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Review Paper:
Tips and Pearls in Chronic Subdural Hematoma

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ABSTRACT

Background and Aim: One of the most prevalent neurosurgery conditions is Chronic Subdural Hematoma (CSDH). Among neurosurgeons, there are various CSDH treatment approaches.

Methods and Materials/Patients: This is a narrative review examining the various aspects of the CSDH. To provide up-to-date information on CSDH, we concisely reviewed the related articles. All of the relevant articles retrieved from Google Scholar, PubMed, and Medline were reviewed, and critically analyzed. We searched for keywords including chronic subdural hematoma, burr hole craniotomy versus craniostomy, middle meningeal artery embolization, conservative therapy versus surgical therapy in CSDH, and recurrence of CSDH in published articles from 1960-2020.

Results: CSDH may present with various clinical presentations. Medical symptoms range from general and moderate symptoms (such as headache, tiredness) to severe symptoms (e.g. hemiparesis, coma). A definite trauma history may be obtained in most cases. Contrast-enhanced CT or MRI may help diagnosis. The treatment choice for uncomplicated CSDH is Burr-Hole Craniotomy (BHC). The use of drainage to decrease recurrence rates has been shown to have limited outcomes in most recent studies. Craniotomy is also used for treatment. Only asymptomatic or high-risk operative patients are subjected to non-surgical management.

Conclusion: Management of CSDH is still contentious. It is widely agreed that if neurological signs and radiological observations are present, CSDH should be evacuated. Burr-hole craniotomy appears to be the preferred surgical technique because, in most patients, it gives the best treatment outcomes. Several issues are still uncertain, including the proper surgical technique [Burr-hole craniotomy versus Twist Drill Craniostomy (TDC) and craniotomy], the advantage of 2 perforated holes over one, the location of drainage, the impact of irrigation of the hematoma, and the duration of post-operative immobilization.

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Highlights

- The recommended surgical technique is BHC since in most patients it offers the highest treatment-to-complication ratio.

- The best treatment for high-risk patients could be the bedside Twist Drill Craniostomy (TDC) under local anesthesia.

- A craniotomy is the best surgical procedure for draining CSDH with a significant membrane, acute contribution, multiple recurrences, or calcification.

- After BHC, putting drainage in a closed system leads to a substantial reduction in the rate of recurrence.

Plain Language Summary

The CSDH complex is caused by a biologically active leakage and loose vessel forming mechanism that is vulnerable to spontaneous bleeding. Risk factors include higher age, trauma, chronic alcoholism, anticoagulant, and antiplatelet consumption, and other factors that are not fully known. There is not a clear indication for conservative versus surgical treatment yet.

1. Introduction

Chronic Subdural Hematoma (CSDH) was first defined by Virchow in 1857 who named it pachymeningitis hemorrhagica interna. Trauma has a great importance in the development of CSDH [1]. The hypothesis of damage to bridging veins was later suggested by Trotter and named CSDH as a subdural hemorrhagic cyst [1]. One of the most common neurosurgical conditions is chronic subdural hematoma. There is no agreement among neurosurgeons on the use of preferential techniques for surgical care of CSDH [2]. The annual incidence of CSDH per 100,000 population is around 1.5 to 3 cases. Its incidence is growing due to the increasing elderly population and related medical conditions, such as hemodialysis, anticoagulants, or antiplatelet therapy. While surgical procedures are expeditious; one of the treatment challenges remains recurrences [3]. CSDH is predominantly an elderly condition. CSDH normally follows minor head trauma, but in half of the cases there is no history of direct head trauma. Mental state changes and focal neurological abnormalities are typical symptoms. Complications are higher in symptomatic patients, but the outcomes in patients undergoing neurosurgery are good [4].

Pathophysiological perspectives

Two key theories explain CSDH development, the osmotic theory and the hematoma capsule recurrent bleeding theory. Based on the former, owing to the osmotic pressure gradient through the semi-permeable membrane, fluid is drained from adjacent vessels into the cavity (hematoma capsule). However, hematoma fluid osmolality is the same as blood and cerebrospinal fluid osmolality, so the osmotic hypothesis was rejected [5]. Based on the latter, bleeding from hematoma capsules is an established and accepted hypothesis. The source of bleeding is from irregular and dilated blood vessels in the hematoma capsule. It has also been considered for increasing fibrinolytic activity and coagulation defects in CSDH [6].

