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INTRODUCTION

The first clear account of chronic subdural hematoma (CSDH) was given by neurosurgeons of Paris in 1817. In 1938 Pavidoff and Dyke published their classic treated chronic subdural hematoma. In the pre-computed tomography (CT) era the diagnosis was largely clinical and many patients died undiagnosed or suffered from persistent disability because of delay in diagnosis and treatment.(1)

Chronic subdural hematoma is a localized accumulation of blood in the subdural space which manifests itself from three weeks to several months after injury.(1)

Classically CSDHs contains dark "motor oil" fluid which does not clot. When the subdural fluid is clear (CSF), the collection is termed a subdural hygroma.(2)

The mean age of onset is 63 years. It has an incidence of 1-2 cases per 100.000 inhabitants per year, its incidence increases (7 / 100.000 individuals) in persons older than 70 years of age. The highest risk of CSDH exits in comatose patients and in elderly. The mortality rate of CSDH treated by drainage ranges from 0 to 8% and the accompanying conditions such as cardiac diseases are the major causes of death. The recurrence rate of CSDH after surgery ranges between from 3% to 27 %.(2)

Anatomy of the subdural space

The subdural space is a potential space between dura matter and arachnoid; this space contains a film of serous fluid which moistens the surfaces of the opposed membranes. (3)

It does not appear that this space communicates with the subarachnoid space, but it is continuous for a short distance on the cranial and spinal nerves, it is in free communications with the lymph spaces of the nerves. It is continuous around the optic nerve as far as the back of the eye ball; numerous vessels cross this space connecting the cerebral cortex, arachnoid with the extradural space (bridging vessels), the para-sagittal vessels called Mittenzweig`s vessels.(1)

Anatomy of the cranial dura

The cranial dura is a thick and dense inelastic membrane lining the interior of the skull. It is said to be composed of two layers, an inner or (meningeal layer) and an outer or (endosteal layer); the two layers are closely united except along certain lines where they separate to enclose venous sinuses which drain the blood from the brain.(4) The meningeal layer of the dura sends the following septa inside the cranial cavity:

1. The falx cerebri

It is a strong sickle-like process attached anteriorly to the crista galli, posteriorly to the upper surface of the tentorium cerebelli, (where the straight sinus runs along). It has an upper convex margin (where the superior sagittal sinus passes) and an inferior concave free border (where the inferior sagittal sinus runs along).(3)

2. The tentorium cerebelli

It is a crescentic arched lamina of the dura matter between the cerebellum and occipital cerebrum. It has an inner concave free border and convex outer border attached to the occipital bone posteriorly and to the superior border of the petrous part of the temporal bone laterally.(3)

3. The falx cerebelli

It is a small dural space situated below the tentorium cerebelli and projects forward into the posterior cerebellar notch.[3]

4. The diaphragma sellae

It is a small circular horizontal fold of dura matter which forms a roof for the sella turcica and almost the hypophysis.[3]

Structure of the dura matter

It is basically fibrous white collagen fibers predominating with a mixture of elastic fibers. The collagen fibers are arranged in laminae in which the fibers are often arranged in a parallel manner with wide angles between these grouping in adjacent laminae producing latticed appearance particularly easy to see in the tentorium cerebelli. At all foramina in the cranium the endosteal layer is continuous through them with the external periosteurn.[3]

At the sutures before their fusion the endosteal element is continuous with sutural membrane and the dura is more strongly attached at these locations, elsewhere it is more easily detached from the cranial bone. It is important to mention that the meningeal layer contains a very fine interconnected capillary network with ampulla like dilatation of the capillaries. In contrast to this, the arachnoid has no significant vascularization. This capillary network has a very high reaction potential which is entirely more specific for it, leads to formation of a neo-membrane when it comes in contact with blood, fibrin or fibrin degradation products.[4]

The arteries of the cerebral dura mater are numerous; those in the anterior cranial fossa are branches of anterior and posterior ethmoidal arteries, internal carotid artery and middle meningeal artery.[3]

Those in the middle cranial fossa are branches of maxillary artery, ascending pharyngeal artery and internal carotid artery. Those in the posterior cranial fossa are branches of occipital artery, vertebral artery and occasionally of the ascending pharyngeal artery.[3]

The cerebral arachnoid mater

The cerebral arachnoid is a delicate membrane enveloping the brain and lying between the pia mater internally and the dura mater externally. The arachnoid surrounds the cranial nerves and encloses them in loose sheathes as their points of exit from the skull. The arachnoid surround the cranial nerves and encloses them in loose sheathes as far as their point of exit from the skull. The arachnoid invest the brain loosely and does not dip into the sulci between the gyri or into the fissures.[3]

There is a thin film of tissue fluid lying between the dura and the arachnoid, that is to say vessels and nerves pierce the dura mater and the arachnoid both at the same time and never run along between the two membranes.[3]

Pathology and pathogenesis

Chronic subdural hematomas are clearly delineated fluid collections located between the dura mater and the arachnoid. The origin of blood accumulation within the subdural space may be traumatic or atraumatic. Traumatic causes either by direct trauma to the cranium such as acceleration injures with tearing of the para sagittal bridging veins or Mittenzweig`s vessels, by movement of the brain in relation to its coverings. Also it occurs with traumatic arachnoid cysts.[3, 5]

Non traumatic origin of subdural blood accumulation are convexity arterio-venous malformations and aneurysms, other cerebrovascular lesions, hemorrhagic diathesis, infectious diseases, brain tumors specially convexity meningiomas and meningeal carcinomatosis. [6, 7, 8, 9]

In addition to traumatic and atraumatic causes of chronic subdural hematomas, various other factors may increase the vulnerability of the bridging veins or Mittenzweig' s vessels as the patients tendency to bleed, the hematoma size and mechanical factors including low intracranial pressure (I.C.P), cerebral atrophy and excessive deformation of the cranial vault in infancy or during delivery. Low intracranial pressure may occur in disease related to traumatic cerebrospinal fluid fistulae as a result of lumbar puncture, due to iatrogenic or diseases including dehydration and after implantation of or a cerebrospinal fluid (C.S.F) Shunt.[10,11]

Low ICP promotes excessive blood congestion of the bridging veins with subsequent dilation and increases tension of the vessels which are further stretched by a downward displacement of the brain and consequently become more vulnerable to the movement of the brain within its coverings due to decreased cerebral volume. Minor subdural hemorrhage is not immediately stopped because of the reduced counter pressure of the cerebral hemispheres; the same principles are valid in cases of cerebral atrophy, and external hydrocephalus, where the CSF is present in excessive amount on the surface of the hemispheres. Susceptibility of the infant skull to deformation is an additional mechanical factor promoting the development of subdural hematomas. [12, 13, 14]

