Annals of Medical Research
DOI: 10.5455/annalsmedres.2019.07.399 2019;26(10):2240-4

Original Article

Natural course of non-surgical acute traumatic subdural hematomas: Retrospective analysis of 92 patients

Tayfun Cakir[1], Dursun Turkoz[2]

[1]Erzincan Binali Yıldırım University, Mengücek Gazi Education and Research Hospital, Department of Neurosurgery, Erzincan, Turkey
[2]Health Sciences University Samsun Education and Research Hospital, Department of Neurosurgery, Samsun, Turkey

Abstract

Aim: In the present study, we aimed todiscuss the natural course of acute subdural hematoma (ASDH) patients who were not operated according to the admisssion findings.

Material and Methods: In this study patients who admittedwith a diagnosis of traumatic ASDH and underwent conservative treatment according to the admission findings at the neurosurgery clinic of Adıyaman University between 2013 and April 2019 were identified. Patients who had a diagnosis of ASDH less than 10 mm, a midline shift of less than 5 mm and had a repeat computed tomography (CT) scan were included in this study.Two groups were formed with and without increase in the hematoma size according to thecontrol CT. Age, gender, anticoagulation status, comorbidities of the patients, type of trauma, size of the hematoma, GCS scores and presence or absence of a "low-density band" on CT were evaluated.

Results: We identified 92 patients who had coded as "traumatic ASDH" and managed nonoperatively according to the findings of the initial CT.32.6% of patients had hematoma growth according to the control CT and68.4% had either decreased or the same size.According to the statistical analysis results, anticoagulant drug use(p=0.001) and comorbidity status(p=0.03) were found to be related with the increase of the hematoma size. Also there was no statistically significant difference (p=0.07) between the two groups about the low density band but it was seen more differencein the non-increased group.

Conclusion: Conservative treatment is one of the treatment methods of traumatic acute subdural hematomas. Patients who have under 10 mm hematoma size and 5mm midline shift can be followed up by non-surgical methods. But increased headache in these patients may be a sign that should be considered in terms of increased hematoma. Furthermore, the use of anti-coagulant drugs and the presence of comorbidity make patients more risky.

Keywords: Acute subdural hematoma; trauma; low density band; anticoagulation; comorbidity.

INTRODUCTION

Acute subdural hematoma (ASDH) is one of the devastating complication of severe traumatic brain injury with a frequency of 12%-29% (1,2). Although treatment of ASDH has been generally known as surgical treatment, conservative approach may be preferred when certain criteria are met.Many previous studies suggested that patients lower than 10 mm in initial thickness might be approached conservatively (3,4). However, in conservative follow-up, many conditions can be encountered. Although most resolve spontaneously, a few do not disappear and progress to require surgical treatment (5). It is very difficult to predict which patients will have an increase in the size of the hematoma. Reason for this study arose from a recurring clinical question. How can we predict whether the lesion will exacerbate when a patient with traumatic ASDH who was not initially operated?There is no consensus on how and how often the patients will be checked on clinically and radiologically during follow-up.It was stated in the previous studies that the hematomaswith a thickness more than 10 mm or a midline shift more than 5 mm should be surgically evacuated irregardless of the GCSscore (4,6). But to the best of our knowledge, there is no clear information about the criteria for selecting conservative management in the literature. Of course, the knowledge on the natural course and outcomes of initially non-operated ASDH might help neurosurgeons predict prognosis and aid the decision-making process.Thus our primary aim was to identify the predictive factors of the increase in the size of ASDH.

