Chapter 14
Organized Chronic Subdural Hematoma

Mustafa Balevi, Ayşe M. Dumlu, and Mehmet Turgut

14.1 Introduction

Chronic subdural hematoma (CSDH) is a frequently encountered neurosurgical disease characterized by the progressive collection of blood and its breakdown products in the subdural space of the intracranial cavity [74]. Interestingly, CSDH may present a different architecture with multiple loculations and septations with thickened membranes and solid encapsulated areas, called an "organized" CSDH, in some patients [62, 80].

Von Rokitansky reported the first calcified CSDH in an autopsy in 1841 [89]. The first surgery for organized CSDH was performed in 1930 [26]. Feghali et al. reported that CSDH is a problematic disease with an incidence of 1.7–20.6 per 100,000 persons per year, with a greater incidence in the elderly [21]. The incidence of organization or calcification of CSDH is only 0.3–2.7% and its incidence has progressively increased over the years, in particular in the aged population using an anticoagulation/antiplatelet medication [9, 44, 59, 61]. CSDHs tend to develop in

M. Balevi (✉)
Department of Neurosurgery, Konya Numune State Hospital, Konya, Turkey

A. M. Dumlu
Department of Neurosurgery, Aydın Adnan Menderes University Faculty of Medicine, Aydın, Turkey

M. Turgut
Department of Neurosurgery, Aydın Adnan Menderes University Faculty of Medicine, Efeler, Aydın, Turkey

Department of Histology and Embryology, Aydın Adnan Menderes University Health Sciences Institute, Efeler, Aydın, Turkey

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adults and elderly with brain atrophy [83]. Younger adults with compliant brains are far less prone to experience this complication. CSDH is usually seen in older patients or children under 2 years of age [83].

14.2 Dynamic Pathophysiology of Organized CSDH

Pathophysiologically, both formation and expansion of the CSDH are complicated with the formation of new membranes as a result of certain inflammatory events, including angiogenesis [21]. Later, it leads to fibrin deposition, formation of subdural membranes, and the development of fragile capillaries in the membranes [14, 19, 43]. Importantly, it has been suggested that the existence of plasminogen activator in the CSDH increases fibrolytic activity and failure of hemostasis, resulting in "rebleeding from the outer membranes" and "plasma effusion" within the subdural space [35, 39, 90]. More importantly, the degree of reabsorption of the blood collection in the subdural space is a vital factor for the resolution or progression of the CSDH [14, 22, 39, 76]. Furthermore, various coagulation disorders play a role in the development of the new membranes and fragile vessels at the junction of the inner and outer membranes over 6–12 months, resulting in recurrent hemorrhages and finally a solid fibrous structure with fusion of the inner and outer membranes [24, 36, 62, 66, 70, 84, 93].

In some patients, a CSDH may possess a liquefied hematoma within multiple cavities of different ages, as a result of the multiple recurrent bleedings into the cavity over a long period [19]. Afterwards, calcification, metaplastic or dystrophic, and ossification may develop within the CSDH, although its exact mechanism for development is not clear [3, 5, 53, 54]. Calcification may occur 6 months to several years after hemorrhage [17, 33, 60, 94]. After a few years of calcification, ossification of the CSDH may occur [47, 61].

Recently, the senior author and his colleagues on this chapter reported that patients with calcified or ossified CSDH, a rarely encountered disease, may remain asymptomatic for many years [87]. Unfortunately, it has been suggested that the capsule of calcified or ossified CSDH may be adherent to the leptomeninges and the underlying brain surface [44]. Interestingly, the clinical features of calcified or ossified CSDH are very similar to those of noncalcified or nonossified hematomas [18, 58].

An organized CSDH is rare in children [85]. Children with organized CSDH often have a history of ventriculoperitoneal shunt or subduroperitoneal shunt operations in infancy [17, 59]. Shunt procedures for hydrocephalus, meningitis, encephalitis, and epileptic seizures are the other predisposing factors for organized CSDH occurrence [17]. In the non-elderly, surgery should be considered in asymptomatic patients with organized CSDH for the prevention of possible future brain damage due to cerebral compression [30, 44, 46, 55, 58, 63, 64, 87].