The ectatic capillaries in the innermost layer of the dura matter in chronic subdural hematoma are believed to be similar to the vascular network of the skin in alcoholic hepatopathies and elevated estrogens levels.[14]

Consequently, these abnormal vessels would be responsible for repeated bleeding into the subdural space. However, in the majority of chronic subdural hematomas more than one of the previously mentioned contributing factors are present and they have accumulative effect. This explains the fact that chronic subdural hematoma is principally a disease of older age in which physiological brain atrophy, frequent head trauma and coagulation disorders due to therapeutic or prophylactic anti-coagulant therapy play accumulative role.[15,16]

Development of chronic subdural hematoma

The subdural bleeding is stopped by edema induced counter pressure of the brain and may be resorbed and organized if the subdural collection is not too large.[14]

This explains the greater incidence of chronic subdural hematomas associated with minor cranio-cerebral injuries without concomitant brain swelling and also in the older age group with its reduced cerebral counter pressure due to brain atrophy.[14]

The reasons for the development of CSDH and especially its increase in size are 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 disorders as well as excessive fibrinolytic activity in the cells of the membrane.[14]

Ito et al., stressed that the importance of local hyper-fibrinolysis which causes liquefaction of the subdural blood clot and continuous hemorrhage from the sinusoidal vessels of the neo-membrane in the development and enlargement of CSDH. Gradual increase of fibrinogen degradation products level from acute to chronic subdural hematoma suggests that fibrinolysis takes place in subdural hematoma and leads to intermittent hemorrhage and therefore to an increase in size of chronic subdural hematoma.[17]

The phenomenon of recurrent bleeding from the hematoma membranes as an etiological factor for development of chronic subdural hematoma was assumed by Dandy et al. [18]

This hypothesis was supported by experimental studies of Apfelbaum et al., in contrast to the osmotic gradient theory proposed by Thomas Markwalder. [14, 19]

According to the latter theory, a raised osmotic gradient causes the transport of cerebrospinal fluid into subdural sac after encapsulation of original hematoma and breakdown of its cellular constituents. When the pressure on either side of the sac was equal, the sac ceased to enlarge and the subdural fluid was slowly resorbed. [14]

Thomas et al. supported his thesis by serial assessment of the Protein content of the fluid which gradually increased and demonstrating that a cellophane bag containing subdural fluid increased 59 % in volume when immersed in container of CSF for 15 hours. When the same experiment was repeated with human subdural membrane instead of the cellophane container the result was less convincing, the increase in volume amounted to only 2.9%.[14] This osmotic theory has recently been questioned on the following four grounds:

- There was no significant increase in the volume in his model when the hematoma membrane was used.
- Fresh erythrocytes were always present in the hematoma fluid on repeated tapping
- it was not possible to demonstrate that the arachnoid acts as a permeable membrane for cerebrospinal fluid
- it has been demonstrated that albumin, the most osmotically active protein, cannot emerge from the destroyed red blood cells but is derived from the plasma.[14]

Weir et al. compared osmolality of subdural hematoma fluid, versus blood and cerebrospinal fluid and found no significant difference between them. The hemoglobin breakdown products were shown to migrate with alpha II and beta globulins, thus albumin moving from the blood stream to the subdural space act against the osmotic gradient postulated by Thomas. [14, 21]

Recently, Weir et al. compared the oncotic pressure of fluid from the subdural hematomas to that of simultaneously drawn venous blood in 20 Patients. He did not find a significant difference in the colloid pressure of fluid from subdural hematoma and venous blood, whereas, the oncotic pressure of fluid from subdural hygromas was significantly less than that of blood. [20, 21]

In 1970, Suzuki and Takaku were able to achieve resolution or marked reduction of hematoma fluid in 22 of 23 patients by repeated intravenous 20% mannitol. Some shift of fluid out of the hematoma may take place if mannitol does not cross the neo-vascular external subdural membrane. The latter theory is supported by the fact that introduction of

hyperosmolar substances causes the serum to have transient hyper-osmolality with respect to cerebrospinal fluid. [22, 23]

Stages of CSDH

According to internal architecture of CSDH, all hematomas were classified into four types which corresponded to possible stages in their natural history; homogenous, laminar, separated, and trabecular types.[24]

Stage 1: The homogenous stage, it is the stage in which the hematoma exhibits a homogenous density. During this stage the outer and inner membranes develop around the subdural space as the hematoma matures. Blood vessels derived from the middle meningeal artery are believed to extend into the hematoma membranes causing vascular congestion. Arterial pressure stresses the walls of the sinusoidal micro capillaries, resulting in minor, but persistent, subdural bleeding episodes. However, re-bleeding is moderate and the balance between coagulative and fibrinolytic activities is maintained.[24]

Stage 2: In this stage a thin high density layer is present along the inner membranes and is thought to consist of fresh blood from the hematoma membranes. The recurrence rate in this stage has increased to 19%. The higher recurrence rate of this type may be related to a greater vascularity than that of the purely homogenous type. [24]

Stage 3: the separated stage, as the hematoma matures, fibrinolysis occurs. The separated type was defined as a hematoma containing two components of different densities with a clear boundary lying between them; that is, a lower density component located above a higher density component. [24]

Stage 4: The trabecular stage, in which features of high density septa are created by fibrosis, the interstitial hematoma matrix changes from an isodense to a low-density signal on CT scans and on surgical inspection, it changes from dark reddish to xanthochromic translucent liquefied hematoma, which diminishes in volume over time. This is considered to be the resolution stage of CSDH. During this stage, the risk of bleeding from the hematoma capsule seems to abate and symptoms may not be related to bleeding from macro capillaries in the hematoma membranes but rather to cerebral congestion caused by the bulky CSDHs, so CSDHs originate in the homogenous stage and sometimes develop in the laminar stage. The hematoma becomes mature during the separated stage, and is finally absorbed during the trabecular stage.[24]

