ORIGINAL ARTICLE

Is prophylactic anti-convulsive treatment necessary in subdural hematomas?

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ABSTRACT

BACKGROUND: Subdural hematoma (SDH) is usually an emergent clinical condition in neurosurgery. The relationship between the SDH and epilepsy is not well established. Therefore, the use of anti-convulsive treatment in patients with SDH is controversial. The aim of this study is to analyze the presence of seizures in patients who underwent surgery for SDH.

METHODS: Patients who were operated on for SDH in our department between 2016 and 2021 were reviewed retrospectively. Demographic features, Glasgow Coma Scale (GCS) score at admission, type of SDH, location, etiology, type of surgical intervention, presence of seizures, and re-operation were evaluated.

RESULTS: There were 175 patients with SDH. There is a statistically significant difference between the frequency of seizures and the type of SDH. More seizures were observed in acute SDH than in the others. There is also a statistically significant difference between the GCS score and the frequency of seizures. Patients with a GCS score <12 at admission had more frequent seizures than patients with a score of 12 or higher. No statistically significant difference was found between factors such as etiology, re-operation, hematoma location, and the development of seizures.

CONCLUSION: Anti-convulsive treatment may be recommended in patients with acute SDH and a low GCS score at admission. Further studies with larger series should be performed to determine the most appropriate anti-convulsive agent for patients with SDH.

Keywords: Anti-convulsive treatment; epilepsy; subdural hematoma; surgery.

INTRODUCTION

Subdural hematoma (SDH) is the most common type of extra-axial blood collection in the intracranial region, requiring frequent emergency neurosurgical intervention.[1,2] These hematomas are usually secondary to the rupture of bridging veins between the cerebral cortex and dura mater, and minor superficial vascular injuries may also cause SDH in association with arachnoid tears.[3] SDH compresses on the underlying brain parenchyma and causes a headache, nausea, gait disturbance, confusion, and focal neurological deficits.[4] The treatment of such hematomas may be conservative or surgical.[5] The most commonly used surgical options are the evacuation of hematomas using burr holes or craniotomies.

One of the complications of SDH is the development of seizures. Seizures are rare in patients with chronic SDH (cSDH), as the underlying brain parenchyma is intact in most cases. Seizures have been attributed to the presence of encapsulated membranes, occult cortical injury, mass effect, or acute bleeding.[6,7] It is generally accepted that the occurrence of seizures in such patients, although not conclusively proven, is an indicator of a poor prognosis.[8]

A significant portion of patients presenting with SDH may show a fluctuating course of seizures. Such seizures may be encountered at the first admission, during the hospitalization, or in the follow-up period. Most are diagnosed and treated with acute symptomatic seizures (ASz) or epilepsy. Epilepsy

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has been reported to occur in up to 25% of patients after surgery in SDH.[9]

The aims of this study are to evaluate the presence of seizures in the patients who were operated on for SDH and to determine the risk factors for epilepsy.

MATERIALS AND METHODS

Patient Population

The data of patients who were operated on for SDH in our department between 2016 and 2021 was reviewed retrospectively. Patients who were followed up in our clinic for SDH but did not undergo surgical intervention during the specified date range and patients with previously known epilepsy were not included in the study. In addition, cases with pathologies other than SDH in the intracranial region (intraparenchymal hematoma, subarachnoid hemorrhage, tumor, etc.) during the first admission were not included in the study.

In the study, which included a total of 175 patients, factors such as age, gender, Glasgow Coma Scale (GCS) score at admission, type of SDH, location, etiology, type of surgical intervention, presence of seizures, and re-operation rate were evaluated.

Ethical approval for this study was obtained from the ethics committee of our institution.

Radiological Evaluation

Contrast-enhanced cranial magnetic resonance imaging was performed in all cases at the time of admission, after the computed tomography (CT) scan and clinical stabilization. Pre- and postoperative CT scans were reviewed to assess location (right, left, or bilateral), density (hyperdense, isodense, hypodense, or mixed density), presence of hematoma septa, pre- and postoperative midline shift, hematoma thickness, and the presence of residual hematoma. The thickness of the initial and residual hematomas was determined according to the axial sections of pre- and postoperative non-contrast CT scans.

