ORIGINAL ARTICLE

Neurol Med Chir (Tokyo) 53, 26～33, 2013

Retrospective Analysis of Operative Treatment of a Series of 100 Patients With Subdural Hematoma

Bartosz GODLEWSKI,[1] Agnieszka PAWELCZYK,[2] Tomasz PAWELCZYK,[2] Katarzyna CERANOWICZ,[1] Maciej WOJDYN,[1] and Maciej RADEK[1]

[1]*Department of Neurosurgery and Peripheral Nerves Surgery, WAM University Hospital in Lodz, Medical University of Lodz, Lodz, Poland;*
[2]*Department of Affective and Psychotic Disorders, Medical University of Lodz, Lodz, Poland*

Abstract

This retrospective study of medical records, surgical protocols, patient observation cards, and imaging files of 100 patients treated for subdural hematoma analyzed the type of hematoma, patient age and sex, operative technique, neurological status, cause of injury, duration of hospital stay, mortality rate, and the number of and reasons for reoperations to determine the effects on treatment outcomes. The time between the head injury and onset of neurological symptoms was analyzed versus the type of hematoma determined from computed tomography (CT) scans. Acute hematomas accounted for 38% of the cases, with subacute hematomas representing 20%, and chronic ones accounting for 42%. In trauma patients, the mean time interval between the injury and onset of neurological symptoms was 0.38 days for acute hematomas, 13.8 days for subacute hematomas, and 23.75 days for chronic hematomas. Repeat surgery was carried out in 26% of the cases. Improvement was obtained in 44% of cases, deterioration in 20%, and no change in neurological status in 36%. Timing of the operations was between 15:00 and 23:00 in 45%, between 23:00 and 7:00 in 33%, and between 7:00 and 15:00 in 22%. The classification of hematomas based on CT presentation corresponds to the classification based on the time elapsed between injury and onset of symptoms, and appears to be appropriate and useful in everyday practice. No preceding injury was identified in 31.6% of acute hematomas, 50% of subacute hematomas, and 61.9% of chronic hematomas. Analysis of reoperations indicates that trepanation may be superior to craniotomy as primary surgery for subacute and chronic hematomas. Subdural hematoma surgeries take place at all times of the day, with most carried out outside the usual working hours.

Key words: subdural hematoma, treatment outcome, repeat surgery

Introduction

This study analyzed the operative treatment of subdural hematomas, in particular the type of hematoma, patient age and sex, surgical technique, and neurological status on admission and at the end of follow up. Additional data included trauma-related etiology, duration of hospital stay, mortality rate, and the number of and reasons for repeat surgeries. The number of procedures performed at different times of day was also evaluated. These factors were analyzed to determine their impact on treatment outcomes and identify optimal patient management procedures in regard to these factors.

Received October 24, 2011; Accepted March 14, 2012

Material and Methods

This retrospective study analyzed a series of 100 consecutive patients with subdural hematomas who were treated operatively. The analysis involved a thorough review of medical records, surgical protocols, patient observation cards, and imaging files. Hematomas were classified as acute, subacute, and chronic according to a widely accepted division on the basis of computed tomography (CT) presentation. Hyperdense hematoma was classified as acute, isodense as subacute, and hypodense as chronic. For all patients with known preceding head injury, the time elapsed between the injury and onset of symptoms was analyzed versus the type of hematoma as determined on head CT scan. This procedure was intended to evaluate whether our hematoma classification based on CT presentation corresponded with the widely used classification based on the time from injury to the onset of neurological symptoms. Nearly all patients with acute hematomas were treated by craniotomy or craniectomy with evacuation of hematoma. Patients with subacute hematomas were primarily treated by evacuation via burr holes drilled above the hematoma site. Chronic hematoma was removed either via trepanation holes or by craniotomy.