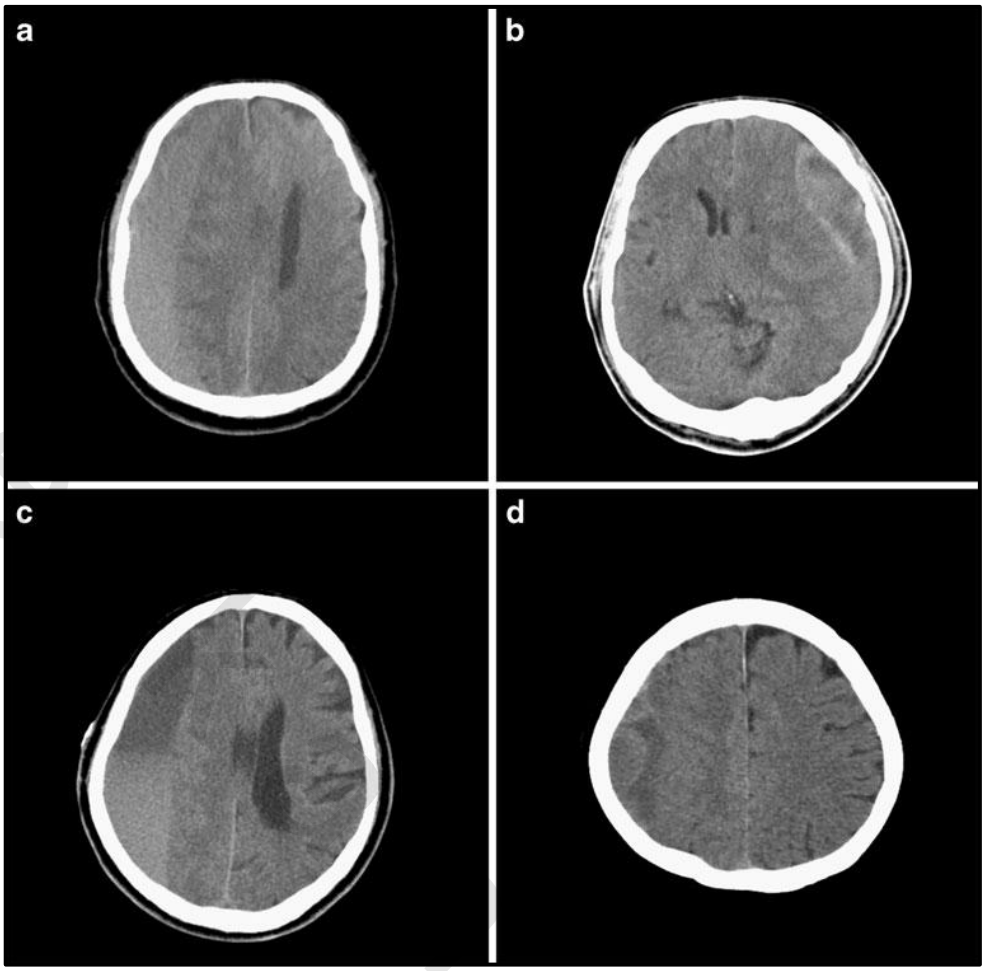

Figure 1: CT scans demonstrating classification of CSDHs according to their internal architectures. (a)The homogeneous type was defined as a hematoma that exhibited homogeneous density (low– high). (b)The laminar type was defined as a subtype of the homogeneous type that had a thin high-density layer along the inner membrane. (c)The separated type was defined as a hematoma containing two components of different densities with a clear boundary lying between them; that is, a lower density component located above a higher density component. (d)The trabecular type was defined as a hematoma wit inhomogeneous contents and a high-density septum running between the inner and outer membrane on a low-density to isodense background.

Causes of chronic subdural hematoma

Chronic subdural hematoma is encountered after trauma, in alcoholics and in patients maintained on hemodialysis. It may also complicate ventricular decompression, Leukemia and other blood disorders are occasional causes of subdural hematoma.[13-26]

Trauma: is the most common cause of chronic subdural hematoma. It may be either a major trauma sufficient to cause loss of consciousness or minor trauma not accompanied by loss of consciousness. It was reported that a significant proportion (37 %) was unassociated with known head injury. [14, 27]

Sometimes, the trauma is so trivial that it is forgotten easily. The trauma may be direct to the head or indirect such as fall on the buttocks, blast injury and even simple injury at home are recorded.[27]

Cameron reported in a review of 114 cases of chronic SDH that trauma was identified in 57% of his cases. No causative or precipitating factors could be identified in the remaining 43 % of patients.[26]

Alcoholism: CSDH is frequent in alcoholic patients because of their unsteadiness and frequent falls. Cameron reported 3 % of his series were alcoholics. [24]

Ventricular decompression: Extradural as well as subdural hematoma may complicate ventricular tapping or shunt operation due to sudden reduction of intracranial pressure leading to traction or tearing of the small crossing veins and subsequently bleeding in the subdural space. In a consecutive series of 175 hydrocephalic cases treated by of ventriculo-peritoneal shunt, Illingrowth et al. reported 8 cases of subdural hematoma. [12]

Anderson et al. reported that hematoma may be found three days to six weeks post-operatively. [27]

Davidoff et al and Feiring et al. reported subdural hematomas three weeks after surgery. They also reported three cases in a group of 85 hydrocephalic children treated surgically. [10]

Moussa et al. reported six cases that developed subdural hematoma from 30 consecutive children with hydrocephalus treated by shunt implant. Four of them presented win a malfunctioning shunt and diagnosis in all six cases was made by CT scan. The problem is great with low pressure valve. [28]

Hematological disorders: Kwan et al. reported that 14 from 35 patients with thrombocytopenia developed intracranial hematomas post-operatively 22 patients with subdural hematoma associated with long term oral anti-coagulant have been recorded. [16]

Clinical presentation was similar to other subdural hematomas but patients were generally older and the clinical course was more rapid. The results suggested that morbidity and mortality could be reduced by early diagnosis and treatment of any patient on oral anti-coagulant therapy who develops a headache or confusion and should have urgent neurological investigations including CT scan.[29, 30]

Hemodialysis: Hemodialysis may cause CSDH by systemic anticoagulation generally accompanying hemodialysis. Leonard et al. found that in approximately 880 patients on maintenance hemodialysis subdural hematoma had occurred in 14 patients. [31]

Epilepsy: Epilepsy may cause subdural hematoma by trauma induced during the grand mal seizures. Minor cerebral trauma due to a fall at the onset of the seizures might result in a considerable bleeding because of Valsalva mechanism during the tonic phase of the convulsions.[31]

Other causes: Subdural hematomas infrequently are secondary to other pathological lesions such as tumors and infections. [8, 9]

Chronic subdural hematoma may develop after aneurysmal surgery or even any intracranial surgery as reported by Komatsu et al. [32, 33]

Clinical picture of chronic subdural hematoma

In the old age, involutional changes in the brain cause it to shrink rendering the cortical veins more susceptible to injury as they traverse the subdural space. Chronic SDH in adults represents one entity different from that in infants.[34, 35]

A.Chronic subdural hematoma in adults

According to Kaste et al. 83% of the patients had a history of head trauma and the mean interval from trauma to operation was 11 weeks. The mean age of patients was 60 years.[35]

Cameron et al. reviewed patients with chronic subdural hematoma and found that 92% of the patients had an approximately equal incidence of this mode of presentation: hemiparesis, personality or intellectual changes and increased intra cranial tension. Fluctuation in the conscious level or other neurological signs was not a prominent feature in his series and was seen in 2% of patients.[26]

Symptoms and signs of increased intracranial tension

Headache: It is the commonest complaint and it occurs in 42% of cases. Even if confused patients deny it, the family may testify to earlier complaint of headache or to increased consumption of aspirin.[26]

