Chronic Subdural Hematoma: A Review

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Abstract

Chronic subdural haematoma (CSDH) is an encapsulated collection of old blood between the dura mater and arachnoid caused by tear of bridging veins. Chronic subdural haematoma is considered when the blood is more than three weeks old. It is fairly common disease, especially in the elderly with incidence ranging 1.72 to 7. 35 per 100,000 population with male predominance[1,2,3]. Elderly population is associated co- morbidities that can impact on immediate postoperative outcome and overall survival. Although history of trivial trauma is present in majority of cases but some cases may be secondary to coagulation defect, intracranial hypotension, and use of anticoagulant and antiplatelet agents. It commonly presents with headaches, confusion, drowsiness, vomiting, and seizures. On examination, patient have various neurological deficits including a low Glasgow coma scale, hemiparesis/hemiplegia, ocular palsy, and other deficits are seen. Diagnosis is usually confirmed by NCCT scan head however MRI are indicated for better visualisation of, multiloculated, intrahaematoma membranes.

Management of this condition is mostly surgery but some patients have also been managed conservative with steroids. Steroids have been used in patient who have minor headache and also used in patients who are unfit for surgery [4].

Key words: Chronic Subdural Hematoma, management, Review

Chronic subdural hematoma was first described by Virchow in 1857 as "pachy meningitis 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 recognised as an important factor in the development of CSDH. [5]

CSDHs are mainly due to trivial head trauma which may go unnoticed. History of direct head injury is absent in 30-50% of the cases. So, indirect trauma is important. About half of the patients had history of fall without hitting of head on the ground.[6,7] Other risk factors are advancing age, intracranial hypotension, anticoagulant medication, antiplatelet medication, chronic alcoholism , epilepsy etc.[5]

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Generalised cerebral atrophy and increased venous fragility associated with aging are the major predisposing factors. With aging, mass of brain decreases which leads to an increase in space between brain and skull from 6% to 11% of the total intracranial space. This causes stretching of the bridging vein and greater movement of the brain within the cranium, making these veins vulnerable to trauma. [8, 9]

Intracranial hypotension due to cerebrospinal fluid (CSF) leakage, could lead to CSDH formation. Spontaneous intracranial hypotension could be the cause of CSDH, especially in young to middle-aged patients, without preceding trauma or haematological disorders. Intracranial SDH can be a complication of puncture of the dura mater. It should be suspected, especially in post Lumbar Puncture (LP) headaches of more than 1 week. Neuroimaging is necessary after 1 week of LP if the patient continues to have a headache. CSDH should be considered in postpartum patients, who have received epidural anaesthesia, and present with mild to severe, persistent, and non-postural headache.[10] CSDH could occur after microdiscectomy complicated by delayed CSF leak. CSDH can develop due to intracranial hypotension secondary to sudden decompression of intracranial pathologies, such as suprasellar arachnoid cyst fenestration and endoscopic third ventriculostomy. [11]

CSDH can also develop in the presence of potential haemorrhagic diathesis due to the deficiency of clotting factors. CSDH could also develop in patients receiving antiplatelet and anticoagulation therapy. [12] CSDH has been seen in intracranial arachnoid cyst. Incidence of this complication is about 6.5% in the arachnoid cysts. [13]

The initial trauma to bridging veins results in haemorrhage into subdural space. A day after the haemorrhage, the outer surface of haematoma is covered by a thin layer of fibrin and fibroblasts. Migration and proliferation of fibroblasts leads to formation of a membrane over the clot by the fourth day. The outer membrane progressively enlarges and fibroblasts invade the haematoma and form a thin membrane during the next two weeks. [14.]