The study data were subjected to a statistical analysis based on descriptive methods. The hypotheses were also verified. The Shapiro-Wilk test was used to determine the normalcy of distributions of quantitative variables. Since most quantitative variables did not follow a normal distribution and variances were heterogeneous between groups, analysis of the significance of differences for quantitative characteristics was based on non-parametric tests. Mann-Whitney's U test was used to evaluate differences between two independent groups and Kruskal-Wallis non-parametric analysis of variance (ANOVA) to evaluate distributional differences between three independent groups. For characteristics described with nominal scales, the structure and frequency of individual classes were evaluated using contingency tables and calculating expected counts. For data expressed as proportions, between-group comparisons utilized the χ^2 test of independence for 2×2 and $r \times c$ tables. If an expected count in a contingency table was below 5, correlations between variables were evaluated by means of exact two-tailed test probabilities p for the χ^2 test. In all analyses, the maximum admissible probability of a type I error (rejecting a true null hypothesis) was $\alpha = 0.05$.

Results

The series of 100 consecutive subdural hematomas included 38 acute hematomas, 20 subacute hematomas, and 42 chronic hematomas. All acute hematomas were unilateral. Two of the 20 subacute hematomas were bilateral. Eleven of the 42 chronic hematomas were bilateral. Mean patient age was 65.1 years for the entire series. Patient age differed significantly for the three hematoma types (Kruskal-Wallis ANOVA by ranks; $\chi^2 = 8.24$, degrees of freedom [df] = 2, p = 0.0163). Patients with chronic hematomas were significantly older than those with acute hematomas (mean age 69.67 vs. 60.68 years; p = 0.0158), but patients with subacute hematomas (mean age 64.05 years) did not differ significantly in age from

Table 1 Differences in distributions of quantitative variables analyzed in 100 patients with different types of hematoma

Variable	Type of hematoma	Mean	Median	Min	Max	SD	Kruskal-Wallis ANOVA		Post hoc tests for Kruskal-Wallis ANOVA by ranks		
							χ^2 (df)	p	Type of hematoma	Z	p
Age (yrs)							8.24 (2)	**0.0163**			
	chronic: C	69.67	75.5	28	89	15.47			C vs. S	1.7	0.2658
	subacute: S	64.05	61.5	46	86	11.44			C vs. A	2.79	**0.0158**
	acute: A	60.68	59	27	96	16			S vs. A	0.59	1.0
Duration of hospitalization (days)							9.74 (2)	**0.0077**			
	C	13.71	13	3	35	8.33			C vs. S	1.26	0.6181
	S	10.20	9	1	22	6			C vs. A	3.11	**0.0056**
	A	8.71	6.5	1	43	9.7			S vs. A	1.28	0.6059
GCS score on admission							24.66 (2)	<**0.0001**			
	C	12.45	13	4	15	2.83			C vs. S	0.18	1.0
	S	12.80	13.5	5	15	2.31			C vs. A	4.43	<**0.0001**
	A	8.03	6.5	3	15	4.28			S vs. A	3.77	**0.0005**
GOS score							20.48 (2)	<**0.0001**			
	C	3.67	4	1	5	0.95			C vs. S	0.79	1.0
	S	3.9	4	2	5	0.91			C vs. A	3.63	**0.0008**
	A	2.63	2	1	5	1.2			S vs. A	3.72	**0.0006**
Time between injury and onset* (days)							32.3 (2)	<**0.0001**			
	C	23.75	21	0	60	21			C vs. S	0.78	1.0
	S	13.8	7	0	60	19.13			C vs. A	5.09	<**0.0001**
	A	0.38	0	0	4	0.9			S vs. A	3.5	**0.0014**

Bold figures indicate statistically significant differences. *Patients with history of injury (n = 52). ANOVA: analysis of variance; χ^2: value of χ^2 statistic for Kruskal-Wallis ANOVA; df: number of degrees of freedom; Min and Max: minimum and maximum value, respectively; p: value of two-tailed asymptotic test probability for Kruskal-Wallis ANOVA and post hoc tests; SD: standard deviation; Z: value of Z statistic evaluating significance of group differences in post hoc tests.