Vomiting: It is less common than headache and occurred in 6 % of cases.[36]

Papilledema: It is not uncommon and was found in 23% of cases.[36]

Signs of hemisphere dysfunction: This is manifested by hemiparesis which is usually contralateral to the site of the hematoma. Sometimes hemispheric signs are ipsilateral to hematoma. They are known as "false localizing signs" such as extensor plantar response, weakness ipsilateral to the lesion. This was the case in 2% of all subdural hematomas in the series of Cameron 1978. It is due to displacement of brain stem so that the contralateral crus come in contact with the adjacent edge of the tentorium. [26]

Hemiparesis is present in 40 % of cases of Cameron et al. series.[26]

Cameron et al. reported a decline in the level of consciousness, this decline usually takes days or even several weeks, at first a lack of alertness happens which later becomes very evident. [26]

Drowsiness, fluctuation of symptoms, ptosis, impairment of upward movement of the eye, sluggishness and dilatation of the pupils may be present. [35]

A characteristic feature is the variability of the consciousness level, the patient unarousable at one time, and few hours later awake.[26]

According to Kaste et al. the prevalence of most commonly encountered symptoms and signs were headache 72%, mental symptoms 48%, Papilledema 41%, vertigo 31%, nausea 28%, reduced consciousness 28%, walking difficulties 24%, hemiparesis 24%, and paraparesis 14%. [35]

Luxon 1979 reported that the combination of raised intracranial pressure, headache, fluctuating drowsiness and mild hemiparesis although highly suggestive of chronic subdural hematoma is not always encountered and epilepsy, aphasia- hemianopia and complete hemiplegia can all occur contrary to "textbook" description. He stressed also that head injury and other etiological factors are commonly absent.[37]

Luxon et al. also reported that the presentation of CSDH may mimic tumors, dementia, cerebrovascular accidents or subarachnoid hemorrhage.[37]

Melamed et al. reported a patient in whom the neurological manifestations were those of recurrent transient episodes of expressive dysphasia preceded by sensory deficit simulating transient ischemic attacks.[38]

James et al. described presentation of the inter-hemispheric subdural hematoma in which either hemiparesis worse in the lower than the upper extremity or lower extremity monoparesis is characteristic of this lesion.[39]

Nobuhiko 1990 described a case of 35 years old woman who presented with parkinsonian syndrome associated with CSDH. The exact etiology responsible for the development of parkinsonian syndrome is not well understood and many mechanisms can be postulated such as mechanical pressure on the basal ganglia nuclei either directly by the hematoma or by torsion or displacement of brain structures and herniation through the tentorial notch. [40]

B. Chronic subdural hematoma or effusion in infants

Infantile chronic subdural hematoma is most common within the first few months of life and its symptoms are those of raising intracranial pressure, drowsiness, vomiting difficulty of feeding and loss of weight.[40]

Convulsions are frequent and may be the first symptom. The infants commonly show enlargement of the head, tense fontanels, separation of the sutures, malnutrition, disturbed consciousness, pyrexia and hemiparesis. Papilledema may occur, and retinal hemorrhages are almost pathognomonic and occur in more than half of cases. [40]

Investigations of chronic subdural hematoma:

Plain radiography

Skull fracture may be present with subdural hematoma. It is less common with chronic subdural hematoma as the trauma is usually minor. In contrast with acute subdural hematoma, fracture is common as the trauma is much greater.[41]

In infancy, enlargement of head is common and there may be widening of the sutures and bulging of the soft tissue of the anterior fontanels. Calcification and even ossification of chronic subdural hematoma may be seen radiologically. Pineal displacement remains the most important finding in subdural hematoma if calcification has rendered it visible. In the majority of cases, the calcified pineal body is displaced backwards and frequently downwards. [40]

Computerized Tomography (C.T.)

There is no doubt that computerized tomography (CT brain) is the most effective and harmless diagnostic tool for detecting subdural hematoma The hematoma is shown as a concave or sickle shaped region between the skull and the brain.[42]

Chronic subdural hematoma is classified into four different degrees of density on CT images: high, low, mixed and isodense.[43]

There is general agreement among neurologists and neurosurgeons that chronic subdural hematoma may be present on CT scan in at least three different types:

Type I: Is characterized by low density of the hematoma content. The hematoma is shown as a concave or sickle shaped dark region between the brain. This type was the most frequent.(44)

Type II: Is characterized by parts with different x-ray attenuation. There are regions with reduced unchanged and increased density. A combination of reduced and increased absorption values prevail. Usually the frontal part of the hematoma has a low density while the posterior part has a high absorption values. Hematoma particles of higher specific weight sediments to the occipital region and sharp border between the two compartments may be seen. In This type, it was assumed that a fresh bleeding has occurred into the pre-existing chronic subdural hematoma which usually causes the clinical deterioration. The frequency of this type was 24%.(45)

Type III: Is characterized by a density which is equivalent to the brain matter (Isodense) and may therefore not be diagnosed directly. It was presumed that the liquefaction of hematoma leads to density values equivalent to brain at certain time after the injury.(43)

Isodense lesion also can occur during the acute stage of subdural bleeding if the patient has low hemoglobin. The frequency of isodense subdural hematoma on CT scanning has ranged from 7 % to 17 %.(42,43)

The presence of isodense lesions if unilateral can be suggested by indirect CT findings, such as ventricular deformity, obliteration and displacement of the cortical sulci, cisterns and sylvian fissures and shift of the midline structures. None of which are specific to this condition. In thin or bilateral isodense chronic subdural hematoma especially, diagnosis by CT is even more difficult. (43)

CT findings of bilateral isodense chronic subdural hematoma

Marcu's et al. reported that the main part of anterior horns is sharply pointed and approaching one another. This ventricular configuration was called (Hare's ear sign). Other signs are midline shift if the size of the hematoma varies, changed density of brain tissue, nonappearance of cerebral sulci especially in elderly patients and eventually visualization of membrane after intravenous injection of contrast material. (46)

Syozi et al., has proven coronal computerized angiotomography to be very useful in the diagnosis of isodense chronic subdural hematoma but it is not nowadays. (43)

Chronic subdural hematoma usually presents in the parietal convexity. The coronal plane is directly perpendicular to the hematoma in this region and grossly parallel to the superficial cerebral veins and cortical branches of the middle cerebral artery Therefore, this permits better visualization of convexity lesion and blood vessels affected by the hematoma than does the standard axial transverse plane.(41)

In the coronal computerized angiotomography; superficial cerebral veins and cortical branches of the middle cerebral artery can be clearly demonstrated as high density lines or dots separated from the inner table of the skull, which have been displaced by the subdural hematoma.(43)