The developing hematoma capsule shows gradual changes in cellular and vascular organization with progression in hematoma age. Initial changes include angiogenic and aseptic inflammatory reactions. It is followed by fibroblast proliferation and development of collagen fibrils. Young hematomas (15-21 days after trauma) show numerous capillaries, suggesting formation of new blood vessels. Older hematomas (40 days after trauma) usually show numerous capillaries and thin-walled sinusoids accompanied by patent, larger diameter blood vessels. Blood vessels are frequently occluded by clots in fibrotic outer membrane of 60 or more day's old hematoma. [15]

Two major theories have been proposed to explain growth of a CSDH—namely, the osmotic theory and the theory of recurrent bleeding from the haematoma capsule. First, Osmotic theory was based on the hypothesis that liquefaction of haematoma increases the protein content and oncotic pressure in the encapsulated fluid. This attracts fluid from neighbouring vessels into the cavity due to osmotic pressure gradient across the semipermeable membrane haematoma capsule [16], however this theory was disproved by Weir, who demonstrated that osmolality of the haematoma fluid was identical to that of blood and cerebrospinal fluid. [17]

Second, is recurrent bleeding from the haematoma capsule which is a proved and more widely accepted theory. The haematoma capsule has been shown to have abnormal and dilated blood vessels are considered as the source of haemorrhage. [18]

The intracranial pressure is usually normal or only slightly increased. Atrophied brain and lack of tamponade effect contributes to the gradual expansion of CSDH. Nature of the subdural collection may vary between watery, altered blood and fresh blood clots, depending on the age of the CSDH and the frequency of recurrent haemorrhages

CSDH consists of an outer membrane, hematoma cavity, and an inner membrane. Hematoma fluid is usually liquid that does not clot. Usually hematomas are liquefied, but mixed lesions with solid components are also seen. The higher concentrations of fibrinogen and D-dimer are seen in the layered and mixed types of CSDH. The fibrinolytic factors appear to be associated with evolution in CSDHs with heterogeneous density.[19]

CSDHs cause decrease in the blood flow in the underlying brain. The drainage of the hematoma results in the improvement of cerebral blood flow and clinical recovery.[20] CSDH could be associated with reversible distortion of the underlying nerve fibers and vasogenic edema.[21]

The presentation of chronic SDH varies from no symptoms to altered mental status, headache, seizure, confusion, drowsiness, and ataxia. Onset of symptoms may be delayed by weeks or even months. Patients could have difficulty in speech, swallowing, walking, isolated third nerve palsy weakness and numbness in arm, face, and leg. CSDH has an important differential diagnosis in reversible dementia.[22] CSDH can present in many ways that is why it has been described as "the great neurological imitator". [23]

CSDH generally occurs in elderly but rarely can it be present in infants. Bilateral CSDH infants can be due to non accidental trauma.[24] CSDH is most common on the frontal and occipital convexity of the left side than right side of hemisphere. [25]

The most common presentation in elderly (50%–70%) is altered mental state. [19, 26] It may manifest as varying degrees of confusion, drowsiness, or coma. Also the diagnosis may be very easy to miss in patients with psychiatric or neurological illnesses in whom any change in behaviour or functional state is usually attributed to their pre-existing illness. [27]

Hemiparesis was found in 58% of cases in Luxon *et al* series [28], weakness of limbs is usually mild but drowsiness is out of proportion to the degree of neurological deficit. Mostly, the deficit is contralateral but there are reports of ipsilateral symptoms. Direct pressure on the cerebral hemisphere is thought to be the underlying mechanism. Fluctuating neurological symptoms are uncommon and usually the symptoms start insidiously and progress gradually. [28] Gerstmann's syndrome (right-left disorientation, finger agnosia, agraphia, and acalculia) and progressive quadriparesis due to CSDH has been reported in the literature.[29,30] These patients made a good recovery after the evacuation of haematoma. The incidence of headache varies in different studies, ranging from 14% to 50 %.[31, 32].

