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Original Article

A novel imaging classification system for the neuroendoscopic treatment of chronic subdural hematoma

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Abstract: Chronic subdural hematoma (CSDH) is common, especially in patients over 50 years of age, and represents about 10% of all intracranial hematomas. The pathogenesis, diagnosis, and treatment of CSDH are controversial. The purpose of this study was to document the clinical application of a novel imaging classification system for the neuroendoscopic treatment of CSDH. This was a prospective study of sixty patients who underwent neuroendoscopic CSDH treatment beginning in January 2017, with a 6-month follow-up. Hematomas were classified into two types based on imaging features: simple (type I) and complex (type II). Complex type was further subclassified as septated (type II-A), stratified (type II-B), recurrent (type II-C), thin-layer (type II-D), bilateral (type II-E), or mixed (type II-F). Most hematomas were located on the left side. Type II hematomas had fibrous septa and bridging veins in the cavities. Bender classification and Glasgow Outcome Scale (GOS) scores were improved after neuroendoscopic surgery and hematoma thickness was improved significantly in all CSDHs on days 1, 7, and 14 after surgery (all $P<0.05$). Lung infection, pneumocephalus, and seizures occurred in 17, 12, and 8 patients, respectively. Neither a recurrence of symptoms nor CSDH occurred based on the analysis of images. All patients recovered well and none suffered additional bleeding, recurrence, or intracranial infection. This novel imaging classification for CSDH provides a useful guide for the successful neuroendoscopic treatment of CSDH.

Keywords: Chronic subdural hematoma, classification, neuroendoscopic surgery

Introduction

Chronic subdural hematoma (CSDH) is a common disease in neurosurgery, accounting for approximately 10% of all intracranial hematomas. Most of these hematomas occur on the frontoparietal lobe convexity, with blood volumes ranging from 30-170 mL [1]. While CSDH can occur at any age, morbidity and mortality are higher in patients over 50 years old, and the incidence increases gradually with age [2, 3]. The pathogenesis of CSDH may be linked to brain injury, brain atrophy, spontaneous lower cranial pressure, blood coagulation dysfunction, anticoagulant drugs, and other factors. However, the pathogenesis, diagnosis, and treatment of CSDH are controversial, and CSDH is an encapsulated structure encompassed by a characteristic "new membrane". Especially for the treatment, there are different surgical methods. While most patients have a history of mild head trauma, traumatic subdural effusion and acute subdural hematoma are the two main causes of the disease. CSDH is diagnosed by computed tomography (CT) and magnetic resonance imaging (MRI) and treatments include twist-drill and burr-hole evacuation, craniotomy, and neuroendoscopy. The surgical method is simple, safe, and effective. However, regardless of the surgical method, septa within the hematoma lead to recurrence in a small number of patients after twist drill or burr-hole evacuation [3, 4], and common complications include hematoma recurrence, intracranial gas

accumulation, effusion, cerebral hemorrhage, and epilepsy. Because hematoma recurrence is associated with a poor prognosis, it is important to identify the factors that lead to recurrence. Neuroendoscopy, a new treatment for CSDH, allows the removal of septa and can reduce recurrences. Based on CT and MRI features, we identified simple and complex types of CSDHs and determined the relationship of these types to the clinical outcomes and complications after neuroendoscopic surgery in this study. Moreover, we further classified CSDH using neuroendoscopy providing additional information as a basis for treatment.

Materials and methods

Patients

This prospective study began in January 2017 with 60 CSDH patients at Renmin Hospital of Wuhan University. All patients were diagnosed with CSDH by CT and/or MRI, underwent neuroendoscopic treatment, and were followed for 1-6 months with postoperative imaging. Atorvastatin (20 mg) was administered daily to all patients for 3 months at the same time without corticosteroids. This study was approved by the Medical Ethics Committee of Renmin Hospital of Wuhan University.

Imaging

All patients underwent preoperative CT and/or MRI. Early-stage CSDH generally showed high signal intensity on T1- and T2-weighted images. Late-stage CSDH showed low signal intensity, higher than that of cerebrospinal fluid, on T1-weighted images and high signal intensity, higher than that of cerebrospinal fluid, on T2-weighted images. Uneven, mixed-signal intensity on both T1- and T2-weighted images was present at intermediate stages of CSDH. Hematoma laterality, CT density, MRI signal intensity, thickness, and associated midline shift were recorded.

Surgical procedure

The surgical technique has been previously described [3]. Briefly, a straight or curved scalp incision was made over the hematoma and a small craniotomy, approximately 3 cm in diameter, was performed. After the dura was incised, liquid blood was slowly aspirated using a needle and syringe to reduce intracranial pressure. A rigid endoscope (Aesculap, Tuttlingen, Germany) was introduced into the hematoma cavity to break through one or more septa and remove the hematoma. After removal, a silicone subdural drain was placed in the cavity under direct vision and the cavity was irrigated with normal saline at 37°C through the drain until the irrigant was clear. The drain was left in place while the dura was closed. The bone flap was replaced, and the scalp incision was closed.

Statistical analysis

Statistical analyses were performed using SPSS software version 19.0 (IBM Corp., Armonk, NY, USA). Continuous data are expressed as mean ± standard deviation and were compared using the unpaired *t*-test. Categorical data are expressed as numbers with percentages and were compared using the chi-square test. $P<0.05$ was considered significant.

Results

Patient characteristics

Among the 60 patients studied, 42 (70%) were men and 18 (30%) were women. Overall mean patient age was 66.2±13.42 years. The mean ages of men and women were 64.36±13.29 years and 70.5±12.74 years, respectively. Presenting symptoms included headache (n=16), dizziness (n=10), hemiplegia (n=10), ataxia (n=5), speech disturbance (n=2), urinary incontinence (n=6), epilepsy (n=4), blurred vision (n=5), oculomotor nerve paralysis (n=1), and consciousness disturbance (n=2). Inattention and memory loss were commonly associated symptoms. Demographics and clinical characteristics of patients according to CSDH type and subtype are shown in **Table 1**. Patient age, gender, and presenting symptoms did not significantly differ between patients with simple (type I) versus complex (type II) hematomas (described in the following section).

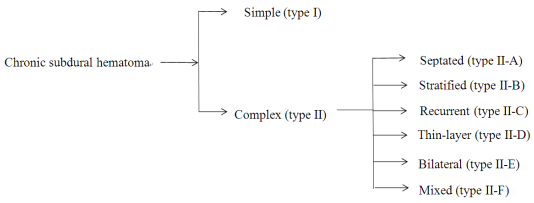
Table 1. Patient demographics and clinical characteristics

Clinical type	No.	Gender		Age (y)	Complication			History of head trauma
		Male	Female		Hypertension	Diabetes	Anticoagulant or antiplatelet	
Type I	9	6	3	60.22±9.89	3	0	1	3
Type II-A	16	9	7	67.38±10.57	8	3	2	5
Type II-B	10	6	4	70.90±6.19	3	3	1	3
Type II-C	5	5	0	64.60±15.55	2	1	0	4
Type II-D	5	4	1	76.00±5.83	1	0	0	1
Type II-E	8	7	1	61.38±13.5	3	2	1	2
Type II-F	7	4	3	64.71±19.15	4	1	1	1

Values are number or mean ± standard deviation. CSDH, chronic subdural hematoma.