Received: 19.07.2019 **Accepted:** 05.09.2019 **Available online:** 21.10.2019
Corresponding Author: Tayfun Cakir, Erzincan Binali Yıldırım University, Mengücek Gazi Education and Research Hospital, Department of Neurosurgery, Erzincan, Turkey **E-mail:** tayfuncakir198375@gmail.com

MATERIAL and METHODS

We retrospectively collected data from the patients diagnosed with ASDH who were treated at the neurosurgery department of the ErzincanBinali Yildirim UniversityMedical School Hospital between 2013 and April 2019.This study was approved by local ethics committee (Erzincan Binali Yildirim University Medical School, Local Ethics Committee; session number: 2019/4-8). Patients who had a diagnosis of ASDH less than 10 mm and a midline shift of less than 5 mmand had a repeat CT scan were included in this study. Age, gender, anticoagulation status, comorbidities of the patients,type of trauma, size of the hematoma, GCS scores and presence or absence of a "low-density band" on CT were evaluated.

Measurement of the hematoma size and and presence or absence of a "low-density band" were performed by the first author with the help of a radiologist who was blinded for this study's hypothesis.Comorbidities as follows; hypertension, atrial fibrillation, diabetes mellitus, respiratory insufficiency, renal disease and infection were defined. Resolutionof the hematoma was defined as a decrease in axial thicknessby at least 50% and growth was similarly definedas an increase in thickness at least 50%. Then we compared the patients who experienced resolution of their hematoma(group1) with those who did notexperience resolution of their hematoma(group2).

Statistical analyses were performed using a t-test and a Pearson's chi-squared test. Statistical significiance was defined as a p value of less than 0.05. Descriptive statistics were reported for all variables as means and standard deviations for numerical variables, andas percentages for categorical data.

RESULTS

Patient Characteristics

We identified 92 patients who had coded as "traumatic ASDH" and managed nonoperatively according to the findings of the initial CT.The characteristics and findings of the patients are shown in the table. Although there were some deficient values, the mean time from injury to initial CT was <6 h for most patients of both groups (93% and 97%, respectively) and second CT time was 9±1.7 hours on average for all patients.

In the group 1 there were 30 patients (32.6%) who had an increase in the size of the hematoma at the first control CT and in the group 2 there were 62 patients (68.4%) who had the same size or resolution at their hematoma.

The mean age of the group 1 was 58.7±1.1 years and 69% of this group was male, the mean age of the group 2 was 51.2±2.4 years and 61% was male (p=0.121, p=0.240, respectively).

The most three common mechanism of injury was ground level fall (group 1, 21.3%; group 2 24.2%), falling over 2 meters (20.9% versus 22.9%) and motor vehicle accident (20.7 % versus 16.5%). Falling over 2 meters and motor vehicle accidents were thought as high impact traumas and analyzed statistically between the groups. No significiant difference between the two groups about the high impact mechanism of the injury was found (p=0.11).

Clinical Features

In the group 1 the mean admission GCS was 12.9±1.1and in the group 2 it was 13.6±0.13. There was no significant difference in the admission GCS scores of the patients between the groups(p=0.40). In the first group, 3 patients who needed surgery regressed consciousness within the hours. And they were discharged with healing after surgical intervention. In the second group, there was no GCS score regression.

In the first group, 22 of the patients had worsening headache complaints and 7 patients complained of nausea and vomiting. In the second group, 6 patients had worsening headache complaints. 19 patients had nause and vomiting complaints. Worsening headache was statistically higher in the first group (p=0.002) and there was no significant difference between the two groups in terms of desire for nause and vomiting (p=0.19).

Radiological Findings

In the group 1 the hematom size at the initial CT was 8.7±1.9 mm and in the group 2 it was 7.4±2.8mm (p=0.101). In the group 1, 3 patients (10%) had newly formed midline shift at the control CT obtained in 1-6 hours (2 of these patients underwent control CT while they were still in the emergency department) and these 3 patients were urgently operated.In the second group, no patient required surgery(Figure 1).

Low density band (Figure 2A-B).Which was seen between the hematoma and the skull was found in 17% of the patients in the first group. This rate was 42% in the second group. But there was no statistically significant difference (p=0.07) between the two groups about the low density band.