Large isodense subdural hematoma with middle line shift must be differentiated from brain tumor, edema and infarction, and it has been said that angiography is necessary for

this purpose. But computerized angiotomorgraphy provides an easy and non-invasive method to differentiate chronic subdural hematoma from other conditions.[43]

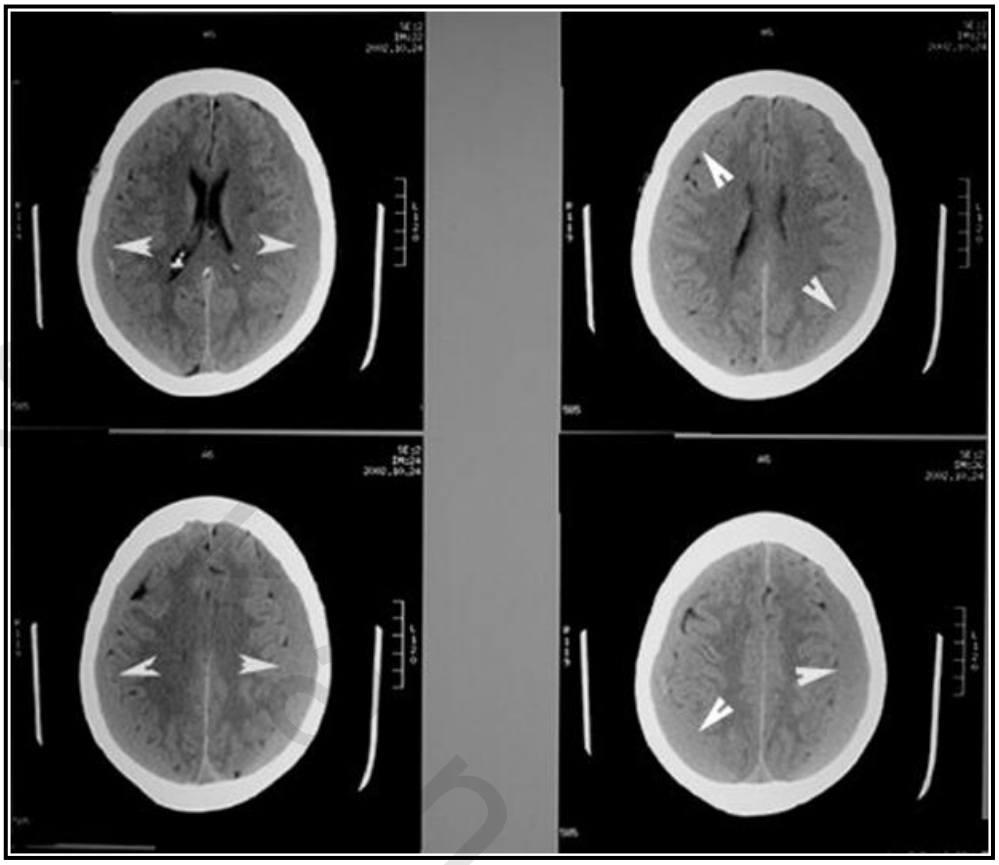

Figure 2: Radiological features of bilateral CSDH in CT brain. These are bilateral and are isodense with the brain because of gradual loss of density of blood. On close exam you see that the sulcal markings do not reach the surface of the skull, due to the intervening subdural collection. Arrowheads point to subdurals separating the brain surface markings from the skull. There is no midline shift.

Angiography

Taveras et al. reported that angiography is a reliable means of localizing a supra-tentorial hematoma. In the average case, the middle cerebral vessels of convexity are displaced away from the inner table of the skull in the frontal angiogram, leaving a fusiform avascular clear space representing the hematoma in the cross section. In some patients with a thin subdural collection, the displacement away from the inner table is not always conspicuous. Usually inspection of the frontal film is the most satisfactory means of detecting the vascular displacement of the hematoma. [47]

Magnetic Resonance Imaging (MRI)

Previously, CT was the most important imaging method in the evaluation of head trauma. Chronic subdural hematomas often show characteristic CT findings that are virtually diagnostic. However, isodense subdural hematomas and bilateral subdural hematomas may be difficult to detect on CT.[42]

Kokichi's study showed that MRI can overcome these difficulties by providing better localization as well as delineation of the extent of the hematoma and its mass effect on adjacent structures. (48)

In general, chronic subdural hematomas are more hyper intense than normal brain tissue on both T1 and T2 weighted MRI images. Chronic subdural hematomas typically have shorter T1 values and longer T2 values than normal brain tissue. Actually, T1 values of chronic SDH were significantly shorter than those of grey matter and longer than those of white matter. The T2 values were significantly longer than those of both. However, 30% of cases were iso-dense or hypo-intense on T1 weighted images and this might represent a re-bleeding phase or a phase immediately after that. (48)

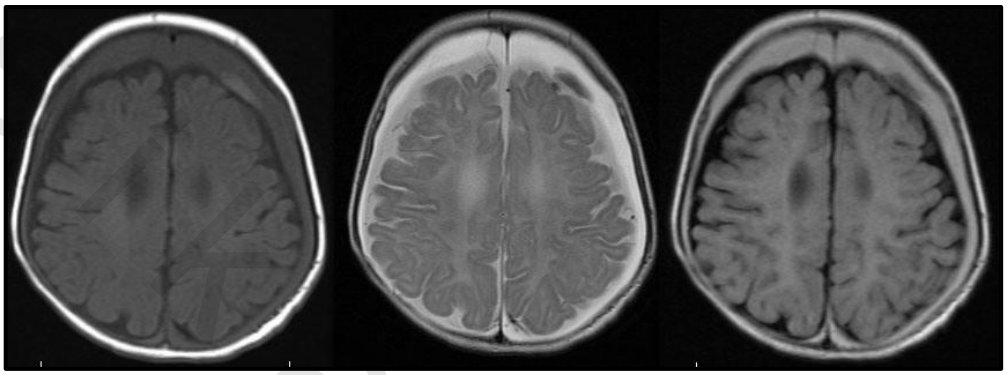

Figure 3: Radiological features of CSDH in MRI brain. Axial T1 (left), T2 (center) and FLAIR (right) weighted images from an MRI scan of the brain performed without intravenous contrast obtained at the same level as the previously performed CT scan of the brain better demonstrates the large, bilateral chronic subdural hematomas with the linear focus of acute subdural hematoma in the frontal aspect of the left chronic subdural hematoma. Note the appearance of the signal intensity of the CSF in the patient's normal subarachnoid space on the T1, T2 and FLAIR weighted images which are the same signal intensity as the CSF in the ventricular system.