Classification of CSDH

CSDH was classified into two types according to image features: simple (type I) and complex (type II). Complex (type II) was subclassified into six subtypes: septated (type II-A), stratified (type II-B), recurrent (type II-C), thin-layer (type II-D), bilateral (type II-E), and mixed (type II-F).



Simple (type I): This type has three main characteristics: no stratification, no septation, and no organization (**Figure 1**).

Septated (type II-A): This type describes a hematoma divided into separate portions by fibrinous septa, which are an independent risk factor for recurrence [3]. The hematoma and the related abnormal septa within should be removed to reduce the risk of recurrence and other complications (**Figures 2**, **3**).

Stratified (type II-B): This type shows stratified layers of blood with different degrees of density signal intensity and thickness (**Figure 4**).

Recurrent (type II-C): This type describes a recurrent hematoma after CSDH surgery (**Figures 5**, **6**).

Thin-layer (type II-D): This type has no clear definition; we tentatively set the maximum hematoma thickness <1.5 cm (**Figure 7**).

Bilateral (type II-E): This type is located bilaterally. Although midline shift is less common with this type compared with unilateral hematomas, the symptoms are typically severe. Patients with bilateral CSDH are older because of an age-related decrease in brain volume and increased venous fragility. Patients with bilateral CSDH show rapid and progressive deterioration and should be treated as soon as possible, even if the neurological impairment is mild (**Figure 8**).

Mixed (type II-F): This type shows various image characteristics comprising two or more of the above types (**Figures 9**-**12**).

Hematoma laterality and surgical data

Most hematomas were located on the left side. Fibrous septa and bridging veins were found in the cavities of complex (type II) hematomas (**Table 2**).

Surgical results

For each type and subtype of CSDH, CT after neuroendoscopic surgery confirmed satisfactory hematoma clearance. Hematoma thickness improved significantly in all types and subtypes of CSDH on days 1, 7, and 14 after surgery (all $P<0.05$), indicating that surgery was effective (**Table 3**).

Complications and surgical outcomes

Complications according to CSDH type and subtype are presented in **Table 4**. Patients with all CSDH types and subtypes recovered well after surgery, as demonstrated by Bender classification and Glasgow Outcome Scale scores (**Table 5**). There were no recurrences of symptoms or CSDH based on analysis of images.

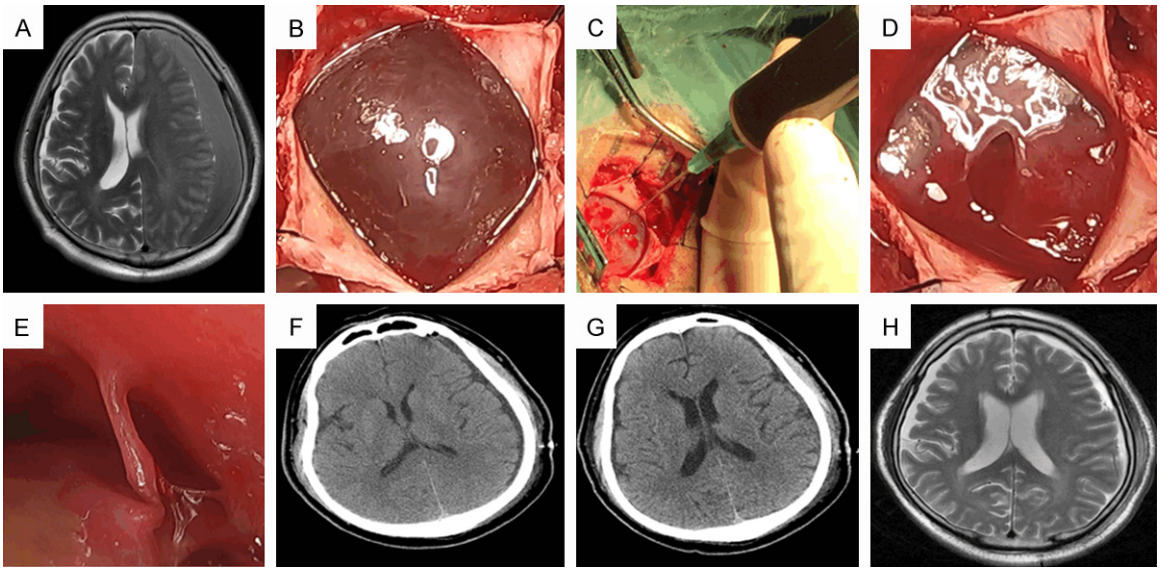


Figure 1. A 72-year-old man with a headache and weakness of the right limb for 5 days was admitted to the hospital after sustaining an external head injury 1 month earlier. Images and the surgical procedure indicated a simple (type I) CSDH. A. MRI showed a low-signal intensity hematoma overlying the left frontotemporoparietal region with left-to-right midline shift and no septation or blood stratification. B. The outer membrane was revealed after dural opening. C. The liquefied hematoma was aspirated with a syringe to decrease intracranial pressure. D. The hematoma was uniform, without septation or stratification. E. Bridging veins were seen by neuroendoscopy. F. CT on the first postoperative day showed hematoma removal. G. On the fourth postoperative day, CT showed restoration of brain tissue to its original position. H. MRI showed complete restoration of brain tissue and absence of midline shift 2 weeks later.

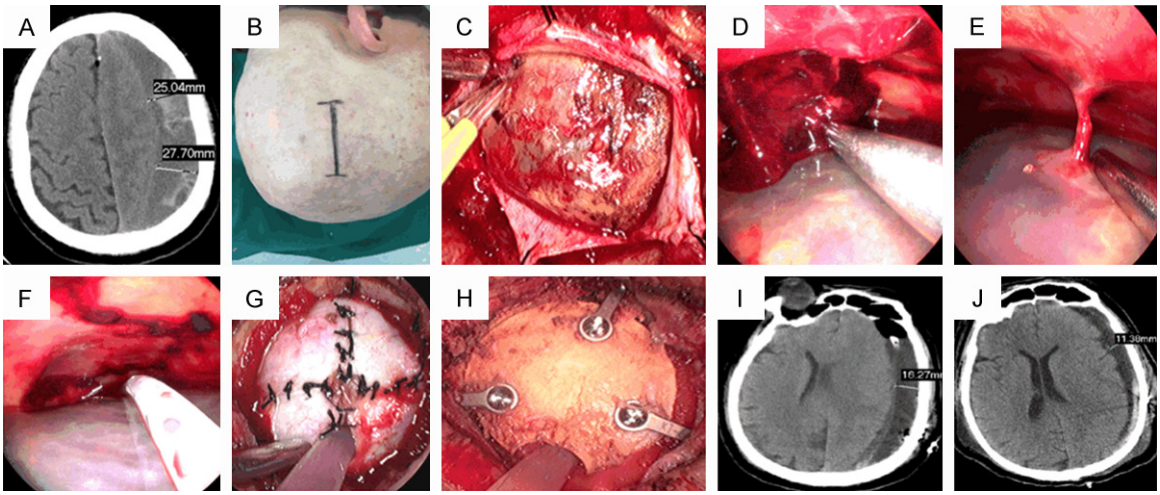


Figure 2. A 68-year-old man was admitted to the hospital for 8 days because of right limb weakness. Images and the surgical procedure indicated a septated type II-A CSDH. (A) CT showed a left frontotemporoparietal hematoma with distinct septa and varying thickness (range, 25.04-27.7 mm). (B) A surgical incision was made over the thickest part of the hematoma. (C) After the dura was opened, a thick organized hematoma with membrane was found. (D) Neuroendoscopy showed septa in the hematoma cavity and (E) a bridging vein. (F) A subdural drain was placed using direct endoscopic visualization and (G) the dura mater was sutured tightly. (H) The bone flap was replaced and fixed. (I) CT on the first postoperative day (maximum distance between the brain and skull: 16.27 mm) and (J) the sixth postoperative day (maximum distance: 11.38 mm).