14.3 Clinical Presentation of Organized CSDH

Clinically, headache, nausea and vomiting, lethargy, confusion, apathy, dizziness, weakness, behavioral changes, voiding dysfunction with decreased bladder capacity and the presence of high-amplitude overactive detrusor contractions but intact sphincteric response, and epileptic seizures are classical symptoms of patients with an organized CSDH [5, 28, 44, 58]. In some patients, an organized CSDH frequently presents with symptoms of dementia. Calcified or ossified CSDH may remain asymptomatic for many years. Organized CSDH generally occurs in the elderly although it may present in young patients but is rarely seen in infants.

14.4 Radiology of Organized CSDH

The appearance of an organized CSDH on computed tomography (CT) has a mixed density, is multiseptated, with signs of new hemorrhage, midline shift, and thickening, or calcification of the inner membrane (Fig. 14.1) [4, 6, 9–11, 13, 37, 44, 63, 82, 92]. On magnetic resonance imaging (MRI), an organized CSDH is hyperintense on both T1- and T2-weighted images, but it may be hypointense on T1 and hyperintense on T2 scans in some patients; however, it may have a hypointense web-like structure within the cavity of the CSDH (Figs. 14.2 and 14.3) [9–11, 23, 29, 42, 79]. MRI with contrast enhancement may reveal the existence of connective tissue as a sign of maturation of a calcified or ossified CSDH [5]. A thickened inner membrane may be seen on both MRI and CT (Figs. 14.1 and 14.4) [63, 94]. If the initial CT confirms that the CSDH is multiloculated and multilayered, MRI study with contrast enhancement is indicated [15]. The calcified or ossified CSDHs, though rare, could mimic a calvarial mass. Images with contrast administration are essential to determine whether there is any associated primary or metastatic dural disease [16].

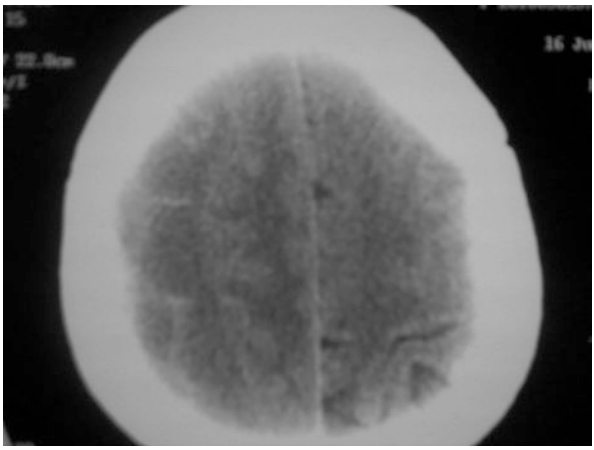

Fig. 14.1 Axial non-contrast computed tomography (CT) showing right, mixed density, multiseptated organized chronic subdural hematoma (CSDH), with signs of new hemorrhage, thickened inner membrane, and the presence of midline shift