Table 2 Correlation between type of hematoma and qualitative variables

Variable	Type of hematoma, N (%)			χ^2 (df)	p
	Chronic	Subacute	Acute		
Sex				3.01 (2)	0.2217
male	27 (64.3)	17 (85.0)	25 (65.8)		
female	15 (35.7)	3 (15.0)	13 (34.2)		
History of injury				7.39 (2)	**0.0248**
no	26 (61.9)	10 (50.0)	12 (31.6)		
yes	16 (38.1)	10 (50.0)	26 (68.4)		
Reoperation necessary				10.51 (2)	**0.0052**
no	26 (61.9)	13 (65.0)	35 (92.1)		
yes	16 (38.1)	7 (35.0)	3 (7.9)		
Death					
no	40 (95.2)	20 (100.0)	31 (81.6)		
yes	2 (4.8)	0 (0.0)	7 (18.4)		

Bold figures indicate statistically significant differences. χ^2: statistic of χ^2 test of independence, df: number of degrees of freedom, N: number of patients, p: value of two-tailed asymptotic test probability for χ^2 test of independence.

those with chronic or acute hematomas. The detailed data on age distribution in the three hematoma types can be found in Table 1.

This study included 69 males and 31 females, with no significant correlations between sex and type of hematoma ($\chi^2 = 3.01$, df = 2, p = 0.2217) (Table 2). A history of head injury was present in 52 patients, and no preceding head injury was found in 48 patients. There was a significant correlation between the type of hematoma and history of head injury ($\chi^2 = 7.39$, df = 2, p = 0.0248), with a positive history found significantly more frequently in patients with acute hematomas (68.4%) vs. those with chronic hematomas (38.1%). No history of previous head injury was significantly more frequent in patients with chronic hematomas (61.9%) vs. those with acute hematomas (31.6%). The detailed statistics regarding the correlation between the type of hematoma and a history of head injury are shown in Table 2.

For all patients with a history of head injury, the time between injury and onset of symptoms was analyzed versus the type of hematoma as determined by CT. These results are shown in Table 3. There were significant differences in the time between injury and onset between the hematoma types (Kruskal-Wallis ANOVA by ranks; $\chi^2 = 32.3$, df = 2, $p < 0.0001$), and the time between injury and onset in patients with acute hematomas differed significantly from that in patients with chronic (Z = 5.09, $p < 0.0001$) and subacute (Z = 3.5, p = 0.0014) hematomas (Table 1). Trauma patients with chronic hematomas did not differ significantly in the time interval from injury to the onset of symptoms from patients with subacute hematomas.

Table 3 Mean time from head trauma to clinical symptoms and hematoma density on computed tomography (CT)

Type of hematoma	Mean time from head trauma to clinical symptoms (days)	Density on CT
Acute	0.38	hyperdense
Subacute	13.8	≈ isodense
Chronic	23.75	hypodense

The mean duration of hospital stay at the Neurosurgery Department by type of hematoma is shown in Table 1. The three hematoma types differed significantly in the duration of hospital stay (Kruskal-Wallis ANOVA by ranks; p = 0.0077). Patients with chronic hematomas were hospitalized significantly longer than those with acute hematomas (Z = 3.11, p = 0.0056). There was no difference between patients with subacute hematomas and those with acute and chronic hematomas. Glasgow Coma Scale (GCS)-based evaluation of the neurological status on admission is also shown in Table 1. There were differences in GCS scores on admission by type of hematoma (Kruskal-Wallis ANOVA rank test; $\chi^2 = 24.66$, $p < 0.0001$). Patients with acute hematomas had significantly worse neurological status (GCS scores) compared to patients with subacute (Z = 3.77, p = 0.0005) and chronic (Z = 4.43, $p < 0.0001$) hematomas.