Discussion

Although CSDH is common, its pathogenesis is unclear. Virchow first proposed the mechanism of dural inflammation for CSDH in 1857 [5]; however, studies have shown a multifactorial pathogenesis [3, 4]. CSDH often occurs in middle-aged and older adults, and older men are

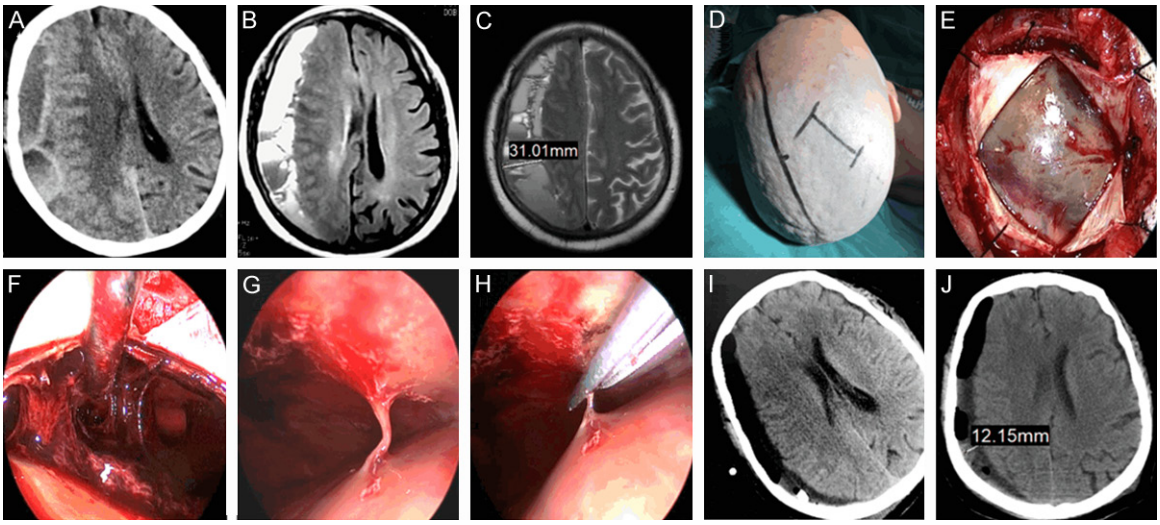


Figure 3. A 70-year-old woman was admitted to the hospital for 2 weeks because of left limb weakness. Images and the surgical procedure indicated a septated type II-A CSDH. (A-C) The right frontotemporoparietal hematoma had obvious septa on CT. The maximum thickness was 31.01 mm. (D) A frontal neuroendoscopic approach was taken. (E) The outer membrane was revealed after the dura was opened. (F) Mixed hematoma and fibrous septa were seen in the hematoma cavity. (G and H) An elongated bridging vein was seen and electrocoagulated to reduce the risk of rebleeding. Postoperative CT on the first (I) and seventh (J) days confirmed satisfactory hematoma clearance.

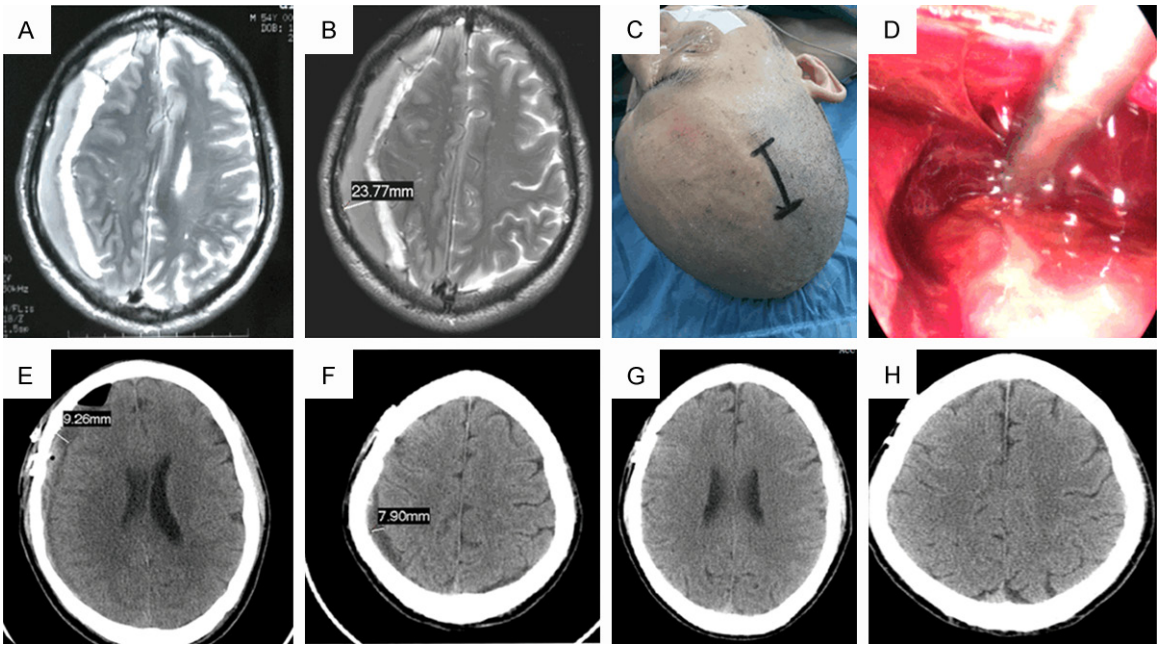


Figure 4. A 55-year-old man was admitted to the hospital after 10 days of dizziness and headache following a head trauma 3 months earlier. Images and the surgical procedure indicated a stratified type II-B CSDH. (A and B) Preoperative MRI showing a right frontoparietal hematoma with stratified layers of blood. (C) A frontoparietal neuroendoscopic approach was taken. (D) Obvious delamination was observed intraoperatively. (E and F) CT on the first day after surgery and a 3-week (G) and a 4-month (H) follow-up showed satisfactory hematoma clearance.

more likely to develop CSDH than women because the width of the cerebral sulcus is more significantly affected by age in men [6]. Among the 60 CSDH patients in our study, the number of men was much higher than women (70% vs. 30%).