Anticoagulant and Comorbidity Status

43.3% of the patients in the first group had anticoagulant or antiplatelet drug use. 54% of these patients had been usingvitamin K antagonist, 23% oral anticoagulant drug and 23% trombocyte inhibitor. 3 patients who had undergone surgery according to the control CT findingswere usingontrombocyte inhibitor.These drugs were discontinued after admission to the emergency department.All operated patients receivedenoxaparin subcutaneously 10-12 hours afterthe operation, and afterward at least 40-60 mg enoxaparinwas administered daily for thromboembolism prophylaxis.Patients who underwent conservative treatment received 40-60 mg enoxaparin once a day regularly. In the second group, the rate of anticoagulant drug use was 17.7%.There was a statistically significant difference between the two groups(p=0.001).In the first group, 78.4% of the patients had one or more comorbities, whereas this rate was 58.6% in the second group. There was a statistically significant difference between the two groups (p=0.03) about the presence of a comorbidity.

Table. Comparison of the groups' characteristics

Variable	Group 1 (n=30)	Group 2 (n=62)	P value
Age	58.7±1.1	51.2±2.4	0.121
Gender (% male)	69	61	0.240
Hematoma size at the initial CT (mm)	8.7±1.9	7.4±2.8	0.101
Mechanism (%high impact)	41,6	39,4	0.11
Worsening headache(%)	73.3	9.6	0.002
Nausea and vomiting (%)	23.3	30.6	0.19
Admission GCS score	12.9±1.1	13.6±0.13	0.40
Comorbidities (%)	77.0 (n=23)	58.6 (n=36)	0.03
Hypertension (%)	72.1 (n=16)	50.0 (n=18)	<0.0001
Atrial Fibrillation (%)	56.5 (n=13)	16.6 (n=6)	<0.0001
DM Type 2 (%)	43.4 (n=10)	13.8 (n=5)	<0.0001
Respiratory Insufficiency (%)	36.5 (n=7)	11.1 (n=4)	0.06
Renal Disease (%)	8.6 (n=2)	5.5 (n=2)	0.30
Infection (%)	10.0 (n=3)	5.5 (n=2)	0.08
Pre admission blood thinner use (%)	43.3 (n=13)	17.7 (n=11)	0.001
Low density band (%)	17	42	0.07

DISCUSSION

Conservative treatment of traumatic ASDH has been conceivable for many patients with thin hematom, no mass effect and mild neurologic deficit (2). Most published datas indicate that hematoma with thickness greater than 10 mm, or a midline shifting greater than 5 mm are suggested as critical indicators for surgical treatment (4,6-8). As known the purpose of surgical treatment is to release from herniation and to reduce secondary ischemic injury minimally. Thus, ASDH causing significant mass effect, which may be effectively reduced by surgery, is supposed to be an indication for surgery. But in the cases of ASDHs with thickness lower than 10 mm, or a midline shifting lower than 5 mm conservative treatment may be decided. According to the previous studies the criteria used to select patients for conservative treatment were clinical stability or improvement during the time from injury to evaluation at the hospital (9-12). However, the natural course of ASDH is different among patients therefore it is very difficult to manage these patients. Because hematoma may increase so these non-operated patients may worsen within the first minutes or following days (13,14).

32% of 92 patients with whom we decided conservative treatment acccording to the admission findings had increased hematoma and 3 of these patients (10%) had newly formed midline shift at the control CT and they were urgently operated. According to our results this increasewas associated with 3 parameters, worsening of headache, comorbidite of patients and using prehospital pharmaceutical anticoagulation. In addition, in the non-increased group more presence of low density band which has been usually associated with dilution of hematoma was noted.In previous studies, the rate of delayed surgery requirement in ASDH patients ranged from 6-16% (4,15). Also in the previos studies progression of the hematom size and mass effect with the symptoms of development of intracranial hypertension was reported as the most common cause and seizure comes in the second place with a rate of 2-3%. In our cases no antiepileptic drugs were used and no seizures were seen. In all cases of our present study, the reason for delayed surgery was progression of hematoma size with accompanying symptoms. We usually take the first control CT within the first 6 hours; in some cases with the worsening of headache, this time could be taken earlier and sometimes when the patient was still in the emergency room. Of course headache is a main complaint of ASDH patients. Yamada et al. (16) argued that the increase in headache complaints was related with increase of the hematoma especially with presence of midline shift. The fact that we paid attention to worsening of headache. Also it is evident from his study that Wong had used severe headache as a sign to take a control CT in patients managed with conservative treatment (15). As known anticoagulants reduce the risk of thromboembolic complications following many cardiovascular diseases. In our study, the rate of anticoagulant drug use was found to be 26%. In previous studies, the rate of ASDH patients who were receiving oral anticoagulant therapy was