Chronic subdural hematoma versus subdural hygroma

Excess fluid in the subdural space (may be clear, blood tinged, or xanthochromic and under variable pressure) is almost always associated with head trauma, especially alcohol-related falls or assaults. Skull fractures were found in 39% of cases. Distinct from CSDH, which is usually associated with underlying cerebral contusion, and usually contains darker clots or brownish fluid ("motor oil" fluid), and may show membranes formation adjacent to inner surface or dura (hygromas lack membranes).(49)

"Simple hygroma" refers to hygroma without significant accompanying conditions. "complex hygroma" refers to hygroma with associated significant subdural hematoma, epidural hematoma, or intracerebral hematoma. On CT, the density of the fluid is similar to that of CSF. (49)

Treatment of CSDH:

- The fact that spontaneous resolution of chronic subdural hematoma has been observed has encouraged some clinicians to treat patients with bed rest alone and dehydration methods. However, at least one clinical trial did not advocate this convective method,

mostly because the subdural hematoma did not decrease in size within a reasonably short period of time. Ultrastructural studies have shown considerable fibrosis of the hematoma capsule after osmotherupy.[23, 25]

- **Seizures prophylaxis:** used by some. It may be safe to discontinue after a week or so if there are no seizure. If late seizure occurs with or without prior use of AEDs, longer-term therapy is required. Some feel that the incidence of side effects from AEDs approximate the incidence of seizures and therefore they do not recommend prophylactic AEDs.[50]
- **Coagulopathies:** (and iatrogenic anticoagulation) should be reversed.[50]
- **Surgical evacuation of hematoma indicated for:**

✓ Symptomatic lesions: including focal deficit, mental changes.[50]

✓ Or subdural hematoma with maximum thickness greater than 1 cm. [50]

Surgical options

There is not uniform agreement on the best method to treat CSDH

- Placing two burr holes, and irrigating through and through with tepid saline until the fluid runs clear.[51]
- Single large burr hole with irrigation and aspiration.[51]
- Single burr hole drainage with placement of a subdural drain, maintained for 24-48 hours (removed when output becomes negligible).[51]
- Twist drill craniostomy: note that small twist drill drainage without subdural drain has higher recurrence rate. [52,53,54]
- Formal craniotomy with excision of subdural membranes (may be necessary in cases which persistently recur after above procedures, possibly due to seepage from the subdural membranes) still a safe and valid technique. No attempt should be made to remove the deep membrane adherent to the surface of brain. It is generally accepted that craniotomy is indicated for these conditions: [55,56]
  1. The subdural collection re-accumulates.
  2. There is solid hematoma.
  3. The brain fails to expand.[55,56]

Techniques that promote continued drainage after the immediate procedure and that may thus reduce residual fluid and prevent recurrence:

- Use of a subdural drain.[57]
- Using a generous burr hole under the temporalis muscle.[57]
- Bed rest restriction with the head of the bed flat (1 pillow is permitted) with mild over hydration for 24-48 hours post-operatively (or if a drain is used, until a 24-48 hours after is removed) may promote expansion of the brain and expulsion of residual subdural fluid. Allowing patients to set up to 30-40 degree immediately post-op was associated with higher radiographic recurrence rate but usually did not require reoperation.[58]

Twist drill craniostomy for chronic subdural hematoma

This method is thought to decompress the brain more slowly and avoid the presumed rapid pressure shifts that occur following other methods which may be associated with complications such as intraparenchymal hemorrhage.[52,54]

It may even be performed at the bed side under local anesthesia. A 0.5 cm incision is made in the scalp in the rostral portion of the hematoma, and then a twist drill hole is placed at a 45 degree angle to the skull, aimed in the direction of the longitudinal axis of the collection. If the drill does not penetrate the dura, this is done with an 18 Ga. Spinal needle. A ventricular catheter is inserted into the subdural space, and is drained to a standard ventriculostomy drainage bag maintained 20 cm below level of the craniostomy site.[52,54]

The patient is kept flat in bed. Serial CTs assess the adequacy of drainage. The catheter is removed when at least 20% of the collection is drained and when the patient shows signs of improvement, which occurs within a range of 1-7 days (mean of 2.1 days).[52,54]

Burr holes for chronic subdural hematomas

To prevent recurrence, the use of small burr holes (without a subdural drain) is not recommended. A generous (> 2.5 cm diameter- it is recommended that one actually measure this) craniotomy should be performed, and bipolar coagulation is used to shrink the edges of the dura and subdural membranes back to the full width of the bony opening (do not try to separate this layers as this may promote bleeding). This allows continued drainage of fluid. A piece of gel foam may be placed over the opening to help prevent fresh blood from oozing into the opening.[51]

Subdural drain

Use of a subdural drain is associated with a decrease in need for repeat surgery from 19-10%. If a subdural drain is used, a closed drainage is recommended. Difficulties may occur with ventriculostomy catheters because the holes are small and are restricted to the tip region, especially with thick oily fluid. The drainage bag is maintained 50-80 cm below the level of the head.[54,57,59,60]

Postoperative, the patient is kept flat. Prophylactic antibiotics may be given until 24-48 hours following removal of the drain. CT scan prior to removal of the drain (or shortly after removal) may be helpful to establish a base line for later comparison in the event of deterioration. There is a case report of administration of urokinase through a subdural drain to treat re-accumulation of clot following evacuation. [54, 57, 59, 60]

Treatment of infantile chronic subdural hematoma

Emptying of chronic subdural hematoma through anterior fontanel tap is a method of treatment only possible in neonates and infants. [40]

Internal drainage of the subdural fluid into body cavity such as peritoneum or circulatory system has improved treatment possibilities in infants and young children. This method has an advantage over external drainage or tapping that no fluid, electrolytes and protein loss occur. [61]

Because of frequent valve malfunction due to high protein content of the hematoma fluid and because of high infection rates associated with subdural circulatory shunt, peritoneal drainage of chronic subdural hematoma by using very low pressure valve and tube free from frequent connections to reduce the chance of obstruction became the therapeutic method of choice in children.[61]

Closure of peritoneal shunt has been found to occur within several weeks, an interval usually adequate for complete resolution of the hematoma. The fear that brain development may be hindered if membranectomy is not performed in

infantile membranous CSDH does not appear to be justified for Collins et al. and and Pucci et al. They demonstrated that by repeated biopsies at varying intervals and reported that capsule thickness and vascularization rapidly decrease in cases with properly functioning peritoneal drainage. The latter finding has been confirmed in other series. [62, 63, 64]

Perret and Graft proposed subdural subgaleal shunting in chronic subdural hematoma or hygroma. They believed that the latter operation was simpler to perform and lead to results as good as those associated with subdural peritoneal shunt procedures.[65]

Complications of chronic subdural hematoma surgery

1. Re-accumulation or recurrence

This complication may result from failure to evacuate the hematoma completely and from continued oozing from the incised outer membrane. The incidence of significant clinical re-accumulation varies considerably depending upon the type of surgical procedure used for removal and the degree of re-expansion of the brain following evacuation of the hematoma. Significant degrees of re-accumulation should be suspected whenever a patient either fail to improve clinically or improves and subsequently shows progressive neurological deterioration following surgery.[26]