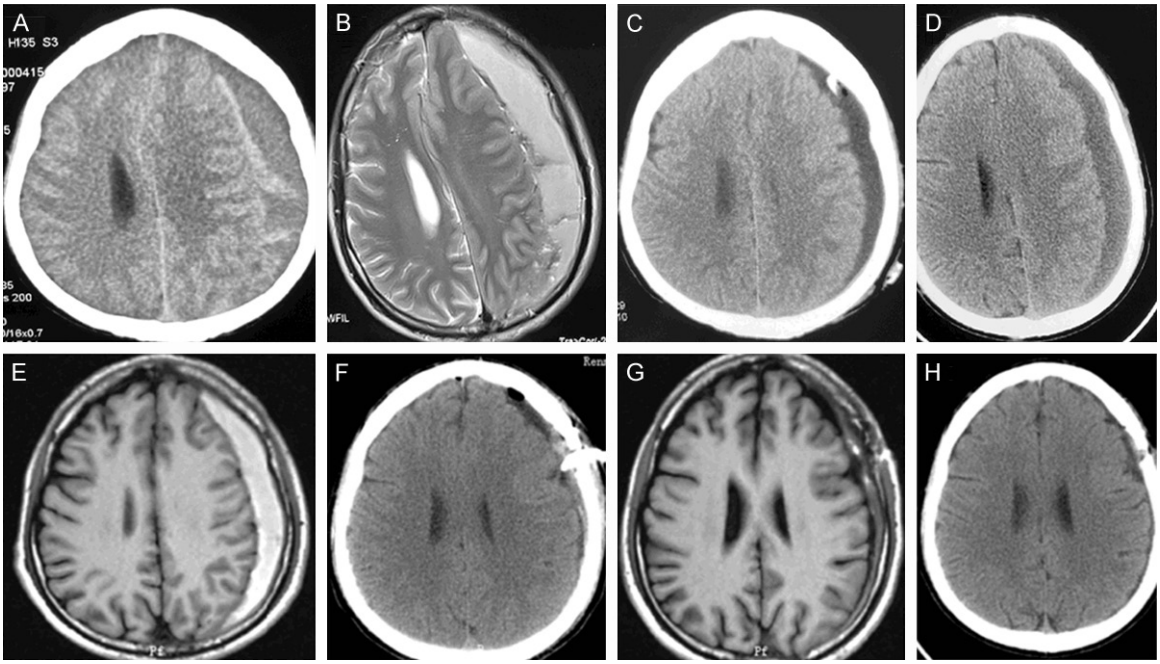


Figure 5. A 44-year-old man was admitted to the hospital more than 1 month after CSDH surgery. Images of a recurrent type II-C chronic subdural hematoma. (A-D) Initial follow-up CT after conventional hematoma drainage confirmed satisfactory hematoma clearance. (E) One month after surgery, the patient presented with a hematoma at the same site. (F) CT after neuroendoscopic surgery showed hematoma removal and subdural drain. Postoperative MRI (G) and CT (H) 1 month later showed no recurrence.

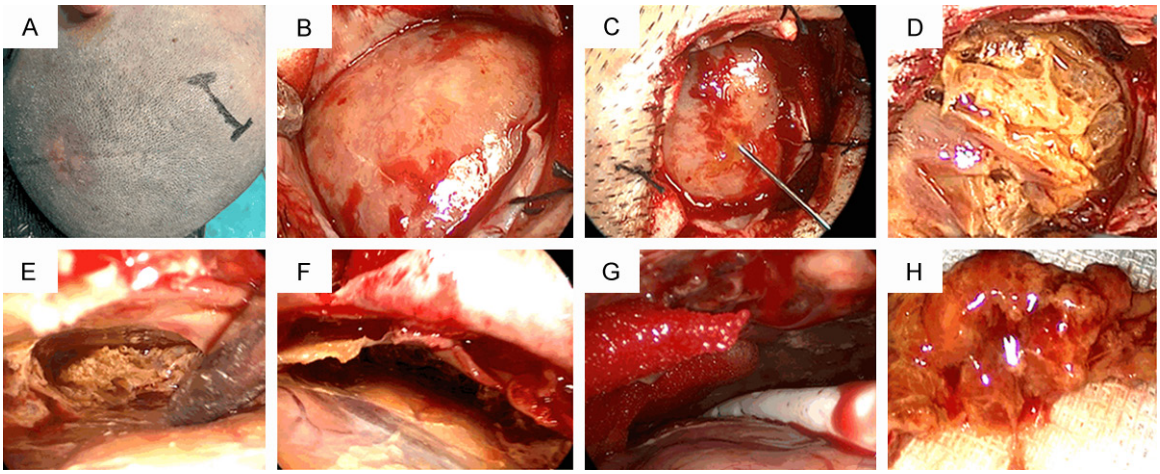


Figure 6. Images and the surgical procedure indicated a recurrent type II-C chronic subdural hematoma. A. An occipital neuroendoscopic approach was taken. B. After opening the dura, a thick-walled hematoma wall was observed. C. A needle with a syringe was used to aspirate liquefied blood to reduce intracranial pressure. D-F. Multiple fibrous septa in the hematoma cavity were fully cleared and the hematoma removed. G. A subdural drain was placed using direct endoscopic vision. H. Photograph of septa and other tissue removed from the hematoma cavity.

CT is most commonly used to diagnose CSDH and is effective, accurate, and non-invasive. For treatment, surgery is superior to conservative management. Surgical procedures include burr-hole craniotomy for drainage, double burr-hole drainage, lavage through a small bone window, burr-hole craniotomy and needle drainage, middle meningeal artery embolization, subperiosteal drainage, twist drill craniostomy, subdural-peritoneal shunting, and exfoliation of the hematoma capsule [3, 7-9]. However, the best procedure remains controversial [10]. While all