Epilepsy is a rare presentation and is reported in up to 6% of cases as an initial symptom.[28] 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 ischemic attack.[33] The incidence of CSDH presenting with Transient neurologic deficits (TND) varies from 1% to 12 %.[34] The most common symptom is disturbance in language and most frequent sign is hemiplegia or hemisensory deficit. The mechanisms proposed to explain

TND in CSDH are intermittent mechanical pressure on the neighbouring vessels, transient increase in parenchymal swelling causing vascular displacement and ischemia, small repeated haemorrhages in the subdural space, seizure activity with postictal deficits, and spreading cortical depression. [35, 36]

Patients presenting with vertigo, nystagmus, and isolated oculomotor palsy due to CSDH have been reported.[37, 38] Increased intracranial pressure causing uncal herniation and stretching of cranial nerves was thought to be the mechanism involved.

CSDH causing Parkinsonian symptoms is a well recognised phenomenon.[39] The mechanisms suggested are pressure on the basal ganglia, compression of midbrain, and circulatory disturbances in the basal ganglia caused by displacement and compression of anterior choroidal artery.[40]

CSDH is preferred to be diagnosed by CT scan.[41] Hematomas are usually hypodense (CT value<30 Housenfield units), but sometimes isodense (30-60 Housenfield units) or mixed density lesions are also observed. The transition from hyperdense appearance of an acute haematoma to that of CSDH is seen typically after 3 weeks. They are usually concavo-convex. The CSDHs could be globular. Huge hemispheric CSDH, occupying whole cerebral hemisphere and compressing falx with almost complete obliteration of ipsilateral lateral ventricle, has been reported.[42]

Findings on CT scan, shows subdural collection with effacement of the sulci, compression of the ipsilateral ventricle and midline shift, deformity of the normal ventricular anatomy, and obliteration of the basal cisterns which could aid in the diagnosis. Bilateral haematomas may show slit-like elongated ventricle (so called "squeezed ventricle" or "rabbit's ears").[43,44,45] It has been clearly shown, that MRI is better than computed tomography in identifying small and transversely oriented collections where the computed tomogram has failed to identify a collection in as much as 80% of cases.[46]

Bilateral isodense CSDH may cause considerable difficulty in diagnosis by CT scan. MRI could help in making the diagnosis of such lesions.[47] MRI is more sensitive than CT in determining the size and internal structures of CSDH, such as multiple loculations and intra hematoma membranes. Fresh bleeding, haemolysis, and haemoglobin changes can also be observed by MRI. The diffusion tensor imaging can examine anisotropic changes of the pyramidal tracts displaced by CSDH. These anisotropic changes are considered to be caused by a reversible distortion of neuron and vasogenic edema by the hematoma. These changes in the affected pyramidal tract correlate to motor weakness in CSDH. [22] Contrast-enhanced MRI could detect membranes, thick and extensive membranes. The calcified CSDHs are rare and could mimic calvarial mass. Contrast images required to find out any associated primary or metastatic dural disease. [48]

Magnetic resonance imaging (MRI) scan is also required in patients with isodense haematoma without midline shift and in identifying small collections at the vertex, base of the skull and in the posterior fossa.[49,50,51]

Even though MRI has advantages, CT scan remains the investigation of choice in the acute setting because of shorter examination time, which is important in acutely ill patients, reliability in identifying other parenchymal lesions, no magnetic interference (especially in patients on life support machines) and the ready availability.

SDH could be seen in association with primary dural diseases or metastasis. Such lesions could be missed and could be the cause of recurrence in CSDH. A contrast-enhanced brain CT scan is recommended to diagnose dural metastases.

