Growth Potential of Subdural Hematomas Under Clinical Observation: Which Subdural Hematomas Tend to Grow and Why They Do

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OBJECTIVE: **To study the prognoses of patients with subdural hematoma (SDH) who were not operated on at the time of the first diagnosis and the causes of enlarged hematomas in some patients during the follow-up period.**

MATERIALS AND METHODS: **The records, service files, and radiologic examination results of the patients with diagnoses of SDH were reviewed. The SDH patients were recorded under 5 different categories: acute SDH (ASDH), subacute SDH (SSDH), chronic SDH (CSDH), acute component with chronic SDH (A-CSDH), and subacute component with chronic SDH (S-CSDH). The symptoms, clinical findings, and progression in the patients were correlated with radiologic examinations.**

RESULTS: **A total of 291 patients received diagnoses of SDHs: 80 patients with acute, 29 patients with subacute, and 163 patients with chronic hematoma. Thirty-five patients had diagnoses of SDH with a combination of different components. It was determined that in the follow-up period, patients with A-CSDH showed the greatest increase in hematoma size over time and required surgical intervention the most often.**

CONCLUSION: **SDHs reveal different prognoses in different age groups. Multicomponent SDHs are within the group that shows the greatest increase in size in the follow-up period. SDHs and CSDHs cause recurrent hemorrhages by sustaining the tension on the bridging veins. The greater the hematoma volume, the greater the growth potential of the hematoma tends to be. CSDHs that do not manifest changes in volume for a long time can be monitored without surgical intervention as long as the clinical picture remains stable.**

INTRODUCTION

Subdural hematomas (SDHs) are among the most common intracranial hemorrhages. Symptomatic or enlarged SDHs are treated surgically. Trauma plays the most common role in the etiology of acute SDHs, whereas in other forms, anticoagulant and antiaggregant drug use, cerebral atrophy, and advanced age are the most frequently identified etiologic factors.[1-5] These etiologic factors are also the factors that play a role in SDH recurrence in patients undergoing surgical intervention.[1-3] It is argued that the surgical techniques applied also play a role in recurrence. Along with the modified surgical techniques, it is still under debate which technique is more efficient and leads to fewer complications.[6,7]

Clinical and radiologic processes in SDHs other than ASDHs are generally followed up by a similar approach because they show a more prolonged course than ASDH. Moreover, similar surgical techniques are applied to the treatment of these patients. Although the symptoms and neurologic findings of the patients are similar, there is no consensus on how and how often the patients will be checked on clinically and radiologically during follow-up. It may not be possible to foresee which patients will show what kind of prognosis in what amount of time. Although numerous studies have described the risk factors for recurrence and the surgical treatment of these patients, there are not many

Key words
- Chronic subdural hematoma
- Multicomponent subdural hematoma
- Subdural hematoma
- Subdural hematoma follow-up
- Subdural hematoma risk factors

Abbreviations and Acronyms
A-CSDH: Acute component with chronic subdural hematoma
ASDH: Acute subdural hematoma
CSDH: Chronic subdural hematoma
GCS: Glasgow Coma Scale
ICP: Intracranial pressure
S-CSDH: Subacute component with chronic subdural hematoma
SDH: Subdural hematoma
SSDH: Subacute subdural hematoma

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Table 1. Primary Symptoms and Neurologic Findings at Time of Diagnosis

Admission Symptoms	Headache	Dizziness	Neurologic Deficit	Epilepsy	Decrease in GCS
CSDH	71	84	6	1	1
SSDH	9	11	3	1	5
A-CSDH	8	3	2	0	7
S-CSDH	6	7	1	1	0
ASDH	X	X	X	X	80

GCS, Glasgow Coma Scale; CSDH, chronic subdural hematoma; SSDH, subacute subdural hematoma; A-CSDH, acute component with chronic subdural hematoma; S-CSDH, subacute component with chronic subdural hematoma; ASDH, acute subdural hematoma.

studies on the follow-up period and the prognosis prediction for patients not operated on other than ASDH patients.