reported to be between 10% and 24% and recent studies reported a further increase up to about 60% (15,17-19). It was reported by Won et al. (15) that use of anticoagulant drugs in 116 patients increased bleeding and increased mortality.And Lieberman et al. (20) demonstrated that 13(1.1%) of 1187 patients with anticoagulation suffered from serious intracranial hemorrhages. A similar finding was also reported by Forfar that 0.4% of 501 patients under anticoagulant therapy suffered from intracranial hemorrhages (21). Also Kawamata et al. (19) stated that that need for surgery was even higher in these patients. In accordance with previous studies, anticoagulant drug use was found to be associated with increasing of the ASDH in our study.It is not effortless to establish a balance between the intensity of anticoagulation and hemorrhagic events. So worsening intracranial hemorrhagies usually may be particularly devastating complication of anticoagulant therapy.

Comorbidity had been found to be an independent factor adversely affecting rebleeding and mortality in most case series as in our present study (3,4). In our study, among the comorbidities hypertension, atrial fibrilasyon, type 2 DM and respiratory insufficiencies were the most common diseases. In most studies, cardiac and respiratory problems were associated with high mortality and increased bleeding (4,19,20). In those studies it was stated that especially cardio-pulmonary diseases reduces systemic physiological reserve capacity as well as tolerance to traumatic stress leading to delayed enargement of subdural hematoma, or delayed worsening of traumatic contusional edema.

Also we observed that the low density band which was seen between bone and hematoma was observed more in the non-increased group although not statistically significant. Previous studies reported that the low density band had been a sign for rapid resolution of ASDH (22,23,24). Moreover, when we examined the literatüre, we saw that the low density band was mentioned in almost every cases with rapid resolution of ASDH presentation. Hadjigeorgiou et al. (25) stated that a low density band showes CSF coming from the subarachnoid space through arachnoid membrane tear.

CONCLUSION

Conservative treatment is one of the treatment methods of traumatic acute subdural hematomas. Patients who have under 10 mm hematoma size and 5mm midline shift can be followed up by non-surgical methods. But increased headache in these patients may be a sign that should be considered in terms of increased hematoma. Furthermore, the use of anti-coagulant drugs and the presence of comorbidity make patients more risky.

Competing interests: The authors declare that they have no competing interest.
Financial Disclosure: There are no financial supports
Ethical approval: This study was approved by the Institutional Ethics Committee and conducted in compliance with the ethical principles according to the Declaration of Helsinki.