Surgery is the treatment of choice in most of the CSDHs. Some of the small CSDHs after antiplatelet drug use can be managed conservatively by stopping the antiplatelet drugs and follow-up with repeat CT scans. When a patient is on antiplatelet drugs, surgery is delayed, if the patient's condition permits, allowing for the wearing off effect of the drugs. This delay could minimize the risk of haemorrhage. If urgent burr holes are indicated, measures to improve platelet function should be undertaken.[52] There is general agreement that significant coagulopathy should be reversed expeditiously in CSDH before surgery is undertaken. [(53)]

The efficacy of prophylactic anti-convulsive medication has been debated and its use is not consensual.[54,55,56] Anti-epileptic drug (AED) prophylaxis could be given in higher-risk patients. The mortality is high in postoperative seizure cases, therefore seizure prophylaxis should be given in high-risk patients low Glasgow Coma Scale (GCS< 9), preoperative cognitive impairment and history of alcohol abuse.[57] Lower mean GCS on admission is predictive of seizures in CSDH.[58] The preoperative AED prophylaxis is likely to reduce the incidence of postoperative seizures in CSDH.[54] Various imaging finding such as significant underlying brain injury, mixed density of blood and presence of membranes and location of blood near epileptogenic focus like temporal lobe and tentorium are also indication of prophylactic AED use, levitracetam and valproate are commonly used as prophylactic AED in high risk patient.

Some surgeons recommend use of corticosteroids. Steroids are anti-inflammatory so, they decrease the inflammatory reaction which is the known cause for maintenance of vascular membrane. Spontaneous resolution occurs when the vascular membrane around the haematoma matures; bringing an end to recurrent small bleeding that increases or maintains the size of

haematoma. Corticosteroids may facilitate this process by down-regulating the inflammatory reaction that is thought to maintain the vascularised haematoma membrane. [59]

Burr hole craniostomy (BHC) is the most efficient choice for surgical drainage of uncomplicated CSDH. Burr holes are made at a site of maximum haematoma thickness. Dura is opened to reveal the greenish-yellow membrane associated with haematoma. The membranes are incised and very often subdural fluid comes out under considerable pressure. The cavity is then irrigated with normal saline till clear saline comes out of the cavity and the patient is tilted a little head down, so that the brain can expand in subdural space. But in some patients a catheter is left for 24-72 hours to allow any residual haemorrhage to be removed using gravity drainage and gentle suction. Burr-hole craniostomy is associated with a low recurrence rate and lesser complications.[60,61,62,63,64] Treatment of CSDH through a burr-hole irrigation and closed-system drainage under local anaesthesia is simple, safe, and effective.[65] Surgeons differ in their choice regarding usage of one or two burr holes. One burr hole craniostomy with closed drainage could be sufficient to evacuate CSDH with lower or similar recurrence rate, as compared to two burr hole groups. [66, 67, 68] However, Taussky *et al* [69] found that treatment of CSDH with one burr hole was associated with a higher postoperative recurrence rate, longer hospitalization length, and higher wound infection rate, as compared to treatment with two burr holes.

The Twist drill craniostomy (TDC) drainage is indicated in high-risk surgical candidates in non-septated CSDH.[53] TDC can be performed at bedside by making an skull opening of < 5mm and is effective in treating CSDHs.[70] The ventricular catheter is usually placed into subdural space and then connected to reservoir placed 20 cm below head and patient is instructed to lie flat post operatively for 24-48 hrs.

Although TDC for evacuation of a CSDH is a rapid and minimally invasive procedure, it carries the risk due to its blind nature. Some of these patients can have inadequate drainage, brain penetration, acute epidural hematoma, and catheter folding. High recurrence rate was observed in TDC. [71] These complications can be prevented by doing some modifications in the technique. Irrigation catheter should be more to avoid inadequate drainage. The increased angle of skull penetration can reduce brain penetration chances. The risk of extradural hematoma due to separation of dura mater from skull can be reduced by pointed drill and entry of subdural space by sudden push. Insertion of catheter with Kirschner wire can prevent catheter folding. [72] Posterior positioning of the drill at parietal tuberosity can reduce brain penetration chances. Skull entry at the most curved position on the skull could also prevent brain penetration. This allows direct entry of the catheter in the hematoma cavity only, rather than the brain. One of the major concerns in TDC is bleeding from dura mater, especially from the middle meningeal artery. Dural penetration at the middle meningeal artery should be avoided by suitable entry point 1 cm anterior to the coronal suture at the level of the superior temporal line. [73] The advantage of twist drill is that it decompresses the subdural space slowly,thus avoiding rapid pressure release that can lead to cortical hyperaemia or even intracerebral haemorrhage.