MATERIALS AND METHODS

The records of the patients who were being followed up after they had received diagnoses of SDH were retrospectively reviewed. The computed tomographic and magnetic resonance imaging examinations of all patients were obtained by scanning the picture archiving communication systems, and their radiologic findings were recorded. According to their radiologic findings, they were classified as having acute SDH (ASDH), subacute SDH (SSDH), chronic SDH (CSDH), acute component with chronic SDH (A-CSDH), or subacute component with chronic SDH (S-CSDH). All ASDH patients were examined based on coma symptoms. The SSDH and CSDH patients and their symptoms were recorded.

Radiologic examinations were evaluated by an independent and experienced neuroradiologist who was not informed about the clinical findings and the follow-up of the subjects. The obtained radiologic examination data and measurements were evaluated with the results of the obtained reports.

The admission symptoms and neurologic examination findings depending on the age groups are shown in Table 1. The distribution of the patients by age is shown in Table 2. In patients with ASDH, radiologic findings and coma assessments were considered independently of symptoms.

Ethical Approval

Ethics Committee Approval was obtained from Ahi Evran University Clinical Research Ethics Committee on August 8, 2017: no: 2017—13/137.

Statistical Analysis

For statistical analysis, SPSS (version 20.0 for Windows, SPSS Inc., Chicago, Ill, USA) was used. Comparison analyses of the 2 groups were performed with 1-way analysis of variance. Statistical significance was assumed if $P < 0.05$.

RESULTS

Of a total of 291 patients with diagnoses of SDH, 80, 163, 29, and 35 patients had diagnoses of ASDH, CSDH, SSDH, and double component SDH, respectively. Surgical intervention was performed in 82 patients under follow-up because of SDH. Sixteen patients underwent repeated operated because of recurrence.

Surgically, all patients were operated on by applying the methods of a craniotomy or 2 twisted burr-hole drainage. Surgical techniques and their results were not discussed. The growth potential of hematomas in patients in the preoperative period was assessed. A total of 225 patients were followed up without surgical intervention.

Regarding the variation of hematoma size in the follow-up radiologic examinations in patients with diagnoses of ASDHs,

Table 2. Patients with Increasing Hematoma Volumes According to Age Group

	Age (years)_			
Hematoma Type	0—16	16—50	50—60	>60
CSDH (n = 41/163)	0/1	2/3	2/16	37/143
SSDH (n = 13/29)	0/1	0/2	2/7	11/19
A-CSDH (n = 15/20)	0	0/1	4/5	11/14
S-CSDH (n = 10/15)	0	1/2	2/4	7/9
ASDH (n = 13/80)	1/25	9/48	1/4	2/3

CSDH, chronic subdural hematoma; SSDH, subacute subdural hematoma; A-CSDH, acute component with chronic subdural hematoma; S-CSDH, subacute component with chronic subdural hematoma; ASDH, acute subdural hematoma.

Table 3. Comparison of Hematoma Widths at Time of Diagnosis with Those in Control Radiologic Examinations

	Width			
Hematoma Type	**0–5 mm**	**5–7 mm**	**7–10 mm**	**>10 mm**
CSDH (n = 42/163)	Increase: 23 Same: 61 (n = 84)	Increase: 5 Same: 26 (n = 31)	Increase: 10 same: 29 (n = 39)	Increase: 4 same: 5 (n = 9)
SSDH (n = 13/29)	Increase: 2 Decrease: 8 (n = 10)	Increase: 6 Decrease: 6 (n = 12)	Increase: 5 Same: 2 (n = 7)	X (n = 0)
A-CSDH (n = 15/20)	Increase: 1 Decrease: 2 (n = 3)	Increase: 5 Decrease: 1 (n = 6)	Increase: 8 Decrease: 2 (n = 10)	Increase: 1 (n = 1)
S-CSDH (n = 10/15)	Increase: 2 Same: 1 (n = 3)	Increase: 1 Same: 2 (n = 3)	Increase: 6 Same: 2 (n = 8)	Increase: 1 (n= 1)
ASDH (n = 13/80)	Increase: 9 Decrease: 47 (n = 56)	Increase: 2 Decrease: 15 (n = 17)	Increase: 2 Decrease: 5 (n = 7)	X (n = 0)

CSDH, chronic subdural hematoma; SSDH, subacute subdural hematoma; A-CSDH, acute component with chronic subdural hematoma; S-CSDH, subacute component with chronic subdural hematoma; ASDH, acute subdural hematoma.

no etiologic correlations associated with age or the anatomic structure of the brain could be found. The rate of antiaggregant and/or anticoagulant use in non-ASDH patients was found to be 57%. The rate of hematoma growth was found to be 63.17% in the follow-up recordss of these patients.

