural space [5,10] (Figure 1). A subacute subdural haematoma can be nearly isodense with the adjacent brain. Certain. CSDH of more than 3weeks old are typically seen as crescentic shaped hypodense subdural collection.

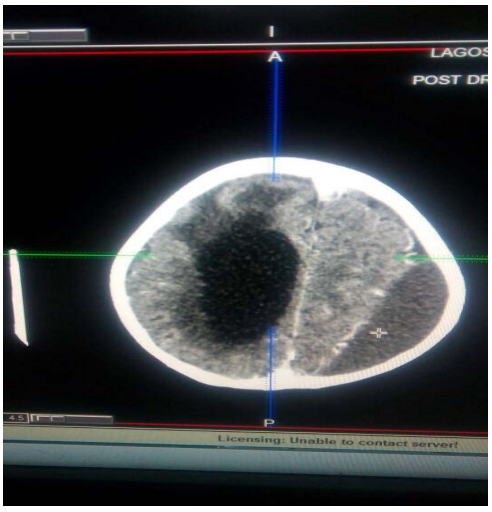

Figure 1: Brain CT scan showing a left sided extra-axial crescentic shape chronic subdural haematoma in a 68-year old who has trivial fall

Cranial magnetic resonance imaging (MRI) scan is useful to demarcate the various phases of the subdural haematoma and provides detailed information of the site, size, dimension, age and complexity of CSDH. However, due to limited availability, increased cost and acquisition often complicate it routine use.[10]

Historically, several investigative tried in the past with varying demerit and demerit. Plain skull radiograph which sometimes clearly demonstrates the CSDH, owing to the presence of calcification and/or ossification of the membranes have been used but it cannot reveal intracranial haematoma; Electroencephalogram (EEG) may occasionally indicated the hemisphere over which the haematoma is isolated, although it may be normal, especially in small volume CSDH [44]. Also used were Echography previously used which could show midline shift pointing ipsilateral hemispheric lesion which could be CSDH. Brain Scintigraphy have also been tried with afrmore accuracy compared to the former. The penultimate test before the advent of Brain CT scans is the angiography which can reveal subdural haematoma showing acute and chronic form when no computerized scanner was available. Arteriography till date still has a role[44] in case of isodense bilateral subdural haematoma.

Treatment

CSDH can be treated via operative and non-operative options of treatment, However, the mainstay of treatment is surgery. Surgery leads to rapid improvement of neurological symptoms. Postoperative prognosis is generally good [5,51]. Different operative options for treatment of CSDH have been devised but still remains a controversial issue with different studies for and against each. Burrhole (BH) craniostomy, twist drill craniostomy and craniotomy, with different claims to their successes as regards recurrence [18,51-53].

Burrhole craniostomy is the most common and popular surgical option for treatment of CSDH across the globe.[42,55,56] In 2006, a research in the United Kingdom and Ireland found that 92% of neurosurgeons preferred and opted for burrhole craniostomy.[55] This surgical intervention have been noticed to be competitively efficacious in the elderly, with a 2012 retrospective study showing neurological outcome and quality of life improvement of as much as 83% in the 65–74 age range of the 322 patients by the Rankin Scale.[42] Most of the current literature regarding burrhole craniostomy is in the refinement of technique whether single of multiple and also whether drain is used or not and the practice of nursing patient in supine position post-operatively and mobilized later period of care to reduce incidence of recurrence.[37,55-58]

Supine positioning and delayed mobilisation have been noticed from studies to help in brain re-expansion, thereby reducing dimension of subdural space and in effect reduce risk of recurrence.[59] Post-operative positioning of CSDH patients has been the subject of recent investigations. Assuming head-up positioning in the early post-operative period have been note from few studies not to be is not associated with increased risk of recurrence [59,60] yet a contrary finding was noted by Abouzari and his colleagues [61].

A study by Adeolu et al [9] showed insignificant difference between early and late mobilisation post burrhole drainage, but it was advocated that early mobilisation of patients may be beneficial in reducing the length of hospital stay with attendant complications.

There are quite a number of researches in selected patients who benefitted from non-operative (medical therapy) management of CSDH. Medical therapies for CSDH rely mostly on steroids(dexamethasone) although other agents such as atorvastatin were examined as well.[50,62] A proforma evaluating the various treatment modalities for CSDH in the UK, one-quarter of the patients were conservatively managed, of which,55% was treated with steroids.[55] the role of steroid was further buttressed in an article published by Thotakura and Marabathina.[50] Steroid role in the treatment of CSDH is based on the inflammatory response in the pathophysiology of CSDH. It has also been noted that steroid disrupts the dura border cells and triggers a cascade of inflammatory reaction which leads to border cell recruitment, proliferation, migration with associated macrophage and granulation tissue deposition.[63] Expansion and neovascularization are additional inflammatory response, [64,65] that often leads to formation of membrane and recurrence of CSDH and this complicate treatment. A research by Adeleye, [66] is also a pointer to the possibility for steroid use in selected patients with CSDH. Hashimoto and co-researchers showed great success in the management of recurrent, recalcitrant and refractory CSDH with the embolization of middle meningeal artery which is believed to be feeder to the re-bleeding outer capsule wall in advancement extirpation of the artery [67].

Conclusion

Chronic subdural haematoma is a relatively common neurosurgical pathology in the elderly, and it surgery is the mainstay of treatment, though encouraging results are currently observed from selected group of patient managed conservatively.

List of abbreviations

CSDH: Chronic subdural haematoma; CSF: cerebrospiral fluid; CT: Computer tomography; CT-Scan: Computer tomography scan; mRS: modified Rankin scale.

Declarations

Ethics approval and consent to participate

Not applicable

Consent for publication

Not applicable.

Availability of data and materials

The data and materials used in this study are available from the corresponding author on request.

Competing interest

No conflict of interest is associated with this work.

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Contribution of authors

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