Craniotomy is indicated in neurological deficit related to the clot, failed less invasive BHC management. Craniotomy is also indicated in CSDH with significant membranes,[53] multiloculated,[74] organized[75,76] and calcified or ossified CSDH.[77] Although an ossified membrane could be excised[78] or drilled by high-speed air drilling[77] the optimal surgical procedure for such CSDH has not been established because it is hard to obtain brain re-expansion after surgery. The residual rigid inner and outer membranes facilitate dead space and hematoma recurrence. Multiple tenting procedures could help in obliteration of the dead space in a large CSDH to prevent recurrence [79]

Coronal view of CT head is useful for planning craniotomy especially in case of mini craniotomy. Identification of clot in relation to external auditory meatus helps to define anterior and posterior boundary. A linear incision in the coronal plane centred over the largest extent of haematoma. [80]

After exposing the calvarium 1 or 2 burr holes were made along the border of craniotomy. Penfield dissector No 3 is used to strip dura from the inner surface of bone prior to using craniotome. Care is taken not to extend the craniotomy up to superior sagittal sinus then dura is opened in cruciate pattern then gentle irrigation of outer and inner membrane is done and clots are removed. Aggressive traction and resection are not done. All visible membranes (outer layer) are coagulated with bipolar cautery. After haemostasis bone flap was replaced with titanium screws and plates. [80]

Considerable body of evidence supporting the use of external drainage after evacuation of primary CSDH exists in most of the reported series.[81,82,83,84,85,86]Santari us *et al*.,[84]Ramachandran *et al*.,[87]Wakai *et al*.,[88]Tsutsumi *et al*.,[89]Gurelik *et al*.[90]and Sarnvivad *et al*[91] reported 9.3%, 4%, 5%, 3.1%, 10.5%, and 16% recurrence rates, respectively, in the with drain group, as compared to 24%, 30%, 33%, 17%, 19%, and 26%, respectively, in without drain group (Figure-1) Continuous drainage therapy for CSDH is superior to the one-time drainage method, with shorter post-op hospitalization and low recurrence.[82,83]

Santarius *et al*[84] had reported lower 6 months mortality of 8.6% in the drain group, as compared to 18.1% when drain was not used. On the other hand, there was no significant difference in the postoperative recurrence and complication rates in with drain and without drain groups

in some studies [92, 93] but advocates of no drain group argue that placing a drain could lead to complications such as brain injury, haemorrhage from membranes, and infection without reducing recurrence. Subdural empyemas have been reported after subdural drain. [93, 94, 95, 96, 97] Postoperative infection in the subgaleal space has also been reported after subdural drain [98]

Irrigation and drainage are aimed to reduce recurrence in CSDH.[89] The burr-hole drainage with irrigation is associated with good outcomes and lower recurrence rate, as compared to drainage alone.[99] Irrigation with large amount of fluid during surgery may reduce the recurrence rate in CSDH.[100]

Endoscopic treatment is indicated in removal of solid clots under direct vision in organized and multiloculated CSDH.[101,102] Endoscopic treatment could make the procedure safer with enhanced intraoperative visualization. It may allow the identification and removal of membranes.