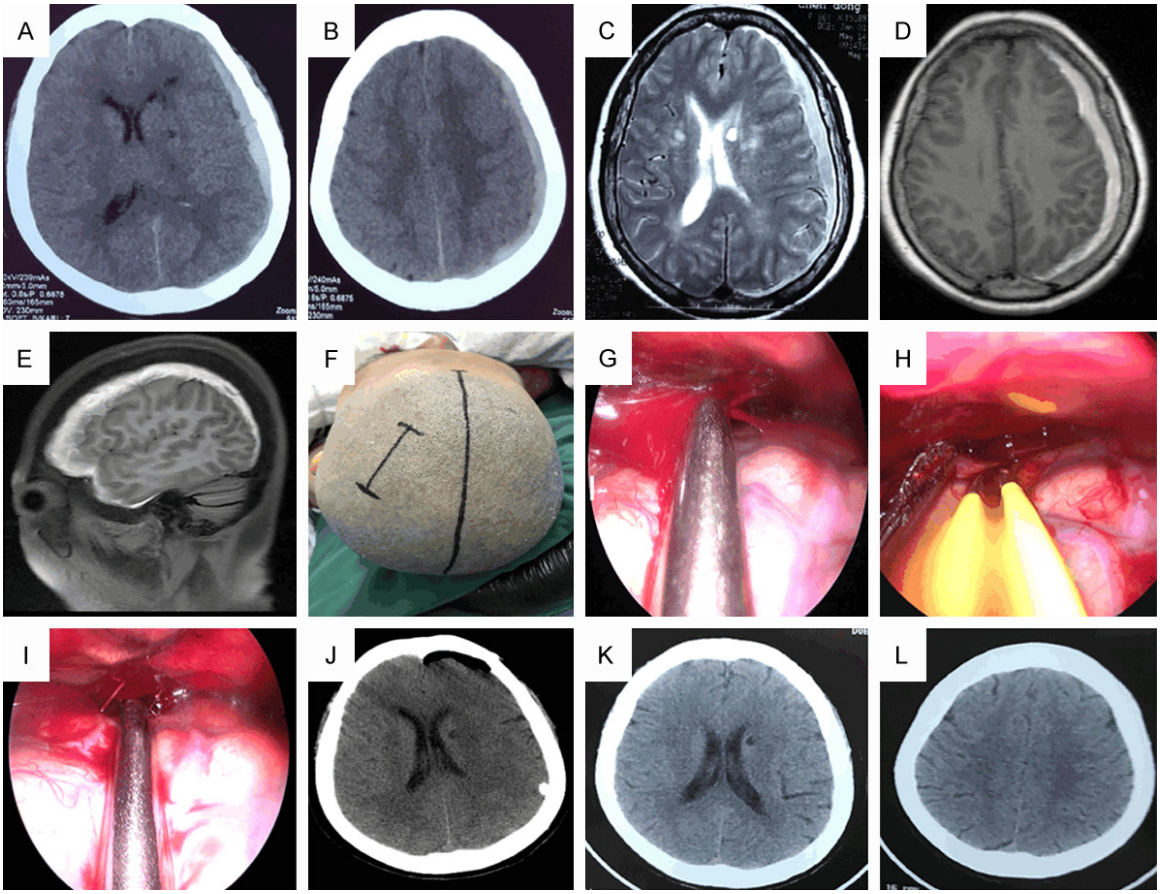


Figure 7. A 66-year-old woman was admitted to the hospital for 5 days with dizziness and headache. Images and the surgical procedure indicated a thin-layer type II-D CSDH. A-E. Preoperative MRI and CT revealed a left frontotemporoparieto-occipital thin-layer hematoma. F. A bifrontoparietal neuroendoscopic approach was taken. G-I. Active bleeding was seen intraoperatively; bipolar electrocoagulation was used to stop the bleeding under direct vision. J. CT on the day after surgery confirmed satisfactory hematoma removal. K, L. CT at 1-month follow-up confirmed that the brain tissue was restored to its original position.

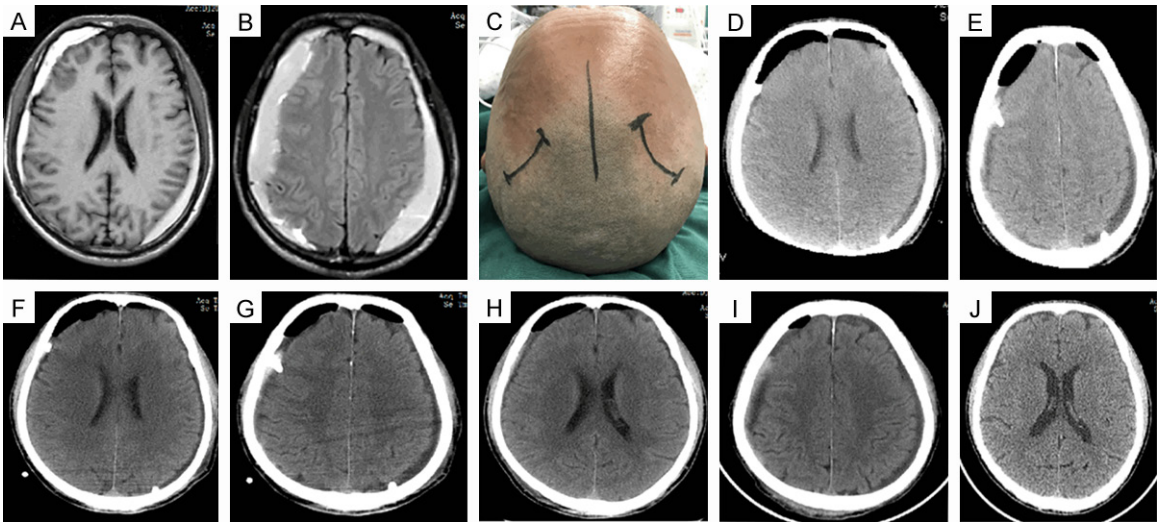


Figure 8. A 62-year-old man was admitted to the hospital with a 2-month history of headaches. Images and the surgical procedure indicated a bilateral type II-E CSDH. (A and B) Preoperative MRI showed bilateral hematomas. (C) A bilateral frontoparietal neuroendoscopic approach was taken. (D and E) CT scan on the day after surgery confirmed satisfactory hematoma clearance. (F and G) CT on the third day after surgery. (H and I) CT 1 week after surgery and on postoperative day 15 (J) showed no recurrence.

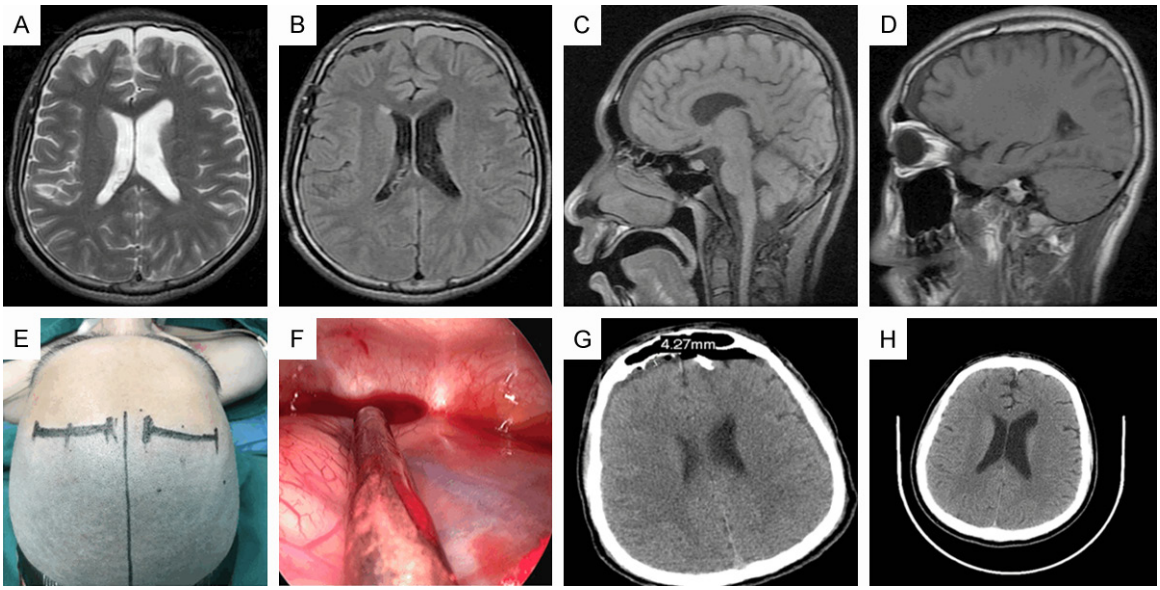


Figure 9. An 18-year-old man was hospitalized in a psychiatric department for 2 months for treatment for mania, which was caused by CSDH. Therapy was ineffective and involved anti-psychotic drugs, nutritional nerve therapy, and therapy to improve circulation. Images and the surgical procedure indicated a mixed type II-F CSDH. A-D. Preoperative MRI revealed that thin bilateral frontal subdural effusions caused pressure on the frontal lobes. E. A bifrontal neuroendoscopic approach was taken. F. Endoscopic view of the hematoma cavity, dura, and subarachnoid space. G. Postoperative CT confirmed satisfactory hematoma clearance. H. At a 3-month follow-up, CT showed no recurrence.