Seizure Evaluation

Status epilepticus was clinically defined as generalized tonic-clonic seizures lasting longer than 5 min or complex-partial seizures lasting longer than 10 min, according to the latest International League Against Epilepsy (ILAE) definition.[10,11] ASz, either documented clinical seizure, the symptom was defined as clinical suspicion by ictal pattern on electroencephalography (EEG) recordings or interictal epileptiform discharges on EEG recordings, as recommended by ILAE, and was in close temporal association with acute brain injury.[12] All EEG reports were reviewed by a special neurologist.

Surgery

In all cases, SDH was evacuated using an open craniotomy or burr holes. In all cases, possible residual hematomas were evaluated by CT scans in the early postoperative period. The patients were followed up in the intensive care unit for the first 2 days postoperatively under normal conditions. They were later transferred to the inpatient clinic. Seizure follow-up was performed in the postoperative period as well as in the preoperative period. Prophylactic anticonvulsive treatment was not performed on any patient because it was not recommended in the literature.[13]

Statistical Analysis

IBM SPSS Version 25.0 for data analysis (Armonk, NY: IBM Corp.) statistical package program was used for statistical analysis. Numerical variables of patient data are expressed as mean±standard error of the mean (SH) and minimum (lowest)-maximum (highest values). Categorized variables were explained as the number of patients (n) and percentage (%) with descriptive statistics. The relationship between categorical data in independent groups was first examined with the Chi-square test, and Fisher's exact test was used in the analyses where the Chi-square assumptions were not met. Differences at the $P<0.05$ level were considered statistically significant.

RESULTS

Patient Population

Of the 175 patients, 26.3% (n=46) were female and 73.7% (n=129) were male. The mean age was 65.4 years (range: 21–94 years). Demographic factors and general characteristics of the cases are summarized in Table 1.

Based on the etiology, 41.7% (n=73) of the cases were spontaneous, and 58.3% were traumatic SDH. Based on the type of SDH, 49 patients had acute, 33 patients had acute-subacute, 38 patients had subacute, 31 patients had subacute-chronic, and 24 patients had cSDH. Hematoma was on the left side in 45.1% (n=79) of the cases, on the right side in 41.1% (n=72), and bilateral in 13.7% (n=24).

The hematoma was evacuated by burr hole in 8.6% (n=15) of the cases, by open craniotomy in 81.7% (n=143), and by craniectomy in 9.7% (n=17).

While seizures occurred in the early postoperative period in 24% (n=42) of the cases, no seizure was detected in the pre- and postoperative periods in 76% (n=133) of the patients.

There is a statistically significant difference between seizure frequency and the type of SDH (P=0.017). More seizures were observed in acute SDH than in other types (Table 2).

Table 1. Demographic features and clinical data of the patients with subdural hematoma

Variables	(n, %)
Gender	
Female	46 (26.3)
Male	129 (73.7)
Etiology	
Spontaneous	73 (41.7)
Trauma	102 (58.3)
Type of SDH	
Acute	49 (28)
Acute-Subacute	33 (18.9)
Subacute	38 (21.7)
Subacute-Chronic	31 (17.7)
Chronic	24 (13.7)
Location	
Left side	79 (45.1)
Right side	72 (41.1)
Bilateral	24 (13.7)
GCS score	
3–8	107 (61.2)
9–12	34 (19.4)
13–15	34 (19.4)
Surgical Technique	
Burr-hole	15 (8.6)
Craniotomy	143 (81.7)
Craniectomy	17 (9.7)
Seizure	
No	133 (76)
Yes	42 (24)
Re-operation	
Yes	21 (12)
No	154 (88)

There is also a statistically significant difference between the GCS score and the development of seizures (P=0.024). A GCS score <12 at admission increases the risk of seizures (Table 3).

No statistically significant difference was found between factors such as etiology, re-operation, hematoma location, and the development of seizures (P>0.05).

Postoperative Period

Emergency protocols were applied in cases of routine status epilepticus. Afterwards, the patients were diagnosed with seizures by taking EEGs by a special neurologist, taking into account their clinical condition. Then, levotracetam (2×500 mg) treatment was started in these cases for the prevention of seizures.

The cases whose treatment was completed due to SDH were called for follow-up at the beginning of the 1st postoperative month. Anticonvulsive treatment was continued for 1 year in cases with seizures.