Diagnosis of re-accumulation of the subdural hematoma following surgery can be established through computerized tomography CT which may allow differentiation between accumulation of hematoma and failure of cerebral expansion. [26]

Complete resolution of SDH fluid and re-expansion of the compressed brain are both very slow and steady and require at least 10 to 20 days Therefore, a persistent fluid accumulation on a CT scan performed after surgery is no indication for a second surgical procedure unless the patient shows no recovery at all or deteriorates markedly.[59]

A recurrence of CSDH was defined as a subsequent increase in hematoma volume in the ipsilateral subdural space for which reoperation was performed, as described by Torihashi et al. The re-operation was performed to the patients having subsequent radiographically increasing hematoma with neurological deficits due to the hematoma. [66]

Markwalder et al., suggested that additional surgical procedures such as repeated tapping of chronic SDH fluid and craniotomy and membranectomy should not be evaluated earlier than 20 days after the initial operative intervention. [59]

Previous studies have reported several factors that are associated with the recurrence of CSDH, including age, bleeding tendency, hematoma density, width of the hematoma, postoperative midline displacement, postoperative subdural air collection, bilateral CSDH, postoperative position, pre-operative Glasgow Coma Scale score, and post-operative Glasgow Outcome Scale score.[58,67-75]

Considering the increasing number of aged patients who use antiplatelet and anticoagulant medications, attention should be focused on the possible risks of these treatments. Theoretically, the use of antiplatelet and anticoagulants has a positive influence on the recurrence of CSDH. In the previous studies, there was no association between medication with antiplatelet or anticoagulants and recurrence of CSDH. They concluded that the reason for this finding may be that the patients were not adequately anti-coagulated, especially in the non-recurrence group. [76]

Although unilateral convexity CSDH occurs in the majority of patients, bilateral lesions are not uncommon in neurosurgical practices. The overall incidence of bilateral CSDH has been reported to vary from 16% to 20%. Most clinicians consider bilateral CSDH equivalent to unilateral CSDH as there is no difference in the presentations or treatment strategies. However, rapid and progressive aggravation of bilateral CSDH has been documented and the authors recommend operation as early as possible with simultaneous decompression of bilateral hematoma pressure. Furthermore bilateral CSDH is identified as a risk factor for recurrence by some researchers. All these reports suggest that bilateral CSDH has a different clinical significance, and hence, should be distinguished from unilateral cases in order to establish an appropriate management. [77,78]

2. Infection

Post-operative septic complications include superficial cellulitis, epidural abscess, osteomyelitis, cortical vein thrombosis, meningitis, brain abscess or a combination of these may occur. Clinically the patient commonly exhibits temperature elevation post-operatively and inspection of the wound reveals varying degrees of cellulitis. Patients with epidural or subdural empyema or cortical vein thrombosis may exhibit focal neurological signs, seizures and impairment in the level of consciousness in addition to the febrile response.[79]

Computerized tomography (CT brain) is mandatory to diagnose epidural or subdural empyema and intracerebral abscess. Infection is treated by massive doses of third generation cephalosporin and vancomycin till culture and sensitivity is determined.[79]

3. Intracerebral hemorrhage

Domenico et al. reported two cases of intracerebral hemorrhage occurring after evacuation of bilateral chronic subdural hematoma. Possible pathogenic mechanisms included hemorrhage into previously undetected areas of contusion, damage to cerebral vasculature secondary to rapid post-operative parenchymal shift and sudden increase in the cerebral blood flow combined with focal disruption of auto regulation. This latter mechanism seemed most likely to be responsible for the hematoma formation. [80]

Slow decompression of chronic subdural hematoma with controlled re-expansion by means of lumbar saline instillation with gradual recovery from anesthesia and careful control of blood pressure must be performed to a avoid such complication. Immediate use of CT scanning if there is delayed recovery from anesthesia or if a new neurological deficit develops is a key to early diagnosis of this rare complication. [80]

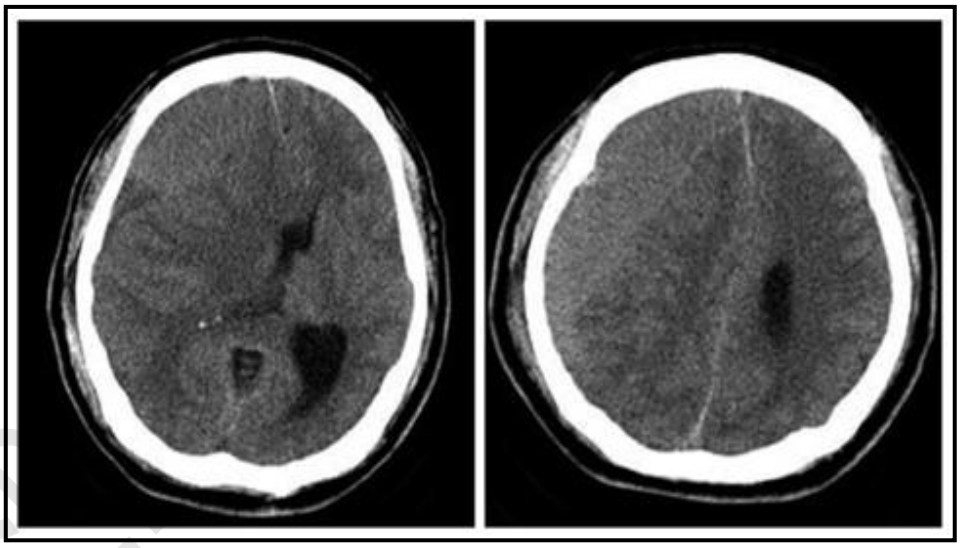

Figure 4: Pre-operative CT scan reveals isodense right chronic subdural hematoma with midline shifting.