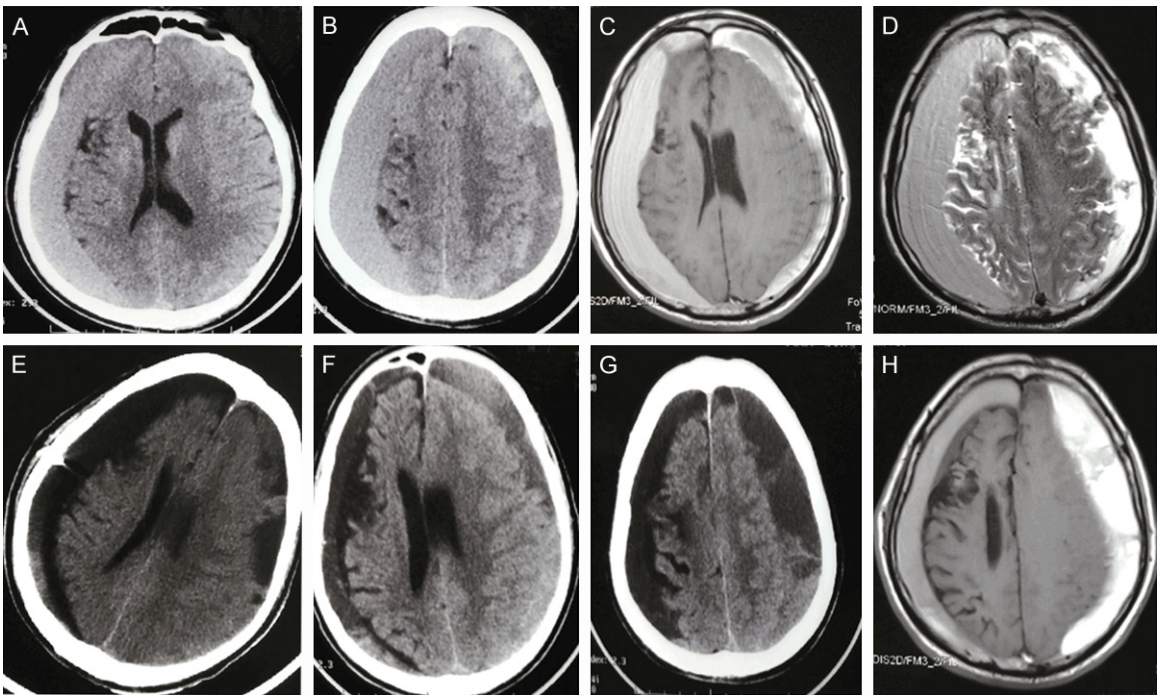


Figure 10. The images are of a 77-year-old man, 11 days after the CSDH operation. Images are of a mixed type II-F CSDH. A-D. Preoperative MRI showed bilateral septated hematomas. E-H. Images after drainage at another hospital showed persistent hematomas. Postoperatively, the patient remained in a trance-like state. He was transferred to our hospital where the hematomas were removed under neuroendoscopy. Intraoperatively, hematoma septation, stratification, and organization were observed. Neuroendoscopic surgery resulted in the resolution of symptoms.

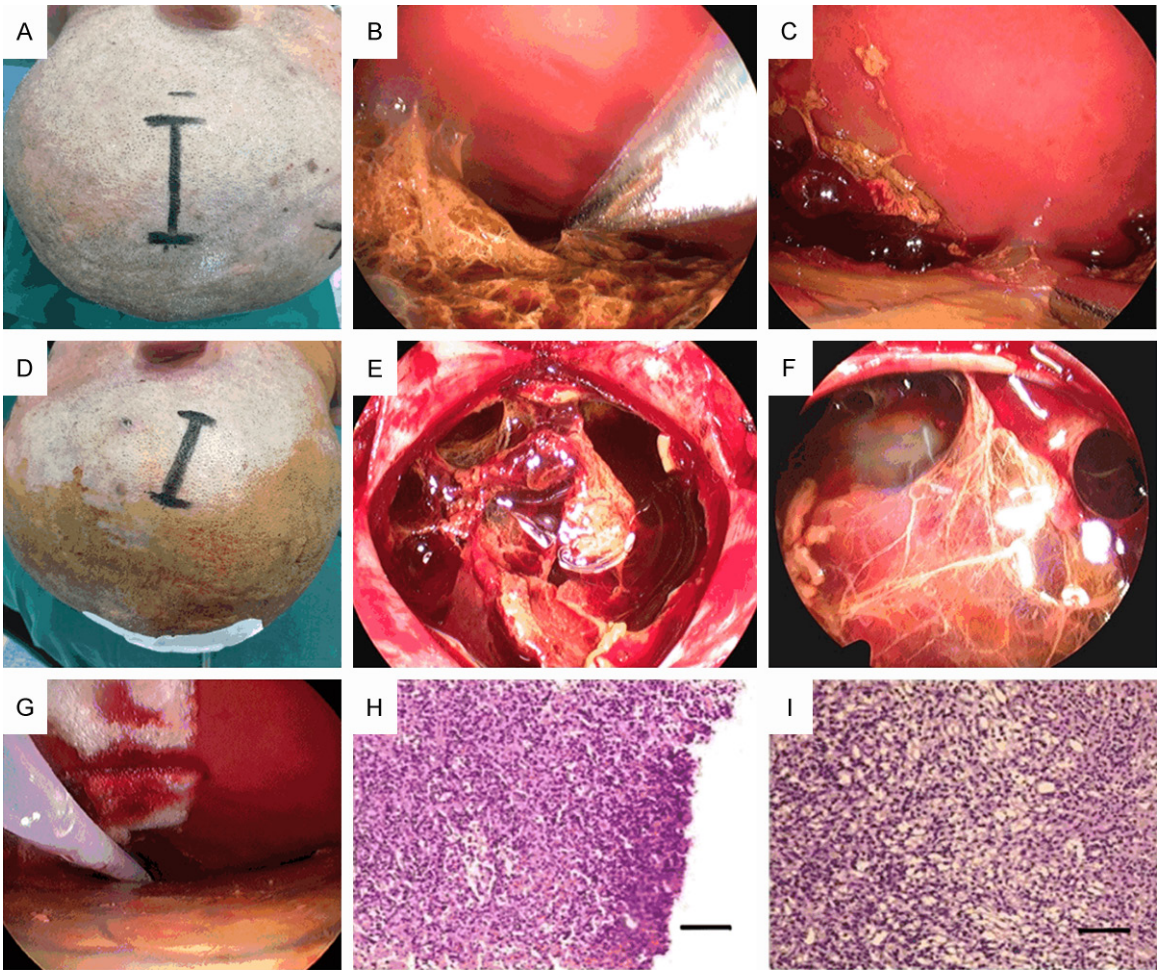


Figure 11. Surgical procedure and histologic findings of a mixed type II-F CSDH patient who underwent bilateral neuroendoscopic surgery. A-C. Left surgical incision and neuroendoscopic findings. D-F. Right surgical incision and neuroendoscopic findings. A large amount of fibrous septal tissue with neovascularization was seen on both sides, which was fully cleared under direct vision. G. After removal of the hematoma and septa, subdural drains were placed under direct vision. H, I. Histopathological analysis of the removed tissue showed fibrous tissue hyperplasia with an increased number of lymphocytes, neutrophil infiltration, and partial hematoma organization. Bar = 20 mm.