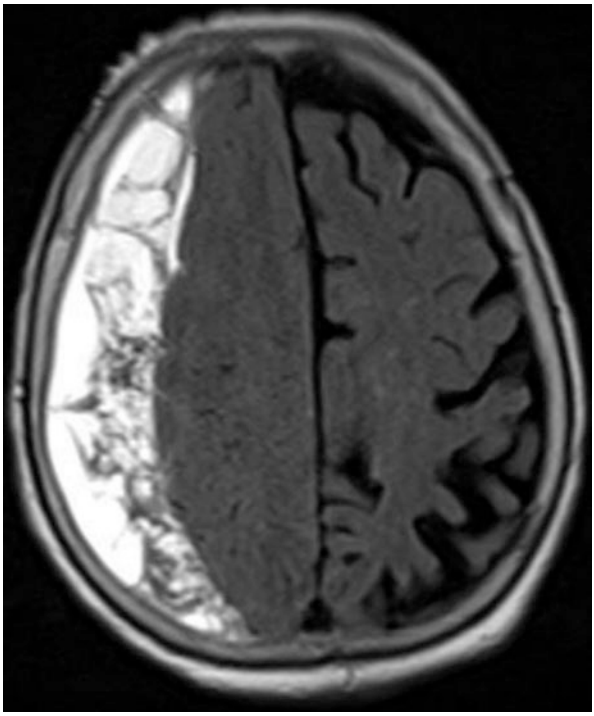

Fig. 14.2 Preoperative T1-weighted magnetic resonance imaging (MRI) demonstrating multiple loculations within the CSDH, which appear as hypointense web-like structures within the organized CSDH over the right cerebral hemisphere

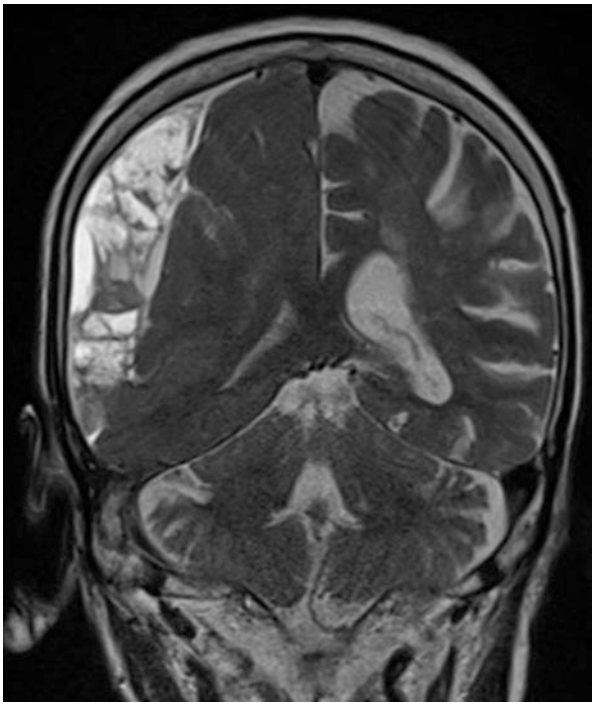

Fig. 14.3 Preoperative T2-weighted MRI showing multiple intrahematomal loculations with a hypointense web-like structure within the organized CSDH over the right cerebral hemisphere

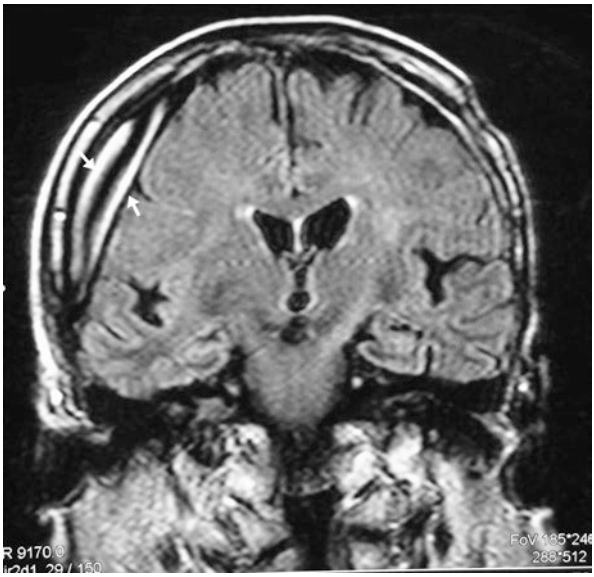

Fig. 14.4 Preoperative T1-weighted contrast-enhanced MRI showing a thickened inner and outer membrane within the right hemispheric organized CSDH (white arrows)

14.5 Risk Factors for Organized CSDH

Various risk factors including elderly age, alcohol consumption, systemic comorbidities such as hepatic and/or renal dysfunction, coagulopathies or anticoagulant drugs, and long-term use of aspirin or anti-inflammatory drugs have been suggested as reasons for recurrence of an organized CSDH [25].