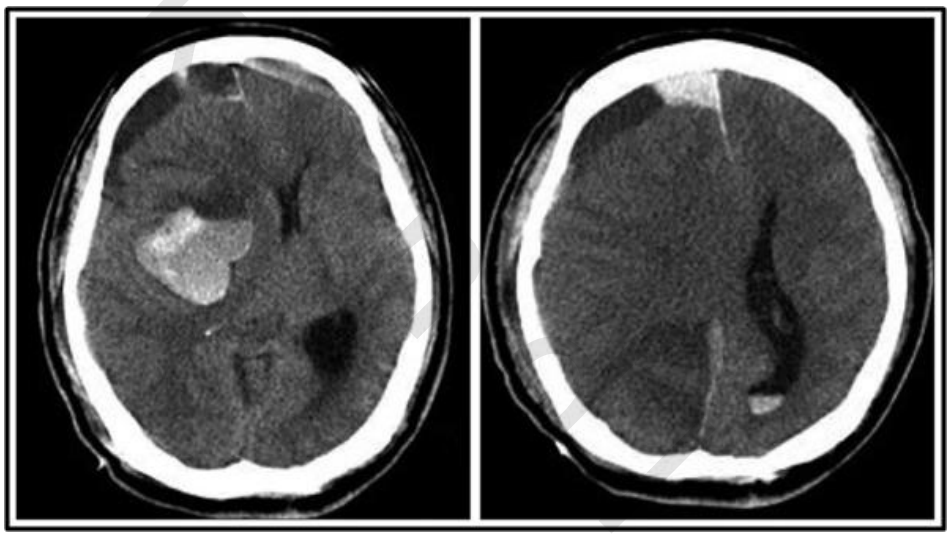

Figure 5: Postoperative computed tomography images showing intracerebral hemorrhage in right basal ganglia and subdural hematoma in right frontoparietal area after bur hole evacuation of CSDH.

2. Tension pneumocephalus

Sharme et al. reported five cases of tension pneumocephalus following evacuation of chronic subdural hematoma. This occurred in 8% of all cases of chronic subdural hematoma treated. The chronically compressed brain contributes to the ingress of this intracranial air, in the early post-operative period resulting in a rise in intracranial tension leading to neurological deterioration. (81)

Subdural air with increased tension separates and compresses the frontal lobes. The collapsed frontal lobes with widened inter-hemispheric space between the tips of the frontal lobes resemble the silhouette of Mt. Fuji. This CT finding was termed the "Mt.Fuji sign". It is Present in 80 % of cases of tension pneumocephalus. (81)

Small air bubbles "air bubble sign" tended to spread diffusely into a variety of cisterns in tension pneumocephalus while those associated with asymptomatic pneumocephalus tended to gather in the cortical subarachnoid or subdural space. This sign is present also in 80% of cases of tension pneurnocephalus. It is postulated that these air bubbles enter the subarachnoid space through a tear in the arachnoid membrane caused by increased tension of air in the subdural space. [81]

The CT appearance of the Mt.Fuji sign and the smaller air bubbles in the subarachnoid cisterns are particularly helpful in making the diagnosis of subdural tension pneurnocephalus following surgery for CSDH. Twist-drill craniotomy and aspiration using a brain cannula with a three-way connecter has produced excellent result. [81-83]

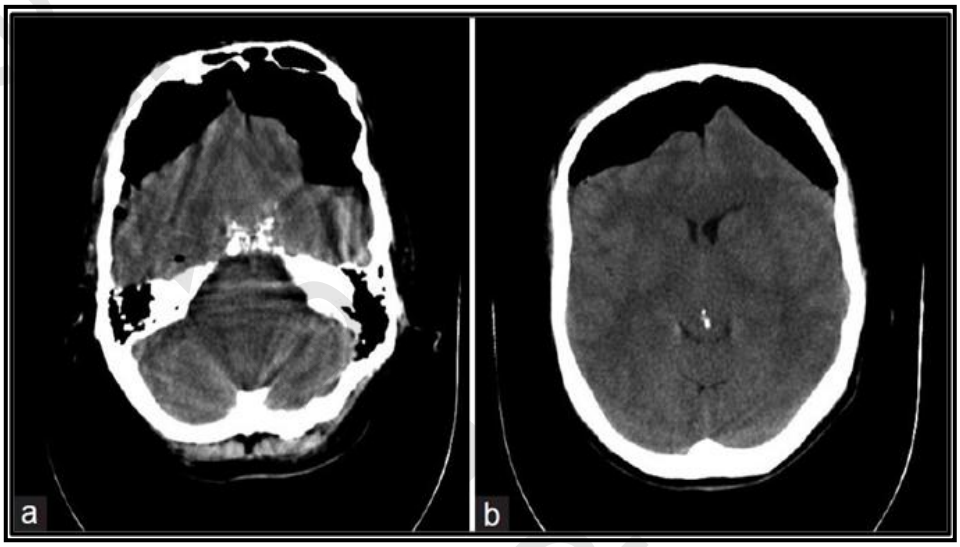

Figure 6: Mount Fuji sign. (a) collapsed frontal lobes with widened inter- hemispheric space between the tips of the frontal lobes. (b) smaller air bubbles in the subarachnoid cisterns.

3. Cerebral edema

The compressive effect of subdural hematoma upon the cerebral hemisphere may cause ipsilateral swelling of the white matter. This may be due to interference with venous drainage caused by the pressure of the extra cerebral mass or it may be a complication of surgery especially if post-operative re-accumulation has occurred. CT scan reveals edema as an area of decreased density. [22] Cerebral edema is treated by dexamethasone 10 mg every / 6 hours intravenously, Furosemide 40 mg single intravenous dose and mannitol 20% solution intravenously in a dose of 1-1.5 gm / Kg body weight over a 1-2 hours period. [22, 84]

4. Seizures

Patients with epidural or subdural empyema or cortical vein thrombosis may exhibit focal neurological signs, seizures and impairment in the level of consciousness in addition to the febrile response. Seizures are not uncommon in chronic subdural hematoma.[70]

5. Hydrocephalus

Subdural hematoma may induce hydrocephalus through:

a. Obstruction in the subarachnoid pathways by mass effect of the hematoma.[86]
b. Trauma which caused subdural hematoma may cause subarachnoid hemorrhage leading to hydrocephalus.[87]
c. Surgical treatment of subdural hematoma may cause hydrocephalus through iatrogenic subarachnoid hemorrhage or through post-operative dural adhesion.[87]

Clinically, hydrocephalus should be considered in the differential diagnosis of a patient who fails to improve or deteriorates after surgery. CT brain is diagnostic. Treatment is by ventriculo-peritoneal shunt. [88]

Complications of shunting procedures

When used in treatment of chronic subdural hematoma in infant and children shunt obstruction may occur due to high protein content of the subdural fluid. This is avoided by using very low pressure valve and tube free from frequent connections to reduce the chance of obstruction. Other complications like infection and migration of shunt tubing may occur. [61]

Outcome after surgery

There is clinical improvement when the subdural pressure is reduced to close to zero, which usually occurs after 20% if the collection is removed. Patients who have high subdural fluid pressure tend to have more rapid brain expansion and clinical improvement than patients with low pressures. Residual subdural fluid collections after treatment are common, but clinical improvement does not require complete resolution of the fluid collections on CT. CT scans showed persistent fluid in 78% of cases on post-operative day 10, and in 15% after 40 days and may take up to 6 months for complete resolution.[59]

Recommendation: do not treat persistent fluid collections evident on CT (especially before 20 days post-operatively) unless it increases in size on CT scans or if the patient shows no recovery or deteriorates. [59]