Chronic subdural hematoma is filled with fluids, blood, and blood-thinning materials. Traumatic injury causes a breakup of bridging veins. Typically, it takes a mean of 4 to 7 weeks following trauma for a CSDH to become symptomatic but if trauma is considered as a cause of CSDH there are some issues against this. Firstly slow venous hemorrhage accumulates enough to cause symptoms within a few days from the beginning. Secondly, imaging scans may be completely normal and without any signs of bleeding, but weeks to months later, the patient may quietly develop CSDH. And thirdly, the blood pattern in CSDH, which involves the brain's convexities challenges the bridging vein as the source of bleeding. Although CSDH may contain acute bleeding areas, many of those are almost exclusively "old" hematomas that are seen on CT scans and the CSDH enlarges gradually so acute bleeding is not the only source of CSDH development and growth [7-10].

High levels of fibrin degradation products suggest excessive fibrinolysis (clot breakdown) in CSDH fluids, which results in continued bleeding. One research clearly defined fresh red blood cells in CSDH fluid, implying that new bleeding occurs with an average daily bleeding rate of 10%. In those patients who have recently been diagnosed with clinical deterioration, the highest levels of new hemorrhage have been observed, indicating that this new hemorrhage could be involved in CSDH growth. In some patients [11], with mixed density and layered CSDH, and those with recent bleeding, the levels of fibrin and fibrin degradation products are higher and this can alter the growth of hematoma and formation of different patterns [12].

There is a higher degree of plasminogen or tPA in the initial surgery in patients who subsequently experience a recurrence of CSDH. Thrombomodulin is another agent that can improve fibrinolysis and is present in CSDH fluid at elevated levels [13].

Tissue Plasminogen Activator (tPA) helps clear out clots, which is demonstrated by a slightly increased amount of post-operative discharge and a significantly lower rate of recurrence, rendering the hematoma more fluid. However, only 15 tPA-treated patients were studied in this study and, thus, this procedure is not well certified [14].

Many CSDH formation mediators play a major role in helping to form new blood vessels (angiogenesis). There is a wealth of evidence that, relative to peripheral blood and CSF (Cerebrospinal Fluid), VEGF (Vascular Endothelial Growth Factor) and VEGF-R are present in CSDH fluid with higher concentration levels. The difference in hematoma VEGF levels can be 28 times more than the serum [15-21]. Angiogenesis and excessive vascular permeability may be caused by excess VEGF, which may lead to recurrent bleeding that is involved in CSDH development. While VEGF has an antigenic and potentially anti-inflammatory function, promoting wound healing and immune defense effects, it also has a contrasting role. In neurogenesis, it has also been shown to play a role in healing Traumatic Brain Injury (TBI) [15-21]. Prostaglandin E (PGE2) which regulates VEGF expression can be found in CSDH. In the CSDH, high levels of PGE2 are identified, being related to time intervals after lesion formation.

Matrix Metalloproteinases (MMPs) play a critical role in angiogenesis. Inhibition of them suppresses the antigenic response, leading to the creation of small and shortened blood vessels. The permeability of the Blood-Brain Barrier (BBB) is increased with MMP proteolysis of junctional proteins of endothelial cells, which helps penetration of inflammatory cells into other compartments [22-27]. Based on two studies investigating MMPs in CSDH and liquid membranes, MMP-1, 2, and 9 have been illustrated as the potential factors affecting the development of brittle capillaries and leakage. These, in turn, help CSDH develop by allowing fluid to secrete from the capillaries into the cavity of the hematoma. VEGF concentrations are also correlated with MMP-2 and -9 levels, suggesting a combined angiogenesis process [22-27].

A study of CSDH pro-and anti-inflammatory cytokines revealed that both are higher than serum in CSDH blood, but the balance is substantially higher for pro- than that of anti-inflammatory ones. The balance of pro-and anti-inflammatory molecules, and how they evolve over time is important to remember. Inflammatory markers are not necessarily harmful, but at later stages, these markers may involve harmful pathways [28-33]. Studies in mice have shown that IL-1β inhibition can lead to decreased activation of microglia and neutrophil and T-cell infiltration, which may be associated with decreased loss of hemispheric tissue and decreased cognition. It is confirmed that patients with high IL-1Ra levels have more favorbale neurological outcomes than patients with low IL-1Ra level. Interleukin-1 Receptor antagonist (IL-1Ra) is used in early clinical and preclinical trials as a possible preventive therapy against brain damage caused by subarachnoid hemorrhage. IL-1β was tested in only one sample, and it may be surprising that its levels were significantly lower in CSDH fluid than in serum [34]. Interleukins-6 and-8 (IL-6 and IL-8) are monitored together because their development, likely due to a common signaling pathway, is coordinated in many instances. In response to damage and bleeding, secreted IL-6 from soft tissue is associated with autoimmune responses. Some neurotrophic effects and immune system functions, especially in the context of TBI, are also known to exist. A large rise in IL-6 levels in CSDH fluids relative to peripheral blood has been illustrated in several pieces of research [33]. IL-8 is a proinflammatory cytokine and with modulation of its own receptor expression have been shown to modulate the inflammatory response. Production of IL-8 from endothelial cells, leukocytes, and fibroblast lead to the formation of capillary tubes, endothelial cell proliferation, and release of MMP-2, all of which play an important role in angiogenesis [34]. In CSDH fluid, multiple chemokines are also present at high concentrations: chemokine ligand 2 (also referred to as MCC1), chemokine ligand 9