The type of primary surgery versus type of hematoma is presented in Table 4. Twenty-six patients were reoperated, of whom 22 underwent one additional procedure and four had two more operations. The type of hematoma correlated significantly with

Table 4 Type of primary surgery versus type of hematoma

Type of hematoma	No. of patients	Primary surgery	
		Craniotomy/craniectomy	Burr holes
Acute	38	34 (89.5%)	4 (10.5%)
Subacute	20	5 (25%)	15 (75%)
Chronic	42	7 (16.7%)	35 (83.3%)
Total	100	46	54

Table 5 Repeat surgeries following primary evacuation of hematoma by craniotomy/craniectomy

Type of hematoma	Craniotomy/ craniectomy	Reoperated cases	Reason for reoperation/number
Acute	34	2 (5.9%)	epidural hematoma/1, recurrent hematoma or residual collection/1
Subacute	5	3 (60%)	epidural hematoma/1, recurrent hematoma or residual collection/2
Chronic	7	3 (42.9%)	epidural hematoma/2, recurrent hematoma or residual collection/1
Total	46	8 (17.4%)	

Table 6 Repeat surgeries following trepanation as the primary procedure for hematoma evacuation

Type of hematoma	Burr holes	Reoperated cases	Reason for reoperation/number
Acute	4	1 (25%)	recurrent hematoma or residual collection/1
Subacute	15	4 (26.7%)	recurrent hematoma or residual collection/2, epidural hematoma/1
Chronic	35	13 (37.1%)	recurrent hematoma or residual collection/13, intracerebral hematoma/1
Total	54	18 (33.3%)	

the need to reoperate ($\chi^2 = 10.51$, df = 2, p = 0.0052) (Table 2). Repeat surgery was necessary significantly more frequently in patients with chronic hematomas (38.1%) compared to those with acute hematomas (7.9%). The distribution of repeat surgeries following primary removal of the hematoma by craniotomy/craniectomy is shown in Table 5, and following trepanation as the primary procedure for hematoma removal is shown in Table 6. There were no significant differences in the frequency of reoperations following different types of primary procedures between patients with chronic and subacute hematomas ($\chi^2 = 0.223$, df = 2, p = 0.3279) (Table 7). Repeat surgery was more often necessary following primary craniotomy (54.6%) than following primary trepanation with puncture (34.0%) in patients with chronic and subacute hematomas, but the differences did not reach the level of statistical significance. One reason may be the small size of this cumulative sample (n = 62), resulting in inadequate statistical power ($1 - \beta = 0.55$) to show moderate effects.

The neurological status was assessed on admission and at the end of the follow-up period. Improvement (i.e. a higher GCS score) was noted in 44 patients, deterioration (lower GCS or Glasgow Outcome Scale [GOS] = 1) in 20 patients, and no change in neurological status since admission (i.e. the same GCS score) was noted in 36 patients. There was a significant correlation between the type of hematoma and treatment outcomes ($\chi^2 = 15.91$, df = 4, p = 0.0031), with deterioration observed more frequently among patients with acute hematomas (36.8%) compared to those with subacute (0%) and chronic (14.3%) hematomas (Table 8).

The patients differed significantly in neurological status assessed with the GOS score at discharge from the hospital (Kruskal-Wallis ANOVA; $\chi^2 = 20.48$, df = 2, $p < 0.0001$) (Table 1). The neurological status (GOS) at discharge in patients with subacute and chronic hematomas was significantly better com-

Table 7 Correlation between type of hematoma and primary treatment in patients with subacute and chronic hematomas

Variable	Repeat surgery necessary, N (%)*		χ^2 (df)	p
	No	Yes		
Primary surgical technique			0.223 (2)	0.3279
craniotomy	5 (45.45)	6 (54.55)		
trepanation with puncture	33 (66.0)	17 (34.0)		
decompressive craniectomy	1 (100.0)	0 (0)		

*Row data percentage. χ^2: statistic of χ^2 test of independence, df: number of degrees of freedom, N: number of patients, p: value of two-tailed asymptotic test probability for χ^2 test of independence.