14.6 Prophylaxis for Seizure

Although there is conflicting evidence about seizure prophylaxis in CSDH, antiepileptic drug prophylaxis may be given in patients with a high risk for seizures [12]. The mortality is high in postoperative seizure patients; therefore prophylaxis for seizures should be administered in high-risk patients [12].

14.7 Treatment and Complications

Many surgical treatment options have been suggested for the management of patients with CSDH, but the best choice of surgical technique for CSDH is still controversial [45, 49]. From the surgical point of view, burr hole irrigation or craniotomy with or without membranectomy are popular treatment options [88]. In

clinical practice, burr hole craniostomy with closed system drainage is generally used in the majority of patients with CSDH, although it may be ineffective in some patients [2, 20, 25, 86].

Even today, the primary risk factor responsible for recurrence of a subdural collection in the postoperative period is not clear and the best option for the surgical treatment of patients with CSDH is still controversial. Some authors suggested that the development of subdural membranes may be an obstacle for re-expansion of the cerebral cortex and neurological recovery of the patient after surgery, resulting with postoperative complications such as recurrence in patients aged 70 years or more [7, 9, 45, 50].

In 2003, Weigel et al. reported that craniotomy for CSDH should be a last resort for reducing the recurrence rate, because of the fact that it has a higher morbidity (12.3%) in those treated with craniotomy in contrast to those treated with twist drill craniostomy (3%), and burr hole craniostomy (3.8%) [91]. In a similar study, Callovini et al. also reported that craniotomy should be used only in patients with recurrent CSDH or failure of expansion of the cerebral cortex after attempted drainage [15]. In 2011, Kim et al. reported that a large craniotomy with extended membranectomy as the initial treatment was successful in reducing the reoperation rate in patients with CSDH compared with a small craniotomy with partial membranectomy [37]. This surgical technique may be useful in elderly patients since the development of an acute subdural hematoma (SDH) or recurrence of a CSDH may be possible owing to the blood oozing from the membrane incision lines [51, 80, 87, 88].

On the other hand, Link et al. suggested that middle meningeal artery embolization is a minimally invasive technique for evacuation of CSDH after failure of conservative management or prophylactic treatment of recurrence of a CSDH following surgery, although its exact role remains unclear [41, 95].

Recently, some authors reported that the endoscopic removal of an organized CSDH has obtained good results [48, 67, 78]. Ishikawa et al. suggested that the drainage technique using a rigid endoscope and aspiration tube through a small craniotomy may be used in patients with multiloculated CSDH [33]. As a rule, a large craniotomy has been advocated for surgical treatment of an organized CSDH by many authors, although a craniotomy procedure may cause various postoperative hemorrhagic complications and recurrence of an organized CSDH [5, 32, 34, 37, 50, 65, 73]. Removal of thick or calcified membranes of the organized CSDH with a large craniotomy may provide re-expansion of the cerebral cortex following surgical evacuation of the hematoma [5, 38, 40, 50, 76, 79]. Old age, persistence of subdural air, and prior cerebral infarction are causative factors for poor brain re-expansion after surgery [52]. Importantly, some authors have suggested that brain compliance is a critical factor in the re-expansion of the cerebral cortex following the evacuation of a CSDH [45, 50]. There is no doubt that poor re-expansion of the brain tissue is associated with recurrence of a CSDH. From this perspective, influx of the air into the subdural space must be avoided during surgical intervention [52]. On the other hand, an extended membranectomy procedure for organized CSDH has a high risk of surgical damage to the underlying arachnoid surface and the potential for rebleeding from new capillary structures [13, 54, 55, 69]. However,

Acakpo-Satchivi and Luerssen suggested that partial inner membranectomy may cause brain herniation through the inner membrane in patients with a calcified or ossified CSDH [1].