(CXCL9), and chemokine ligand 10 (CXCL10, protein 10 or gamma interferon-induced IP-10) [28-30].

2. Methods and Materials/Patients

This narrative review investigated the various dimensions of the CSDH. The related articles were retrieved from Google Scholar, PubMed, and Medline. They were then reviewed and critically analyzed to find the most recent information on CSDH through searching for keywords such as chronic subdural hematoma, burr hole craniotomy versus craniotomy, middle meningeal an artery embolization, conservative therapy versus Surg cal therapy in CSDH, and recurrence of CSDH in all published articles from 1960 to 2020.

3. Results

Isolated neurological defects

There have been cases with bilateral chronic subdural [31], nystagmus [32], oculomotor nerve palsy [31], upward gaze [33], reversible akinetic-rigid syndrome [34], Gerstmann's syndrome [35], quadriparesis [36], and ease of falling [37] due to CSDH. It is thought that increased intracranial pressure can cause uncal herniation and cranial nerve stretching [33]. Extrapyramidal symptoms are well-known in CSDH, which are Parkinson-like symptoms with the suggested mechanism of basal ganglion pressure, midbrain compression, and basal ganglion circulatory disruptions are caused by the anterior choroidal artery displacement and compression [34]. The literature has documented stiff-person syndrome (right-left misalignment, finger agnosia, agraphia, and stiffness of body) and progressive quadriplegia due to CSDH. These patients recovered well after the evacuation of the hematoma [30]. Acute reciprocal inhibition state secondary to basal ganglion lesion is also seen. Small ischemic lesions are typically associated with this manifestation [33, 34].

3. Risk Factors

Higher age

Our brain mass decreases as we age, increasing the brain-skull space from 6% to 11% of the total space inside the skull. This stretches the bridging veins and leaves these veins vulnerable to the effects of further brain movements within the skull [38, 39]. As confirmed in previous research, CSDH in elderly patients is a more common illness. Older individuals are more vulnerable to CSDH due to brain atrophy. Brain parenchymal atrophy allows the subarachnoid space to widen and stretch the bridging veins. The 7th decade of life was the peak age recorded in the CSDH patients [40].

Trauma

In the production of CSDH, trauma is a significant factor. However, in about 30%-50% of cases, there is no history of head injury (direct trauma). It seems that the indirect effect is more significant. Nearly half of the patients have a history of falls but do not strike the ground with their heads [41].

While minor head injuries are often not observable, serious accidents have typically been avoided. Prior head injuries are the most relevant etiological factor [42].

Chronic alcoholism

In chronic alcoholism, the recurrence of CSDH is explained by the fact that constant alcohol intake induces brain atrophy and coagulation disorders. Chronic alcoholism also has a greater risk of unrecognized head trauma. From 6% to 35%, the estimated rate of CSDH with alcoholism was reported in previous studies [43-45].

Anticoagulant and antiplatelet consumption

The use of Anticoagulant (AC) and Antiplatelet (AP) to avoid cardiovascular accidents have recently become widespread due to their specific cost-effectiveness and availability. Warfarin interferes with vitamin K metabolism in the liver which contributes to the synthesis of non-functional coagulation factors II, VII, IV, and X as well as proteins C and S. Using warfarin increases the occurrence of Intracranial Hemorrhagic Complications (ICH); while its relationship with CSDH is uncertain. One theory is that it can allow ICH to achieve clinical significance because these variables inhibit the normal bleeding mechanisms [46-52]. Therefore, the recent rise in CSDH cases is not strange as it is related to these drugs (AP/AC). With the widespread use of anticoagulant and antiplatelet, this phenomenon would possibly become more pronounced in the future. Other variables analyzed in studies do not have statistically significant effects, such as underlying illness, prior shunt surgery, and epilepsy [53]. Around 24% of CSDH patients are on warfarin or antiplatelet treatment.