Table 8 Distribution of treatment outcomes by type of hematoma

Variable	No. of patients	Treatment outcome, N (%)			χ^2 (df)	p
		Improvement	No change	Deterioration		
Type of hematoma					**15.91 (4)**	**0.0031**
acute	38	16 (42.1%)	8 (21.1%)	14 (36.8%)		
subacute	20	8 (40%)	12 (60%)	0 (0%)		
chronic	42	20 (47.6%)	16 (38.1%)	6 (14.3%)		
Total	100	44	36	20		

Bold figures indicate statistically significant differences. χ^2: statistic of χ^2 test of independence, df: number of degrees of freedom, N: number of patients, p: value of two-tailed asymptotic test probability for χ^2 test of independence.

Table 9 Differences in age distribution and Glasgow Coma Scale (GCS) score distribution on admission in patients with subdural hematoma who died vs. did not die

Quantitative variable	Qualitative variable	Mean	Median	Min	Max	SD	Mann-Whitney test	
							U	p
Age (yrs)	Death						400	0.9136
	no	65.2	65	27	96	15.07		
	yes	64.44	70	33	85	19.26		
GCS score on admission	Death						160	**0.0024**
	no	11.23	13	3	15	3.84		
	yes	6.89	5	3	13	3.72		

Bold figure indicates statistically significant difference. Min and Max: minimum and maximum value, respectively; p: value of two-tailed asymptotic test probability for Mann-Whitney test; SD: standard deviation; U: value of U statistic for Mann-Whitney test.

Table 10 Operation time frame during day and night

Operation time frame	Number of operations
7:00–15:00	22
15:00–23:00	45
23:00–7:00	33

pared to that in patients with acute hematomas (post hoc tests; Z = 3.72, p = 0.0006 and Z = 3.63, p = 0.0008, respectively). There were also differences in the mortality rate versus type of hematoma (χ^2 = 7.02, df = 2, p = 0.0323), as 4.8% of the patients with acute hematomas died, compared to 18.4% of those with chronic hematoma, and no deaths among those with subacute hematomas (Table 2). GCS-based neurological status on admission also differed with regard to subsequent death (Mann-Whitney test; U = 160, p = 0.0024). Significantly higher mean GCS score on admission was noted among those patients who did not die (11.23) compared to those who subsequently died (6.89) (Mann-Whitney test; Z = 3.04, p = 0.0024) (Table 9). There were no significant differences with regard to age in this comparison.

The time of day at which surgery took place was also analyzed and the data are presented in Table 10.

Discussion

A widely accepted classification divides subdural hematomas into acute, subacute, and chronic according to the time elapsed between the injury and the onset of clinical symptoms. The borderline between subacute and chronic hematomas is variously defined by different authors. Most authors define chronic hematoma as clinically manifesting after more than 2 or 3 weeks following an injury. In our series, the time between the injury and the onset of neurological symptoms and the type of hematoma as identified by CT on admission to the hospital was analyzed in all patients with a history of injury to the head (52 patients). The mean time interval between the injury and the onset of symptoms that were the reason for hospitalization in this population was

0.38 days for acute hematoma, 13.8 days for subacute hematoma, and 23.75 days for chronic hematoma. These values fall within the ranges established by a conventional and widely used division which defines these time intervals at <3 days for acute hematoma, 4–21 days for subacute hematoma, and >21 days for chronic hematoma.[4,7] These results confirm the utility of our classification in everyday clinical practice. Of all patients in the series, 48 had not suffered head injury. Of note, there was no preceding head injury in 31.6% of acute hematomas, 50% of subacute hematomas, and 61.9% of chronic hematomas.

The data for chronic hematomas are consistent with previous reports, which estimate the frequency of preceding head injury at less than 50% of cases.[4,10] In some cases, because of rather trivial head trauma, some patients (especially older) forget about any trauma which happened some time ago and thus do not associate it with the development of hematoma. Consequently, these patients or members of their family do not report such head trauma to the personnel of the hospital. Other risk factors which can cause development of chronic subdural hematoma are alcohol abuse, seizures, cerebrospinal fluid shunts and coagulopathies (including therapeutic anticoagulation).[6] Acute hematomas have been associated with preceding injury more frequently in the literature than in our series.[18.23] Acute subdural hematomas may occur in patients receiving anticoagulation therapy, usually with but sometimes without a history of trauma. Data from the literature shows that receiving anticoagulation therapy increases the risk of acute subdural hematoma 7-fold in males and 26-fold in females.[6,24]