Surgical treatment of CSDH could be associated with recurrence, infection, new intracranial hematoma, seizure, cerebral edema, tension pneumocephalus, and failure of the brain to expand due to cranio-cerebral disproportion.[103] Although there is a potential risk of pneumocephalus after all surgical techniques in the treatment of CSDH, it is seen in 11% of cases.[104] But sometimes tension pneumocephalus could occur after evacuation of a CSDH.[105,106] The amount of subdural air is correlated negatively with resolution of a CSDH.[107] It impedes the adhesion between the inner and outer membranes, prolonging the widening of the subdural space, thus promoting postoperative re-accumulation. The subgaleal or subdural drain could minimize recurrences by preventing the collection of subdural collection and air. [98] Intraoperative saline flushing, positioning of burr hole at the highest point on the skull, and avoiding nitrous oxide anaesthesia could help in preventing pneumocephalus. [108] The risk of pneumocephalus can be reduced by the skin closure immediately after cessation of spontaneous blood efflux. Valsalva manoeuvre and use of gravity in 30° Trendelenburg position, rather than suction, are helpful in avoiding pneumocephalus.

Tension pneumocephalus as a post operative complication is usually rare with rates varying between 1-2%. [109,110] Development of tension pneumocephalus after burr hole evacuation of CSDH is a rare postoperative complication. The chronically compressed brain is thought to contribute to the entry of intracranial air. The slow re-expansion of the brain and trapped subdural air leads to increase in intracranial pressure leading to neurological deterioration. Craniostomy and aspiration is the usual treatment

Intracerebral and intraventricular hemorrhage can develop after removal of CSDHs. Possible mechanisms include a sudden increase in cerebral blood flow within fragile cerebral vessels, defective vascular autoregulation, and damage to the cerebrovascular tree. Asymmetrical and rapid decompression could lead to vascular disruption and/or sudden increase in cerebral blood flow[111] Combined epidural and intracerebral haemorrhage can occur immediately after evacuation of bilateral CSDH. [112] Hematomas could develop on the opposite side [113] or at any remote [114] place. Slow and simultaneous bilateral decompression of massive CSDHs is recommended to prevent secondary intracranial hematoma. [111]