these techniques can provide satisfactory results, recurrences, which range from 0.4-33.3% [11], and surgical complications are risk factors associated with a poor prognosis [9, 12, 13].

Four factors affect CSDH recurrence after surgical treatment: patient-related factors, imaging factors, surgical factors, and drug-related factors [13]. Diabetes, bilateral hematomas, and a maximum hematoma thickness >20 mm are associated with recurrence, as are trabecular structures and residual space between the brain and skull [14]. We found that many trabecular structures are just pillars of connective tissue; however, other authors have found them to be vascularized [2, 5]. For neuroendoscopic CSDH surgery, active bleeding from trabecular structures and acute blood clots signifying recent bleeding are noteworthy findings. One previous study suggested that finding white adventitia intraoperatively, which represents the degree of inflammation in the evolution of CSDH, is a risk factor for recurrence [15].

All patients with aphasia in our study had left-sided hematomas and frontal base dilatation on CT. Three of our patients had experienced recurrence after previous burr-hole drainage and then underwent neuroendoscopic surgery, which found septa within the hematoma cavity. A CSDH is an encapsulated structure encompassed by a characteristic “new membrane” [16]. After complete removal of the membrane,

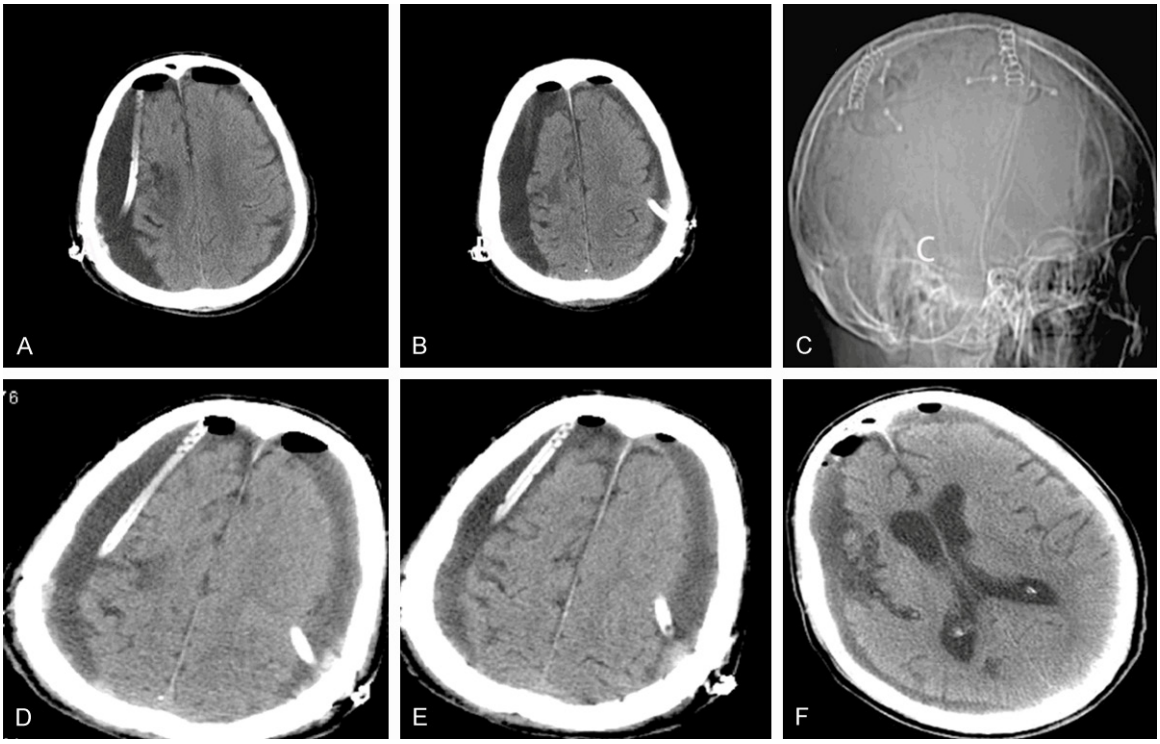


Figure 12. Images after neuroendoscopic surgery for a type II-F CSDH. A-C. CT on the day after surgery confirmed satisfactory hematoma clearance. Subdural drains are visible bilaterally. D and E. CT on the fifth day after surgery showed decreased hematoma size. F. CT on the fifteenth day after surgery showed a further decrease in size.

Table 2. Hematoma laterality, presence of septa and/or bridging veins, duration of subdural drainage, and length of hospital stay

Clinical type	Hematoma site		Trabeculae and fibrin septa	Bridge veins	Drainage days (d)	Hospital stay (d)
	Left	Right				
Type I	6	3	0	0	2.78±1.25	17.67±6.50
Type II-A	9	7	10	8	4.25±1.44	17.44±4.66
Type II-B	7	3	8	4	2.50±1.12	21.10±4.89
Type II-C	4	2	5	3	5.40±1.02	38.2±37.04*
Type II-D	5	0	2	1	1.80±0.75	18.40±4.76
Type II-E	8	8	6	3	3.50±1.12	15.60±2.50
Type II-F	4	3	6	4	3.57±1.05	15.00± 2.14

Values are number or mean ± standard deviation. *, recurrent types: recurrent disease and multiple surgeries.

the patients in our study recovered well without recurrence after 6 months of follow-up. Some studies suggest that the absence of a subdural drain is a risk factor for recurrence [17]. Generally, the subdural drains in our study were left in place for approximately 2 days but no longer than 1 week, which is similar to previous studies; longer durations are associated with a risk of intracranial infection [11]. Because surgical subdural drains can significantly improve the appearance of postoperative images after CSDH, some studies recommend their use [18].

Preoperative anticoagulation therapy is an important risk factor for postoperative acute subdural hemorrhage and both anticoagulation and antiplatelet therapies are associated with a higher incidence of recurrence [12]. In addition, atorvastatin use is beneficial in preventing CSDH and its recurrence in some patients [19], as are corticosteroids [20]. In our study, atorvastatin at 20 mg per day was administered to all patients for 3 months without corticosteroids to eliminate anticoagulation as a factor in recurrence.