Many chronic subdural hematomas probably start out as acute subdural hematomas. Blood within the subdural space evokes an inflammatory response. Within days, fibroblasts invade the clot, and form neomembranes on the inner (cortical) and outer (dural) surface, followed by in-growth of neocapillaries, enzymatic fibrinolysis, and liquefaction of blood clot. Fibrin degradation products are reincorporated into new clots and inhibit hemostasis. The course of chronic subdural hematoma is determined by the balance between plasma effusion and/or rebleeding from the neomembranes and the reabsorption of fluid.[2,4,8]

The mean duration of hospitalization at our Neurology Department was 11.1 days for the entire series, shortest in the patients with acute hematomas (8.7 days) and longest in the patients with chronic hematomas (13.7 days). These results may have been influenced by the fact that some patients with acute hematomas were transferred to the Intensive Care Unit for further treatment following surgery and so their mean hospital stay at the Neurosurgery Department was shorter than for the other patients. Another factor was that the highest rate of reoperations, which lengthened hospital stay, was seen in patients with chronic hematomas.

In the entire series of 100 patients, craniotomy or craniectomy was the primary procedure for removing the hematoma in 46 patients, whereas the remaining 54 underwent trepanation. Craniotomy was used in the vast majority (89.5%) of patients with acute hematomas. Decompressive craniectomy was performed in patients with considerable cerebral edema or in those expected to develop edema postoperatively. Some craniectomies were combined with meningo-periosteoplasties to slightly enlarge and caulk the space for the edematous brain. The craniectomy patients subsequently required cranioplasty, provided the treatment for hematoma had been successful. In a few cases, the removed skull bone was secured in the abdominal wall to be successfully used in subsequent reconstructive surgery.[3,21] In the remaining patients, skull reconstruction used artificial substitutes.

Subacute hematomas, which appear isodense on CT scans, can be conveniently evacuated by burr hole trepanation. These hematomas are sufficiently liquefied and can be evacuated/washed out through burr holes, as a capsule that might impede complete evacuation of the hematoma has not yet formed. The capsule begins to form on the fourth day after injury.[1,9,14] In our series, the mean time between injury and symptom onset in patients with subacute (isodense CT appearance) hematomas was 13.8 days. Trepanation was chosen as the primary surgical technique in 75% of patients with subacute hematomas. None of the patients had a capsule around the hematoma that would impede evacuation. In patients with chronic hematomas, 83.3% of procedures were burr hole trepanations and the remaining patients underwent craniotomy. The primary treatment was burr hole trepanation in patients with hypodense hematomas without visible ''septa'' inside the capsule dividing the hematoma into smaller compartments and without evidence of additional new-onset bleeding. Trepanation holes were planned and drilled so that craniotomy would be feasible if intraoperative inspection should reveal that adequate evacuation of the hematoma through the burr holes was not possible. If CT revealed an unfavorable capsular pattern as described above, the decision was made to perform craniotomy as the primary treatment. In patients with chronic hematomas, 26.2% had bilateral hematomas. Bilateral hematomas are reported to account for 20–25% of chronic hematomas.[16,22] During surgery, the internal capsule, which adheres to the cerebral tissue, should not be removed.[4,16] In our series, during burr hole trepanation procedures, the burr holes were slightly widened once drilled since the trephine diameter was usually too small. It is recommended that burr hole diameter should be larger than 2.5 cm.[4] Following incision of the dura, preferably in a criss-cross pattern, it is also important to coagulate loose fragments of the dura to cause shrinkage, in order to produce an aperture in the dura mater roughly corresponding in size to the trephine hole. The aperture can then be used to evacuate the hematoma across its diameter.