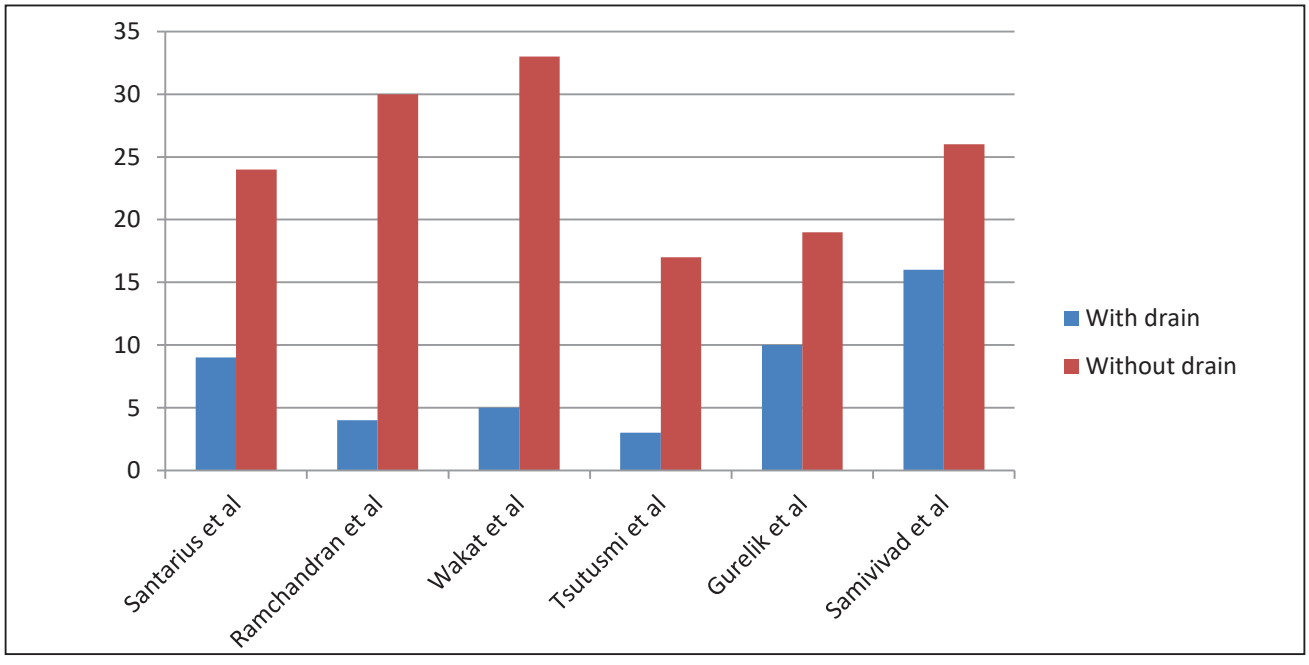

Figure 1: Bar diagram showing recurrence rate with and without drain

Table 1: Summary of recommendations based on the available literature as per classes of evidence has been tabulated in table- 2 below

Subject	Recommendation	Strength of recommendation	Studies
Surgical method	• BHC – "first choice" in most patients. • TDC – in surgical high-risk patients. • Craniotomy – "last choice", with , significant membranes, multiple recurrences, calcification.	B and C	Weigel *et al.*[86] Santarius *et al*[130] Lega *et al.* [61] Ducruet *et al*[53]
Number of burr holes	One or two (both equivalent)	C	Taussky *et al.*[69] Han *et al*[66] Kansal *et al.*[68] Belkhair *et al*[146]
Irrigation	Safe Might lead to less recurrence especially in TDC.	B and C	Ram *et al*[147] Hennig *et al*[148] Aoki *et al*[149]
Drainage insertion	Yes	A	Santarius *et al.*[84]
Drainage location	Subperiosteal (Recurrence rate same as subdural, fewer complications).	B	Kaliaperumal *et al*[150] Bellut et al[151] Zumofen *et al*[152] Gazzeri *et al*[98]
Postoperative mobilization	Studies in favor of both early and late mobilization.	A	Abouzari *et al*[153] Kurabe *et al*[154]
Reversal of anticoagulation	Rapid reversal using PCC and Vitamin K.	C	Woo *et al*[155]
Resumption of anticoagulation	After 72 hours in high-risk patients In atrial fibrillation compare HAS-BLED score and CHA2DS2-VASc score.	B	Kawamata *et al*[156] Yeon *et al*[157] Chari *et al.*[158]
Reversal of antiplatelets	Discontinuation 7 days prior to surgery. In urgent cases platelet transfusion or desmopressin.	C	Mascarenhas *et al*[52] Ranucci *et al*[159]
Resumption of antiplatelets	No recommendations; case-by-case management.	C	Forster *et al*[160] Rust *et al.*[161] Torihashi *et al*[126]
Antiepileptic drugs	None, except in high-risk patients (e.g. alcoholics) or presenting with seizure.	C	Ducruet *et al*[53] Hirakawa *et al*[162] Grobelny *et al*[54]

The recurrence of CSDH is defined as an increase in thickness of hematoma and a change in hematoma density on follow-up CT scans within a month post-operatively or re-appearance of symptoms such as headache, change of consciousness or limb weakness.[115] Incidence of recurrence is between 5 and 33% after surgery[116] Recurrence is significantly more common in the thick hematomas. Reaccumulation of haematoma is the common postoperative problem. Residual fluid can be detected on computed tomography in as many as 80% of the patients, a majority of them asymptomatic and clinically insignificant. Recurrence usually occurs between four days to four weeks with an average interval of 12 days. Clinical deterioration with radiological evidence brings attention to this condition. It is more common in the elderly with inadequate expansion of the brain following the evacuation of the haematoma is thought to play a part. [117] Many factors seem to be responsible for the recurrence. Postoperative midline shift of 5 mm or more, diabetes mellitus, preoperative seizure, and preoperative thickness of CSDH 20 mm or more are the predictors of recurrence in CSDH. The rate of recurrence is lower in the homogeneous and the trabecular type, as compared to the laminar or multilayered type hematoma.[116]Rate of recurrence is higher in high- and mixed-density lesions in CT brain .[117]