Table 3. Hematoma thickness by type/subtype of chronic subdural hematoma before and after neuroendoscopic surgery

Clinical type	Hematoma thickness (mm)				*P*-value
	Pre-op	One day Post-op	One week Post-op	Two weeks Post-op	
Type I	23.02±6.63	9.51±3.74	9.20±1.93	7.51±3.14	0.0094
Type II-A	22.00±7.68	8.41±3.56	8.00±3.78	7.25±3.93	0.0039
Type II-B	19.35±6.69	9.61±3.63	8.21±2.57	9.26±3.47	0.0286
Type II-C	29.80±8.06	18.77±6.80	10.87±4.00	9.41±2.35	0.0047
Type II-D	27.47±2.29	10.58±2.61	8.12±3.39	7.92±3.58	0,0001
Type II-E	20.21±6.20	10.69±5.48	9.09±2.23	7.77±3.86	0.0800
Type II-F	22.44±6.73	9.76±3.72	8.81±2.32	7.47±2.46	0.0300

Values are mean ± standard deviation. *P*-values were derived from unpaired *t*-test. $P<0.05$ was considered significant.

Table 4. Complications following neuroendoscopic surgery for CSDH

Clinical type	Rebleeding	Pneumocephalus	Epilepsy	Intracranial infection	Lung infection	Pressure sores	Recurrence (n)
Type I	0	2	0	0	3	0	0
Type II-A	0	5	3	0	5	3	0
Type II-B	0	2	0	0	2	1	0
Type II-C	0	1	2	0	2	0	0
Type II-D	0	0	0	0	1	1	0
Type II-E	0	2	2	0	2	0	0
Type II-F	0	0	1	0	2	1	0

Table 5. Bender classification and Glasgow Outcome Scale (GOS) scores before and after neuroendoscopic surgery

Clinical type	Bender classification score		GOS score	
	Pre-op	One week Post-op	Pre-op	After 6 months
Type I	1.11±0.31	0.00±0.00	4.67±0.67	5.00±0.00
Type II-A	1.38±0.60	0.13±0.33	4.75±0.56	4.94±0.24
Type II-B	1.30±0.46	0.00±0.00	4.60±0.66	5.00±0.00
Type II-C	2.00±1.10	0.20±0.40	4.40±0.49	4.60±0.49
Type II-D	1.60±0.49	0.20±0.40	4.80±0.40	5.00±0.00
Type II-E	1.50±0.50	0.00±0.00	4.50±0.71	4.88±0.33
Type II-F	1.71±0.70	0.00±0.00	4.29±0.70	5.00±0.00

Values are mean ± standard deviation.

Neuroendoscopy was first implemented as a treatment for CSDH in 1988 [21]. Its advantages include the following [2, 3, 22]: (1) The endoscope can be advanced deeply in all directions of the hematoma cavity under direct vision, providing direct visualization of the hematoma cavity from several angles, allowing complete removal of firm non-liquefied blood clots and the opening of septa in the hematoma cavity, resulting in a high hematoma clearance rate. (2) Recurrence risk factors, such as blood vessels and septa in the hematoma cavity, can be located and treated under direct vision, reducing the risk of postoperative rebleeding and recurrence. (3) Neuroendoscopic surgery causes less bleeding than traditional procedures and does not cause postoperative adhesions or obstruction of the internal membrane wall. (4) Subdural drains can be placed under direct vision to avoid the risk of inserting them into brain tissue or causing trauma to septa, cerebrocortical vessels, or bridging veins; blind subdural irrigation is also avoided. (5) Neuroendoscopic surgery is associated with a low incidence of complications, short hospital stays, and low cost. Our patients recovered well after neuroendoscopic surgery and experienced no recurrence during follow-up.

Our CSDH neuroendoscopic technique fully explores the hematoma cavity for septa, blood vessels, and other structures. After hematoma removal, a subdural drain was placed at the

lowest level in the hematoma cavity under direct vision. The drain was used for intraoperative irrigation with 37°C normal saline until the drainage was clear and was left in place for continuous postoperative drainage. Most simple hematomas, hematomas with soft adhesions of the inner and outer membranes, and soft hematomas without septa can be removed with repeated irrigation. However, for difficult hematomas, those with obvious septa, and mixed-type hematomas, aspiration followed by irrigation was less effective; these hematomas must be crushed or separated under endoscopy and then removed. Non-liquefied hematoma residues with different textures can be seen and directly removed using multiple endoscopes. A variety of thin and thick fibrinous septa, dural thickenings, bridging veins, and other structures were seen in most complex hematomas. Depending on the thickness, septa can be penetrated directly or incised after electrocoagulation to allow communication between separate cavities, facilitate hematoma removal, and reduce cavity size. In patients with bilateral hematomas, which are associated with a higher rate of recurrence, one study showed no difference in recurrence between bilateral and unilateral drainage [23]. We typically treated the side with the largest hematoma first, followed by the contralateral side; however, a sudden intraoperative unilateral release of pressure may displace brain tissue. Several studies have shown that the recurrence rate is higher for unilateral versus bilateral drainage. However, bilateral drainage of small hematomas increases the risk of brain injury [17].

In patients with coagulation abnormalities, we administered preoperative platelet or plasma transfusions, as appropriate, and waited for improved blood coagulation before surgical treatment. Two patients in our study had thrombocytopenia, and four had a history of antiplatelet or anticoagulant therapy. With platelet transfusions, vitamin K injections, and other preoperative preparation, no obvious intraoperative bleeding or postoperative rebleeding occurred.

Patients at high risk for intraoperative or postoperative bleeding or recurrence should undergo neuroendoscopic CSDH surgery. These include patients with: (1) hematomas that are inhomogeneous, incompletely liquefied, or hard and organized; (2) hematomas with multiple compartments; (3) hematomas with ruptured bridging veins or fibrous trabeculae in the cavity; (4) abnormal coagulation function; (5) continued anticoagulant or antiplatelet therapy; (6) recurrent hematoma after previous surgery; and (7) multiple comorbidities. In these patients, burr-hole drainage has higher risk and it is less effective than neuroendoscopic surgery. Neuroendoscopy is superior because it provides good visualization, is less invasive, and has fewer complications, thereby resulting in reduced recurrence, residual hematoma, or even death.

The disadvantages of neuroendoscopic treatment of CSDH include a relatively long operation and specialized surgical skills. Surgeons who are not skilled in neuroendoscopy pose a higher risk of iatrogenic injury for the patient [15]. Rigid endoscopes have navigation limits such that the peripheral portions of the hematoma and septa cannot be completely removed; however, flexible endoscopes may achieve better surgical results.

Although the patients in our study achieved good results, the number of patients was limited and follow-up was short-term. Future long-term, multicenter studies of neuroendoscopic CSDH treatment are needed. In the interim, our data should assist physicians in the individualized treatment of CSDH.

Conclusions

This study produced a CSDH classification system based on CT and MRI findings, illustrated various hematoma structural features identified during neuroendoscopic treatment, and demonstrated the safety and efficacy of neuroendoscopic surgery. Surgically treating several CSDH features associated with recurrence that can be identified endoscopically may improve the clinical outcome. Future outcome studies of neuroendoscopic CSDH treatment that focus on pathological and biochemical assessments are warranted.

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Disclosure of conflict of interest

None.

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