While class I evidence is not available to compare the results of patients undergoing CSDH surgery with and without anticoagulant reversal, there is a consensus that when obtaining anticoagulant therapy, patients

with CSDH require rapid improvement. Otherwise, the possibility of complications during neurosurgical procedures will be quite large [52]. Vitamin K can be used to gradually adjust the International Normalized Ratio (INR) in instances where the immediate return is not critical. We can use Fresh Frozen Plasma (FFP) transfusion, Prothrombin Complex Concentrate (PCC), or recombinant Factor VIIa (rFVIIa) for rapid and urgent adjustment of INR, and for avoiding INR rebound, vitamin K should always be given adjuvant to FFP, PCC, and rFVIIa.

In favor of a conclusive decision to restart oral anticoagulation in these patients, there is no scientific evidence and few studies on this topic exist. When anticoagulants were restarted, they showed a lower risk of bleeding (11 vs. 22%) and, paradoxically, a higher risk of thromboembolism (versus prolonged discontinuation). They argued that their data cannot be deduced because of the limited cohort sample (67 patients in 3 studies) [53-58].

It is not possible to provide definitive advice on when to restart oral anticoagulation after CSDH drainage surgery; however, oral anticoagulants can be used 72 hours after surgery [59-64]. When taking these medications, patients tend to be at higher risk for CSDH. However, it is not clear if antiplatelets have been influenced by the recurrence rate [65, 66]. Stopping antiplatelet therapy for 7 days is the most effective way to reverse antiplatelet therapy. Aspirin irreversibly prevents platelet cyclooxygenase, ensuring that all platelets in the bloodstream are inactive at the time of administration of aspirin, and aggregation is blocked for the entire life of the platelets. The amount of time needed after the last aspirin administration for a full recovery of platelet function is 7 days. For patients undergoing emergency surgery, platelets may be prescribed during surgery [65]. There is little evidence to determine the optimal time for patients undergoing surgery for CSDH to resume post-operative antiplatelet re-treatment. Some research on the risk of recurrence has provided controversial results. There was a substantial difference in hematoma recurrence in patients receiving or not receiving antiplatelet medicines before surgery in 2 trials.

Other risk factors

More than one efficient factor can be present and they have a cumulative effect on the male gender, bleeding propensity, kidney disease, liver failure, chemotherapy agents, epilepsy, previous shunt surgery, renal dialysis, low intracranial pressure, and arachnoid cysts [67-87]. CSDH is more common in men because they are 2-3 times more likely to be subject to head injuries than women [66, 86].

4. Anticonvulsant Therapy in Patients With CSDH

There is no consensus on the efficacy of Antiepileptic Drugs (AEDs) usage in symptomatic CSDH patients. In patients undergoing CSDH surgery, the incidence of seizures reported ranges from 2.3% to 17% and affects 1% to 23.4% of post-operative patients. A substantial difference in the incidence of secondary seizures prescribed for AED prevention was not shown in the 2 studies [89-96]. Those 2 studies concluded that, except for patients at risk for seizures, such as alcoholics, the side effects of AED outweigh the benefits. Another research reported that patients with CSDH and new seizures had a substantial rise in mortality. Therefore, for 6 months after CSDH diagnosis, they suggest AED. The prevention of pre-operative AED has been found to minimize the occurrence of post-operative seizures in BHC-treated patients [88-94].

Management

Different authors reviewed the treatment with corticosteroids and ACE inhibitors and showed good results [95-107].

Atorvastatin

While traditionally used as a drug that lowers cholesterol, laboratory studies have shown that atorvastatin is associated with CSDH-related properties. The widespread effects of atorvastatin make it difficult to understand the processes involved in the pathogenesis of CSDH. Chan et al. [108] identified the lower need for surgical intervention in patients using atorvastatin. In a sample of 24 patients, 80 CSDH patients used a prospective placebo-controlled trial. They demonstrated a decline in surgical requirement in atorvastatin receiving patients. The paper was later retracted due to significant data errors. In addition, the same collection of published data indicates that atorvastatin may decrease CSDH recurrence after initial surgery, but this article was subsequently retracted, leading to more uncertainty about atorvastatin's true effect [109-111].