Among SDH, CSDH, and bicomponent SDH patients, by contrast, the bicomponent hematomas tended to grow at higher rates (Table 3). The CSDH cases with an acute component (Figures 1 and 2) showed expansion most frequently ($P < 0.05$), and patients in this group were operated on most often ($P < 0.05$). Of these patients, those with 1-component CSDHs were determined to have the least expansion potential ($P < 0.05$) and to be operated on the least frequently ($P < 0.05$).

In patients with a hematoma thickness less than 5 mm, no significant difference was found between the amounts of increase for different hematoma types ($P > 0.05$). For hematomas thicker than 5 mm, A-CSDHs had the highest growth potential ($P < 0.05$).

In the same way, it was found that the hematoma volume tended to increase more in elderly patients in whom cerebral atrophy was detected more frequently and in those with greater hematoma volume, compared with the other groups (Figure 3). The results reflecting increased hematoma size in the follow-up period of patients with nonacute SDH with septation within the hematoma in their radiologic examinations and antiaggregant/anticoagulant use are shown in Table 4. The average age of the patients operated on after receiving diagnoses of SSDH was significantly lower than that of the patients not operated on. It was concluded that in comparison with CSDH patients, symptoms and neurologic findings appeared earlier in these

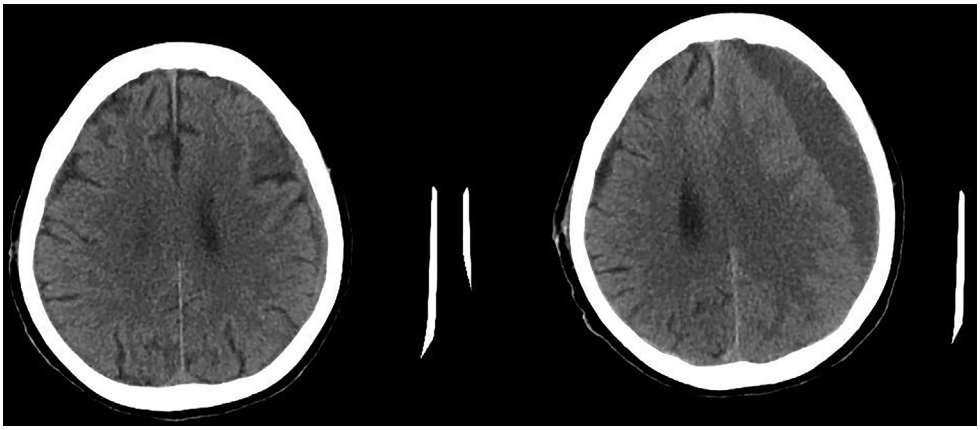

Figure 1. Chronic subdural hematoma with a left frontotemporal localization and a partially acute component had an increase in width during the follow-up period, transforming into the form of a large chronic subdural hematoma.

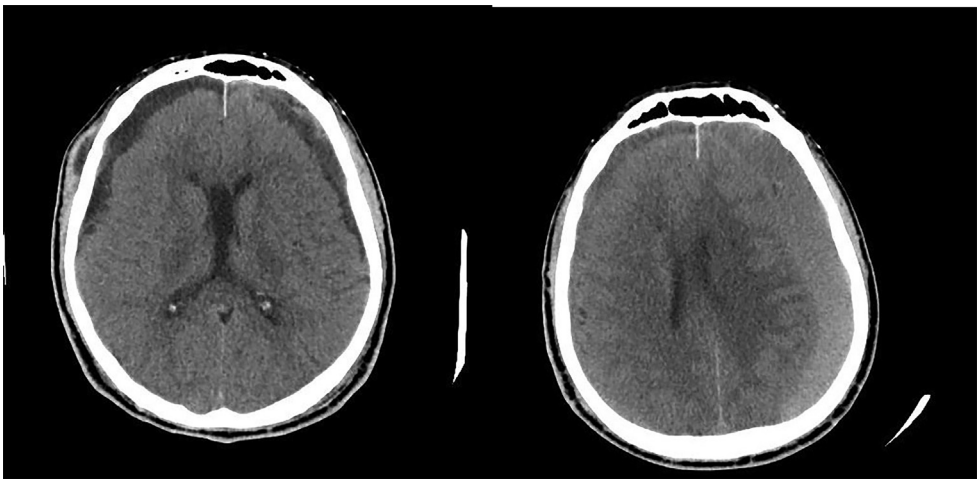

Figure 2. Subdural hematoma with bifrontal localization; the hematoma with left frontal localization and acute component widened during the follow-up period, caused a midline shift, and transformed into the subacute form. Partial decrease in the width of the hematoma with right frontal localization and transition to subacute form were observed.