DISCUSSION

SDH is mostly an emergent condition for neurosurgeons, and the development of seizures in these cases makes management more complex and challenging. It is well known that seizures and status epilepticus are conditions associated with decreased quality of life and increased mortality.[14,15] Seizures are a well-documented complication of SDH, particularly in the early postoperative period.[16,17]

In recent studies, it has been reported that there is significant variability in the incidence of seizures in patients with SDH, and the incidence varies between 0.7 and 18.5%.[18,19] Approximately 2% of the patients had status epilepticus.[20] In some other studies, the incidence of postoperative seizures has been reported in the range of 6–25%.[21] In our study, the incidence of seizures was 24%, which is similar to previous studies.

Table 2. The relationship between the type of subdural hematoma and the presence of seizure

Type of Subdural Hematoma	Seizure		P-value
	No (n=133)	Yes (n=42)	
Acute (n=49)	29 (59.2%)	20 (40.8%)	0.017
Acute-subacute (n=33)	26 (78.8%)	7 (21.2%)	
Subacute (n=38)	32 (84.2%)	6 (15.8%)	
Subacute-Chronic (n=31)	26 (83.9%)	5 (16.1%)	
Chronic (n=24)	21 (87.5%)	3 (12.5%)	

Pearson Chi-square test, P<0.05.

Table 3. The relationship between the GCS score at admission and the presence of seizure

Seizure	GCS score			P-value
	>12 (n=107)	9-12 (n=34)	<9 (n=34)	
Yes (n=42)	18 (16.8%)	13 (30.2%)	10 (29.4%)	0.024
No (n=133)	89 (83.2%)	21 (61.8%)	24 (70.6%)	

Pearson Chi-square test P<0.05.

Some authors have examined the validity of prophylactic antiepileptic drug (AED) treatments in patients with SDH. Level II evidence has been used to support Brain Trauma Foundation guidelines that recommend the administration of prophylactic AEDs for 1 week in severe traumatic brain injury cases.[22] However, no prospective randomized studies have examined the use of prophylactic AED administration in patients with cSDH. Sabo et al. reported that seizures were reduced from 32% to 2.4% in patients treated with prophylactic AEDs, and Grobelny et al. identified AED prophylaxis as the only significant predictor of a lower incidence of postoperative seizures.[8,19] The incidence of seizures in patients with SDH who did not use prophylactic antiepileptics was found to be in the range of 0.7–5.4% in some other studies.[18] Thomas et al., in a prospective study evaluating postoperative morbidity in SDH patients, all of whom received prophylactic levetiracetam in 2019, reported that 5.3% of the patients developed seizures.[23] In another study, seizures were detected in 14–17% of patients with acute SDH using prophylactic AEDs.[24]

The overall incidence of posttraumatic seizures (PTS) in patients with cSDH is generally lower than in patients with aSDH; It ranges from 2% to 42%[19,25] and the incidence of postoperative seizures is reported between 1% and 23%.[6,26] Possibly, the high incidence of PTS between studies may be due to different surgical treatments, the condition of the patients, or the severity of head trauma. In our study, the seizure frequency in acute SDHs was more common than in other types of SDH, and this was statistically significant (P=0.017).

Hirakawa et al.[27] compared 309 patients with cSDH after different surgical treatments and found that the incidence of epileptic seizures was lower in patients treated with burr holes compared to patients who underwent craniotomy and hematoma-membrane resection (capsulotomy).[26,27] In our study, no significant relationship was found between the surgical method and seizures.

It was noted that the risk of seizures increased in some patients with hyperdensity on CT scans, which indicates acute bleeding within the cSDH.[28] Mixed-density cSDH was reported to have a higher risk of seizure.[28] The likely mechanism may be a higher concentration of fibrin degradation products in the hematoma, which can irritate the brain parenchyma and lead to membrane formation.[7,28] Because structural brain damage is itself a risk factor for seizures, patients with associated parenchymal abnormalities may also have an increased risk of seizures.[7] Three independent predictors for ASz/SE were identified: GCS ≤13 at admission, distant stroke, and hematoma recurrence within 14 days.[29,30] In addition, drainage may reduce the remaining hematoma and blood metabolites, resulting in less irritation to the cortex. Moreover, hemoglobin, together with blood compounds and degradation products, is known to be highly epileptogenic due to irritating the cortical surface.[31] A high concentration of fibrin degradation products within the acute SDH may be the cause of more epileptic seizures in patients with acute SDH.