Tranexamic acid

A recent study has previously supported its clinical capacity, showing that patients with acute trauma receiving tranexamic acid had reduced deaths due to bleeding. Twenty-one patients with CSDH, 3 with a combination of surgery and tranexamic acid, and 18 with tranexamic acid alone were studied in a small sample. Both groups

had absolute clarification of their CSDH without recurrence following the duration of various opioid treatment periods [112-114].

Other conservative methods

A small study identified effective treatment with 20% mannitol in 95% of cases, resulting in no recurrence or complications in follow-ups [115]. There is a need for more studies on the normal course of CSDH and the different options of conservative care.

Surgical management

We have classified CSDH patients into 3 groups:

- Patients with neurological symptoms and radiologically proved CSDH that should be evacuated immediately.

- Asymptomatic patients who display no signs of brain compression and/or midline changes should be monitored and studied in a controlled environment. Only if there are major changes in neurological status, surgical operation is recommended.

- CSDH patients contribute to brain compression and/or midline shift, but there are no highly controversial neurological signs, and no study has assessed conservative versus surgical management in this group of patients. In order to suggest surgical treatment, there is no evidence-based research on the hematoma size.

Craniotomy

Craniotomy was the treatment of choice for CSDH until the mid-1960s. Suvin and Jelti published a case series of craniotomies and BHCs for the treatment of CSDH in 1964, showing a lower recurrence rate and better performance results with BHCs than those undergoing craniotomy surgery [115].

Twist drill craniostomy

Twist drill craniostomy can be combined with local anesthesia with a closed drainage system, making it an attractive treatment option, particularly for patients with multiple diseases. In cases where the blood is nearly completely liquefied and there is no membrane, Twist Drill Craniostomy (TDC) is probably the most effective surgical approach. It appears that the mortality rate after Twist Drill Craniostomy (TDC) is similar or even superior to BHC. Furthermore, when carried out in bed, the risk of infection potentially increases [116].

Burr-hole craniotomy

BHC is perhaps the most widely used CSDH procedure and appears to be the most successful approach because it balances the low rate of recurrence better than TDC and craniotomy. While BHC in most of the neurosurgery departments is the treatment choice for CSDH and is frequently performed; many controversies and concerns remain unanswered about organizational strategies and post-operative management. In fact, it is surprising that few studies have been conducted over the past decades to try to resolve these questions [117, 118].

Recurrence

The incidence of post-operative recurrence is between 5% and 33%. In elderly patients, late recurrence is more common. In thick hematomas, recurrence is significantly higher. Late recurrence may be defined as fluid hematoma or persistent CSDH recurrence or enlargement 3 months after surgery [119].

The exact causes of the recurrence of hematoma are not known. It appears that many variables are responsible for recurrence. Partial removal of the hematoma may result in the complete destruction of the hematoma. The rate of recurrence of homogeneous and trabecular hematoma is lower than that of layered or multilayered hematoma [120]. A high incidence of recurrence is associated with high-density, mixed lesions [121]. Recurrence may result from primary and metastatic dura mater diseases. Intracranial hypotension can lead to CSDH recurrence [121]. In determining intracranial hypotension and preventing recurrence, spinal MRI and/or radionuclide cisternography is useful. High levels of tPA, IL-6, VEGF, and bFGF in subdural fluid and outer membrane increase recurrence rate. Increased recurrence is associated with the dense subdural membranes found during surgery and the brain remaining deep to the dura and bone at the end of surgery [119-122].

Hematoma oxygen replacement is a useful treatment for CSDH that has been associated with decreased rates of recurrence. Subcutaneous suction drainage helps the residual hematoma to be drained continuously and is associated with low rates of recurrence and complications. Optimum management should be the simultaneous closure of the CSF dural fistula during CSDH discharge. CSDH management alone can contribute to recurrence without fixing CSF leakage. ACE (Angiotensin-Converting Enzyme) inhibitors treatment decreases the recurrence rate. In chronic cases, middle meningeal

artery embolization is useful to disrupt blood flow to the capsule of the hematoma and avoid recurrence [123].

Spontaneous resolution

In patients with normal neurologic findings, neurological and radiological monitoring coupled with medical care may be necessary. Without the surgical procedure, non-traumatic SDH or non-traumatic hygroma in infants may often undergo substantial resolution within a few months. Post-traumatic CSDH spontaneous resolution can occur in patients without any accompanying coagulation, but rarely. Careful treatment should be taken into account if the neurological and physical state of the patient changes [124].