Tayfun Çakır ORCID: 0000-0002-9979-9291
Dursun Turkoz ORCID: 0000-0003-3599-0895

REFERENCES

1. Bullock MR, Chesnut R, Ghajar J, et al. Surgical management of acute subdural hematomas. Neurosurgery 2006;58:S16-24.
2. Feliciano CE, De Jesús O. Conservative management outcomes of traumatic acute subdural hematomas. P R Health Sci J 2008;27:220–3.
3. Mathew P, Oluoch-Olunya DL, Condon BR, et al. Acute subdural haematoma in the conscious patient:outcome with initial non-operative management. Acta Neurochir (Wien) 1993;121:100–8.
4. Bajsarowicz P, Prakash I, Lamoureux J, et al. Nonsurgical acute traumatic subdural hematoma: what is the risk?. Journal of neurosurgery 2015;123:1176-83.
5. Vega, RA, Valadka, AB. Natural history of acute subdural hematoma. Neurosurgery Clinics 2015;28:247-55.
6. Servadei F, Nasi MT, Cremonini AM, et al. Importance of a reliable admission Glasgow Coma Scale score for determining the need for evacuation of posttraumatic subdural hematomas: a prospective study of 65 patients. J Trauma 1998;44:868–73.
7. Phan K, Moore, JM, Griessenauer, et al. Craniotomy versus decompressive craniectomy for acute subdural hematoma: Systematic review and meta-analysis. World Neurosurgery 2017;101:677-85.
8. Fountain DM, Kolias AG, Lecky FE. Survival trends after surgery for acute subdural hematoma in adults over a 20-year period. Ann Surg 2017;265,590.
9. Lee JJ, Won Y, Yang T, et al. Risk factors of chronic subdural hematoma progression after conservative management of cases with initially acute subdural hematoma. Korean J Neurotrauma 2015;11:52-7.
10. Öğrenci A, Ekşi MŞ, Koban O, et al. Spontaneous rapid resolution of acute subdural hematoma in children. Child's Nervous System 2015;31:2239-43.
11. Bullock MR, Chesnut R, Ghajar J, et al. Surgical management of acute subdural hematomas. Neurosurgery 2006;58:2-16.
12. Laviv Y, Rappaport ZH. Risk factors for development of significant chronic subdural hematoma following conservative treatment of acute subdural hemorrhage. Br J Neurosurg 2014;28:733–8.
13. Son S, Yoo CJ, Lee SG, et al. Natural course of initially non-operated cases of acute subdural hematoma: the risk factors of hematoma progression. J Korean Neurosurg Soc 2013;54:211.
14. Izumihara A, Orita T, Tsurutani T, et al. Natural course of non-operative cases of acute subdural hematoma:sequential computed tomographic study in the acute and subacute stages. No Shinkei Geka 1997;25:307–14.
15. Wong CW. Criteria for conservative treatment of supratentorial acute subdural haematomas. Acta Neurochir 1995;135:38-43.
16. Yamada SM, Tomita Y, Murakami H, et al. Headache in patients with chronic subdural hematoma: analysis in 1080 patients. Neurosurg Rev 2018;41:549-56.
17. Gaist D, García Rodríguez LA, Hellfritzsch M, et al. Association of antithrombotic drug use with subdural hematoma risk. JAMA 2017;317:836–46.
18. Mulligan P, Raore B, Liu S, et al. Neurological and functional outcomes of subdural hematoma evacuation in patients over 70 years of age. J Neurosci Rural Pract 2013;4:250–6

19. Kawamata T, Takeshita M, Kubo O, et al. Management of intracranial hemorrhage associated with anticoagulant therapy. Surg Neurol 1995;44:438–43.
20. Lieberman A, Hass WK, Pinto R, et al. Intracranial hemorrhage and infarction in anticoagulated patients with prosthetic heart valves. Stroke 1978;918-24.
21. Forfar JC. A 7-year analysis of haemorrhage in patients on long-term anticoagulant treatment. Br Heart J 1979;42:128-32.
22. Fujimoto K, Otsuka T, Yoshizato K, et al. Predictors of rapid spontaneous resolution of acute subdural hematoma. Clin Neurol Neurosurg 2014;118:94-7.
23. Gan Q, Zhao H, Zhang H, et al. Rapid spontaneously resolving acute subdural hematoma. J Craniofac Surg 2017;28,287.
24. Wen L, Liu WG, Ma L, et al. Spontaneous rapid resolution of acute subdural hematoma after head trauma:is it truly rare? Case report and relevant review of the literature. Ir J Med Sci 2009;178:367–71.
25. Hadjigeorgiou G, Chamilos C, Petsanas A, et al. Rapid spontaneous resolution of acute subdural haematoma in a patient with chronic alcoholism. Br J Neurosurg 2012;26:415–6.