The risk increases even more in patients whose inner membranes are excised.[27] This can be explained by the fact that surgical removal of the membrane can lead to cortical damage. However, Kotwica and Brzeiński objected to this opinion, arguing that a persistent capsule essentially causes seizures.[32] Goertz et al. and Lavergne et al. also stated that the incidence of seizures was higher in patients who had undergone membranectomy.[33,34] Lavergne et al. also emphasized that the risk of seizures is higher in patients with septations in the subdural collection. Mixed hematoma density on cranial CT scans indicates recurrent and/or active bleeding. Hemoglobin degradation products have been shown to be epileptogenic by irritating the cortical surface and contributing to the visceral hematoma membrane and hematoma septations.[35,36]

Another important point to note is that a lower GCS score at admission was found to be a predictor of a worse outcome as well as seizure risk. This may be associated with parenchymal dysfunction, which is a risk factor for seizures.[31] In our study, a GCS score <12 at admission increased the risk of seizure (P=0.024). In a similar study, Goertz et al. noted that patients with a higher preoperative midline shift were more prone to seizures.[33] In our study, we did not find any relationship between midline shift and seizure development.

Elderly patients have been described as having a higher risk of seizures, but this may be associated with greater blood accumulation and later occurrence.[37] Similarly, an increased risk has been noted in patients with alcohol abuse that more

than one factor may lower the seizure threshold.[25] Possible reasons are increased risk of repetitive trauma, associated brain atrophy, and changes in neurotransmitter pathways.[38,39] In our study, however, no significant difference was found in terms of seizures in traumatic or spontaneous SDHs.

Important risk factors for ePTS in patients with ASDH are a preoperative GCS score of <8, a postoperative GCS score of 8 within 24 h, and a craniotomy.[9]

For patients with cSDH, the risk factors for PTS were different from those for aSDH. One prospective study reported that alcoholism is a risk factor for PTS in cSDH.[40] However, since most studies did not distinguish between PTS types, we were only able to identify risk factors for unspecified PTS: specific symptoms such as previous stroke, altered mental status (e.g., disorientation, frontal lobe syndrome), CT findings (Hounsfield unit density, brain atrophy), craniotomy, and mean Glasgow outcome scale score at discharge.[19,27] Furthermore, the incidence of unspecified PTS in left-sided hematoma was slightly higher but did not reach significance.[19]

From a molecular and biological perspective, the pathophysiology of cSDH relies on neuroinflammation and angiogenesis mediated by vascular endothelial growth factor (VEGF) and clot formation associated with increased bleeding and hyperactivation of fibrinolysis.[41] Cytokines such as interleukin-1, -6 and -8 and matrix metalloproteinase are also involved in the development of the outer and inner cSDH membranes. Angiopoietin-2 and VEGF also act as proangiogenic factors by sprouting leaky capillaries in cSDH membranes.[41] In animal models, neuroinflammation associated with the release of proinflammatory cytokines and other mediators, including cyclooxygenase and prostaglandin E synthase enzymes, has been shown to play a role in neurological disorders, including seizures and epilepsy.[42] In addition, They may cause the accumulation of reactive hemoglobin degradation products and free iron due to the degradation of compounds.[42] Patients with mixed-density hematomas were more prone to seizures than patients with low-isodense hematomas. A pathophysiological explanation might be that blood degradation products can lead to fibrinogen degradation products that contribute to membrane formation as well as irritation of the brain parenchyma. On the other hand, high activation of tissue plasminogen activators may lead to more re-bleeding and thus a greater hematoma mass effect.[28,43]

Seizure medications have significant side effects. Phenytoin, the primary seizure drug studied, may cause some degree of cognitive impairment, multiple drug interactions, and rarely Stevens-Johnson syndrome.[44] In addition, phenytoin was associated with a more frequent fever, more time requiring ventilatory support, and worse patient outcomes in patients with intracerebral hemorrhage and subarachnoid hemorrhage.[44,45] Levetiracetam has been shown to be better tolerated than phenytoin and has a simpler dosing regimen, which has led to its widespread adoption.[46] However, no studies have yet been conducted to examine the effect of levetiracetam on global outcome measures in SDH. Given these potential harms, the risk-benefit profile of these drugs should be carefully considered in patients with preserved GCS. Meanwhile, because phenytoin has significantly more side effects such as hepatic cytochrome P450 induction, cutaneous reactions, cardiac complications, thrombocytopenia, and drug interactions, it has mostly been replaced by newer-generation AEDs such as levetiracetam.[47,48] Comparing levetiracetam and phenytoin, levetiracetam appeared to have similar efficacy in reducing PTS, with significantly lower complication rates (excluding gastrointestinal problems) and better long-term outcomes.[24,46]