patients, age-related cerebral atrophy was relatively less, and therefore, tolerance to increase in intracranial pressure was lower.

The comparison of hematoma widths at first diagnosis and radiologic follow-up is shown in Table 5. The results of the first and last radiologic examinations of patients not operated on were taken into consideration. The average radiologic follow-up period for patients other than those with ASDH was 23 days.

DISCUSSION

The risk factors for SDHs with the tendency to grow were described as a result of the assessment of 291 different patients who were being followed up and did not undergo surgical intervention during diagnosis. It was brought into view that the most prominent differences in patients other than those with ASDH depended on age and anticoagulant/antiaggregant drug use.

In the literature, fibrinolytic activity has also been defined as a risk factor for CSDH patients who were being followed up and whose CSDHs tended to grow. It has been stated that the hematoma volume increases as membranous and vascular structures are affected by fibrinolysis.[8,9] However, fibrinolysis is a process that occurs in hematomas in every part of the body and is not specific to the SDH. Hematomas tend to get smaller

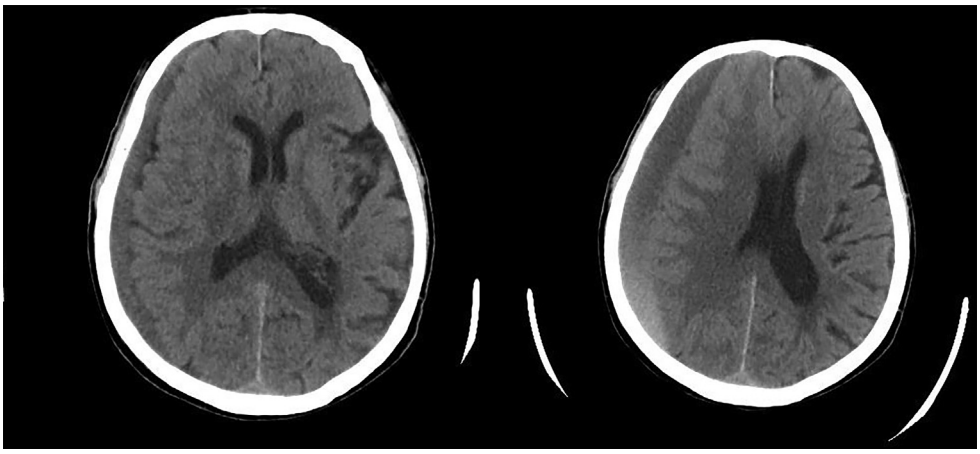

Figure 3. Increase in hematoma width was seen more frequently in patients with cerebral parenchyma atrophy and with hematoma widths thicker than 5 mm. Increase in the width of the subacute-chronic hematoma with a right frontoparietal localization can be observed.

Table 4. SDH Increase, Surgery, and Recurrence Rates in Patients Other Than ASDH

Factor	Increase in Hematoma Dimension	Operated On	Recurrence Rate	Recurrence Surgery
Patients using antiaggregant/anticoagulant (n = 148)	77 (52%)	48 (32%)	15 (10%)	11 (7%)
The presence of septation in hematoma (n = 78)	31 (40%)	Burr-hole: 12 Craniotomy: 8	Burr-hole: 6 Craniotomy: 2	Burr-hole: 3 Craniotomy: 1

SDH, subdural hematoma; ASDH, acute subdural hematoma.

and to be resorbed rather than grow by fibrinolytic activity. For this reason, in the region where fibrinolysis takes place, decreases in the coagulation factors and increases in the partial thromboplastin time and activated partial thromboplastin time values, as well as d-Dimer values, are expected, and these results reflect the natural process of fibrinolysis. Chronic SDHs are hematomas diagnosed at about the third week, and the presence of thrombocytes within the hematoma, which have a lifespan of 7 to 10 days, suggests the emergence of a new hemorrhage.