Theoretically, re-accumulation of a CSDH within 3 months following the surgical intervention is considered an "early recurrence," while persistence or enlargement of a CSDH at 3 months or more after surgery represents a "late recurrence" [56]. The incidence of early recurrence is between 0 and 30% after surgery for an organized CSDH treated by craniotomy with membranectomy [32, 34, 40, 66, 79]. It has been stated that the main cause of recurrence in these patients treated by a large craniotomy was the fragile capillaries at the junction of the inner and outer membranes, resulting in repeated multifocal hemorrhages [11]. It has been speculated that recurrence is a result of stretching and rupturing bridging veins entering the venous sinuses such as the superior sagittal sinus [37, 76].

Basically, the meaning of the term "pneumocephalus" is the existence of air in the intracranial cavity (Fig. 14.5). It is known as "tension pneumocephalus" (TP), an important life-threatening complication after surgery, when intracranial air causes neurological deterioration due to increased intracranial pressure, in particular after evacuation of a CSDH [27, 31]. One of the authors of this chapter reported that the incidence rate of TP after a large craniotomy with membranectomy for OSDH was 28.5% [11, 71, 72, 77]. Clinical symptoms include headaches, nausea and vomiting, dizziness, depressed neurological status, and epileptic seizures [72]. The subdural air with high tension easily separates and compresses both frontal lobes, resulting in compression of the frontal lobes with a widened interhemispheric

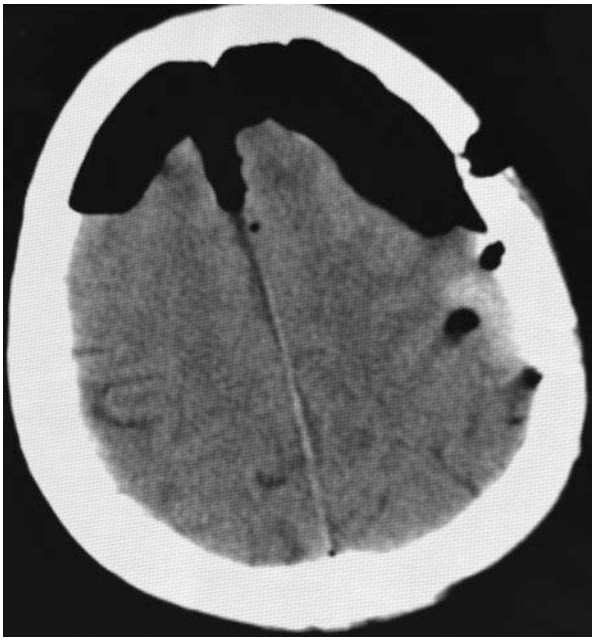

Fig. 14.5 Axial CT scan showing tension pneumocephalus

space between the frontal poles, thus mimicking the silhouette of Mt. Fuji on CT, called a “Mt. Fuji” sign [68]. The main mechanism for recurrence of CSDH is rupturing of stretched veins entering the venous sinuses such as the superior sagittal sinus [77]. Another sign of pneumocephalus is the existence of multiple small air bubbles in the subarachnoid space, particularly in the basal cisterns.

A controlled decompression through a closed water-seal drainage system was applied to the patients for 2 days [8, 81]. The most appropriate treatment of TP is lying in a straight position, fluid replacement therapy, and breathing 100% O_2 [57]. Postoperative epileptic seizures that are medically treated were reported in 25–50% patients undergoing large craniotomy with extended membranectomy [52].

14.8 Prognosis

The mortality rate in patients with organized CSDH varied from 0 to 15.6% [75]. Callovini et al. reported only one fatal case (3%) that was complicated by intraventricular and subarachnoid hemorrhage [15].

14.9 Conclusion

A large craniotomy with extended membranectomy for patients with organized CSDH should be undertaken as a main procedure, despite its high risk of complication. In patients with organized CSDH, the worst prognostic factors were the neurological condition before surgical intervention and the patient’s age >70 [25]. TP and residual SDH are frequently seen complications in elderly patients.

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