In the available literature, there is conflicting evidence for the use of AED prophylaxis, as demonstrated by a Cochrane analysis.[6] Since prospective, randomized studies are lacking,[6] a formal recommendation cannot be made yet. However, there are an increasing number of studies in the recent literature that advise against routine AED prophylaxis for patients with cSDH. For example, Lavergne et al.[34] could not demonstrate the beneficial effect of routine AED administration on seizure prophylaxis in patients with cSDH. Grobelny et al.[8] reported a lower seizure rate in patients with AED prophylaxis; however, this regimen did not translate into an improved clinical outcome. Based on the low incidence of seizures after BHC and the potential side effects of AEDs, Flores et al.[49] do not recommend routine application of AEDs. For the same reasons, Branco et al.[37] recommended that AEDs be used only in high-risk patients, such as alcohol dependents and elderly patients.

The timing of prophylactic AEDs is not specifically specified in most studies with respect to pre-or postoperative initiation. Grobelny et al. noted that patients in whom AEDs were started preoperatively had a significantly lower risk of seizures compared to those given postoperatively.[8] Ohno et al. reported that AEDs should begin 30 min before surgery; Battaglia et al. suggested starting within 24 h of admission without mentioning the timing of the surgery. Rubin and Rappaport reported the onset of AEDs during surgery.[26,50]

There are some limitations to this study. This is a retrospective study, and small sample sizes are a major drawback. Also, EEG was not performed consistently in all patients, and very few had continuous EEG monitoring.

CONCLUSION

Seizures are more frequent in patients with acute SDH. Patients with a GCS score <12 at admission had a higher risk of seizures. Focal seizures after SDH remain the leading cause of postoperative morbidity. Given the prevalence and risk of postoperative seizures, it is crucial to identify seizures appropriately and treat them aggressively when clinical suspicion is high. These patients in particular may benefit from prophy-

lactic AED therapy; however, a prospective, randomized trial will be required to clarify which subgroups of these patients would benefit from AED prophylaxis and to develop a risk assessment score for postoperative seizures.

Ethics Committee Approval: This study was approved by the Gülhane Education and Research Hospital Research Ethics Committee (Date: 25.01.2023, Decision No: 2022/165).

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ORİJİNAL ÇALIŞMA - *ÖZ*

Subdural hematomlarda profilaktik antikonvülsif tedavi gerekli midir?

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AMAÇ: Subdural hematom (SDH) beyin cerrahisinde genellikle acil bir klinik durumdur. SDH ve epilepsi arasındaki ilişki iyi kurulmamıştır. Bu nedenle SDH'li hastalarda antikonvülzif tedavinin kullanımı tartışmalıdır. Bu çalışmanın amacı, SDH nedeniyle ameliyat edilen hastalarda nöbet varlığını analiz etmektir.

GEREÇ VE YÖNTEM: 2016-2021 yılları arasında kliniğimizde SDH nedeniyle ameliyat edilen hastalar geriye dönük olarak incelendi. Hastaların demografik özellikleri, başvuru anındaki Glasgow koma skalası (GKS), SDH'nin tipi, yerleşimi, etiyolojisi, cerrahi girişimin tipi, nöbet varlığı ve reoperasyon gibi faktörler değerlendirildi.

BULGULAR: SDH nedeniyle ameliyat edilen 175 hasta incelendi. Nöbet sıklığı ile SDH tipi arasında istatistiksel olarak anlamlı fark saptanmıştı. Akut SDH'de diğerlerine göre daha fazla nöbet gözlendi. GKS skoru ile nöbet sıklığı arasında da istatistiksel olarak anlamlı bir fark vardır. Başvuru anında GKS skoru 12'nin altında olan hastalar, 12 veya daha yüksek olan hastalara göre daha sık nöbet geçirmişlerdi. Etiyoloji, reoperasyon, hematom yerleşimi ve nöbet gelişimi gibi faktörler arasında istatistiksel olarak anlamlı fark bulunmadı.

SONUÇ: Akut SDH ve başvuru anında GKS skoru düşük olan hastalarda antikonvülsif tedavi önerilebilir. SDH'li hastalarda en uygun antikonvülzif ajanın belirlenmesi için daha geniş serili ileri çalışmalara ihtiyaç vardır.

Anahtar sözcükler: Epilepsi; subdural hematom; cerrahi; antikonvülzif tedavi.

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