It was shown that particularly in elderly patients with advanced cerebral parenchymal atrophy, chronic SDHs were resorbed later than those in younger patients, and that volume increase was most common in this age group (Table 2). The greater the hematoma volume, the greater the tension in the bridging veins will be. Therefore, the threshold value for the rebleeding of the bridging veins before the hematoma of a large volume is resorbed can easily be exceeded; with such recurrent minor hemorrhages, the hematoma volume increases more easily, and the resorption process further extends. In elderly patients with cerebral atrophy, the presence of long-standing SDHs results from stretching of the veins and mechanical impact. In patients with SDHs undergoing surgical intervention, the internal membrane not being opened, the subdural area not being irrigated, the presence of postoperative pneumocephalus, remaining residual hematoma, and factors that prevent the parenchyma from being expanded enough were factors held responsible for the cause of repeated hemorrhage in these patients. These are all mechanical factors leading to mechanical stretching of the bridging veins.[5]

The presence of septation in the hematoma is also thought to prevent or slow down the resorption of the hematoma. Septations are among the signs that suggest the emergence of rehemorrhage within the hematoma. Septation within the hematoma was detected in 23 patients, and it was set forth that the hematoma was not wholly resorbed in the patients and that 16 patients needed surgical intervention.

Besides ASDHs, the SDHs having the fastest and greatest tendency to grow were bicomponent SDHs (Table 3). In these patients, the tolerance of the cerebral parenchyma decreases with the newly developing component, and neurologic deterioration occurs in an earlier period. Bleeding occurring in a later period further increases the tension in the bridging veins and reduces the tolerance of the cerebral parenchyma in the early period, causing symptoms and neurologic manifestations to appear more rapidly.

Among medications intended for the treatment of symptoms and neurologic deficits, the use of drugs such as mannitol that reduce the volume of parenchyma should be considered contraindicated in patients with nonacute SDHs undergoing follow-up care. Such medications further increase the tension in the bridging veins by decreasing the parenchymal volume, causing them to break. Therefore, they also increase the volume of the SDH. Studies are showing that corticosteroids decrease the volume of SDHs during follow-up.[10-12] Even though corticosteroids are not effective in hemorrhagic edema, some information in the literature suggests that they provide membrane stabilization in SDHs.[13,14] However, medical treatment should not be considered sufficient for symptomatic SDHs; surgical techniques that will not cause a compression effect on the cerebral parenchyma should be considered the most effective treatment method, foreseeing that rapid and effective results will be obtained.

Table 5. Hematoma Types and Age Averages in Patients Both Operated On and Not operated On During Follow-Up Period

Hematoma Type	Operated On During Follow-up	Not Operated On During Follow-up	Average Age of Patients Operated On	Average Age of Patients Not Operated On
CSDH (n = 163)	33 (20%)	130 (80%)	75.7	72.3
SSDH (n = 29)	15 (52%)	14 (48%)	60.2	67.5
A-CSDH (n = 20)	13 (65%)	7 (35%)	68.5	78.1
S-CSDH (n = 15)	7 (47%)	8 (53%)	75.2	60.2
ASDH (n = 80)	14 (18%)	66 (82%)	34.9	23.7

CSDH, chronic subdural hematoma; SSDH, subacute subdural hematoma; A-CSDH, acute component with chronic subdural hematoma; S-CSDH, subacute component chronic subdural hematoma; ASDH, acute subdural hematoma.

CONCLUSION

In the follow-up of SDHs other than ASDHs, bicomponent hematomas tend to grow at higher rates. Age and associated cerebral atrophy, the use of antiaggregant/anticoagulants, large hematoma width, and the presence of septation in the hematoma were defined as the etiologic factors for an increase in hematoma volume. The presence of 1 or more of these risk factors may change the risk coefficient depending on the patient. The simultaneous presence of several identified risk factors should suggest the necessity of a shorter radiologic follow-up time, and the follow-up/treatment protocol should be determined accordingly. In patients with SDH who are being followed up, medication for reducing intracranial pressure should be considered contraindicated because it may increase SDH volume by decreasing the parenchyma volume.

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Conflict of interest statement: The author declares (or authors declare) that the article content was composed in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest. (see p. 19 for variations)

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