Middle meningeal artery embolization

In the treatment of CSDH, MMA (Middle Meningeal Artery) embolization has been demonstrated in non-randomized case-control studies. To treat bleeding that is venous in nature, it is important to understand the mechanism by which arterial blood supply stops. Understanding the mechanism of our therapeutic effects allows the population to qualify for MMA embolization [125-127]. It may be a good choice to perform proximal MMA embolization using a coil because unwanted leakage of embolized particulate matter can cause eye and facial nerve injuries. Whether the CSDH recurrence is causative or consistent, or merely a coincidence, embolization as a treatment for CSDH is prohibited by the ophthalmological origins of MMA. The choice of embolizing materials is another technical issue. Most studies have used particles of Polyvinyl Alcohol (PVA) and a small number have used liquid embolizing materials [125-127].

It has also been proposed to use MMA in the treatment of CSDH by observing that MMA is submerged in CSDH. Contrast enhancement of dura, CSDH capsule, CSDH fluid, and septation was shown by CT CSDH images obtained after MMA embolization suggesting continuous vessels between the membranes of CSDH and MMA [125-127].

Compared to conventional open surgery for subdural drainage, endovascular MMA embolization has several benefits, making it a very attractive invasive treatment option for the elderly. Under local anesthesia, transradial or transfemoral access through the skin is usually well tolerated with a low risk of complications. MMA embolization procedures may also be done under mild sedation or local anesthesia, thus reducing the risk of endotracheal intubation and general anesthesia causing cardiac and respiratory complications. Physicians also practice post-operative bed rest as a way to allow the brain to regrow after a subdural evacuation, but after MMA embolization, this is not required because subdural evacuation does not occur. Patients are normally outpatient within a few hours of embolization. Finally, without the need for skin incisions, craniotomy, and drainage, the risk of infection is significantly reduced [125-189].

Head position after surgery

Immediately after surgery, placing the head 30 degrees in CSDH did not have a major effect on outcome and recurrence. Without raising the risk of recurrence in patients 65 years or older, early post-operative mobilization may prevent post-operative pneumonia and urinary tract infections. In the standing position, the rate of recurrence is substantially more [127, 128].

Complications

- Rapid hematoma decompression contributes to focal hyperemia of the cerebral cortex, which, in conjunction with causes such as labile hypertension and amyloid cerebral angiopathy is considered to be the cause of intracerebral hemorrhage. By adding a subdural drainage drain, the intracerebral complication might be iatrogenic. By checking whether the bridging vessel or cortex is damaged or the drain has even entered the brain parenchyma, careful positioning of the subdural drainage under visual control, and further irrigation of the subdural compartment will avoid this very rare complication [129-131].

- Seizure (1%-23%);

- Post-operative infections;

- Pneumonia;

- Brainstem hematoma: This is a very rare complication of subdural hematoma's evacuation. This complication can be avoided by slow decompression, management of clotting disorders, and maintaining proper blood pressure before surgery. Rapid asymmetric decompression, resulting in vascular dysfunction and a rapid increase in blood flow to the brain, can lead to hematoma of the brain stem. Therefore, slow rate decompression and evacuation are recommended for CSDH drainage to prevent severe complications such as secondary intracranial hematoma.

- Tension pneumocephalus [5].

In elderly patients with multiple illnesses and polymorbidity, all complications are more common. Patients over 85 years of age have a lower rate of recurrence [5].

5. Conclusion

In neurosurgery patients, chronic SDH is one of the most common and significant factors. The new membrane forms around the hematoma with delicate neocapillaries. Inflammation contributes to VEGF production and release of the profibrinolytic and anticoagulant factors in the hematoma fluid, elements that are thought to boost the growth of rebleeding and SDH. In the initial assessment of CSDH, CT plays a vital role as it correctly confirms the diagnosis and can predict the age of the hematoma. The only evidence-based advice is the placement of a closed system drain during surgery to avoid recurrence. Anti-platelet therapy should be stopped for 7 days in patients with mild symptoms and anticoagulants transferred to vitamin K alone and with near clinical and radiological follow-up. Antiplatelet therapy may be stopped for those who require emergency surgery, and platelets should be administered during the procedure. Rapid conversion can be achieved using PCC or FFP, an adjunct to vitamin K, in patients receiving anticoagulants. No evidence exists to assess the best time for antiplatelet or anticoagulant re-treatment to resume.

Ethical Considerations

Compliance with ethical guidelines

There were no ethical considerations to be considered in this research.

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Authors' contributions

All authors equally contributed to preparing this article.

Conflict of interest

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