Primary and metastatic diseases of the dura mater can give rise to malignant subdural effusion (SDE) and recurrence. [118] Intracranial hypotension could give rise to recurrence in CSDH.[119] MRI of the spine and/or radionuclide cisternography is useful in the evaluation of intracranial hypotension and to avoid recurrences.[120] Thick

subdural membranes visualized during surgery and brain remaining in the depth at the end of surgery are associated with increased recurrence. Lower GCS score and presence of intracranial air 7 days after surgery are related with increased recurrence in CSDH. [121,122]

High levels of tPA in the subdural fluid and outer membrane have a relatively high probability of recurrence[123], Angiogenic growth factors and inflammatory cytokines are associated with increased recurrence. Higher concentration of IL-6 in the subdural fluid or enhanced expression of vascular endothelial growth factor (VEGF) and Basic fibroblast growth factor (BFGF) in the outer membrane is more likely to be associated with recurrence. Local inflammation seems to be responsible for continuous bleeding by capillary exudation in the earlier phase, whereas angiogenesis appears to help maturation of the outer membrane by sprouting vascular networks in the later phase. [124,125]

Antiplatelet or anticoagulant drugs might facilitate the recurrence of CSDH.[126] Irrigation with small amount of fluid during surgery may increase the recurrence rate of CSDH. [127] Poor brain re-expansion rate could be responsible for a higher recurrence rate in CSDH, especially in bilateral hematoma.[128] Continuous drainage, proper postoperative hydration, prevention of pneumocephalus, and use of gravity can help the brain to re-expand. Excision of the constricting thick membrane in selected patients can help the brain to re-expand. The recurrence could be due to entrapment of CSF in the hematoma cavity through some areas of the inner membrane. Intravenous fluid administration of at least 2000 ml/ daily for 3 days postoperatively has been found to be associated with reduced recurrence in CSDH. [129] Overall incidence of post-CSDH seizures varies from 2.3 to 17% [130.] In patients with new onset seizures after CSDH surgery, the efficacy of prophylactic anti-convulsive medication has been debated and its use is not consensual. [26,54,55,56] Lower mean GCS on admission is independently predictive of seizures, most of which occur within the first three months after CSDH.[138] Patients with a previous history of epilepsy are at particular risk to develop postoperative seizures. It has been recommended that the prophylactic anticonvulsants should be started pre operatively and continued for six months.[26] Infection of subdural space is rare and may also occur with untreated CSDH. [132]

Intracerebral haemorrhage, on other hand, is uncommon in elderly (over 75yrs). It is thought to be as a result of rapid decompression leading to cortical hyperaemia with subsequent risk of spontaneous intracerebral bleed.[133] Incidence varies between 0.7-7%. It is associated with high mortality(1/3 die and another 1/3 are severely disabled).Overall Mortality varies in literature from 1-8%.[97,134,135,136] Age, general condition and neurological grading such as Low GCS score and Markwalder grade 3, 4 are contributing causes.

CSDH is a benign disease. The prognosis in CSDH depends on the age, GCS at presentation, the size of the subdural collection, and associated illnesses like cardiac and renal failure .Various grading scales are used for assessment neurological functional status of CSDH Modified Rankin Scale, Glasgow Coma Scale, Glasgow Outcome Scale, Markwalder grading scale[137,138,139]. Prognosis is good in better preoperative GCS [140] and in younger [141] patients. Markwalder grading scale is mostly preferred by different authors. [142,143,144,145] Duration of symptoms does not have any effect on the mortality or morbidity. [87]

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