In patients with subacute and chronic hematomas, a subdural drain was left in place. Placing a drain reduces the repeat surgery rate by approximately 10–19% compared to undrained hematomas.[11,13] The draining receptacle was usually placed about 50–80 cm below head level[12,19] and the drainage was usually maintained for 2 more days. The amount of intravenous fluids was also increased and the head was positioned flat for 48 hours in order to facilitate expansion of the brain. In patients with subacute and chronic hematomas, follow-up CT of the head was performed about 3 days after the surgery. The decision to reoperate was made if the hematoma was still causing a mass effect. Persistent hematoma visible on follow-up CT of the head should not constitute an indication to reoperate, especially within approximately 20 days of the initial surgery, unless there is no improvement, or actually deterioration, of the patient's neurological status, and the hematoma volume has increased compared to the baseline scan.[12,17,19] Clinical improvement is observed when the subdural pressure is reduced to close to zero, which usually occurs after about 20% of the collection is removed.[19] Patients with high subdural fluid pressure tend to have more rapid expansion and clinical improvement than patients with low pressure.[12] Importantly, in elderly patients, rapid decompression following evacuation of hematoma may be associated with hyperemia of the cerebral cortex directly inferior to the hematoma, which may give rise to an intracerebral hematoma or epileptic seizures.[15]

In our series of 100 consecutive patients operated on for subdural hematoma, 26 patients underwent repeat surgery. There was a significant correlation between the type of hematoma and the need to reoperate ($\chi^2 = 10.51$, df = 2, p = 0.0052). Repeat surgery was necessary significantly more frequently in patients with chronic hematomas compared to those with acute hematomas. Of the 46 craniotomies performed, eight (17.4%) patients needed repeat surgery, compared to 18 (33.3%) of 54 patients who underwent trepanation. The choice of craniotomy/craniectomy as a method of choice in the treatment of acute hematomas is not controversial. For subacute and chronic hematomas, various authors speak in favor of either trepanation or craniotomy. Analysis of repeat surgeries in our series indicates that burr hole trepanation is possibly superior to craniotomy as the primary technique. Reoperations of subacute hematomas following craniotomy were performed in 60% of the patients, compared to 42.9% of patients with chronic hematomas, whereas the respective rates following trepanation were 26.7% and 37.1%. However, these differences did not reach the level of statistical significance. One reason for this failure to reach statistical significance might have been the small sample size (only 62 patients with chronic and subacute hematomas), which resulted in inadequate statistical power ($1 - \beta = 0.55$) to reveal moderate effects.

Comparison of neurological status on admission and at the end of the follow-up period revealed improvement in 44 patients, deterioration in 20, and no change from baseline in 36. Evaluation of the neurological status was based on the GCS and the GOS.[5,20] The mean duration of hospitalization at the Neurosurgery Department was 11.1 days for all patients. Patients were evaluated on admission to the hospital and at discharge from the Neurosurgery Department or the Intensive Care Unit. Deaths (GOS = 1) occurred in 9 patients, mostly with acute hematomas and poor neurological status on admission. The mortality rate was 18.4% in patients with acute hematomas and 4.8% in those with chronic hematomas. There were no deaths among the patients with subacute hematomas. The mean GCS score on admission in the patients who subsequently died was 6.89, and was lower than the mean GCS score of 8.03 for all patients with acute hematomas. The mean GCS score for the entire series was 10.8. The mean age of the patients who died was 64.4 years and the mean age of all patients in the series was 65.1 years. Mortality in patients with subdural hematomas was apparently linked directly to the type of hematoma and baseline neurological status, and less so to age. The time intervals during which operations were performed were also analyzed, showing that the overwhelming majority of subdural hematoma surgeries were undertaken outside usual working hours, as 45% of the operations were carried out between 15:00 and 23:00, and 33% between 23:00 and 7:00, with only 22% carried out between 7:00 and 15:00. This reflects the degree of work load and involvement of the entire medical personnel of Neurosurgery Departments contributing to the treatment, who need to be alert and ready to act around the clock.

Our classification of hematoma based on CT presentation corresponds to the classification based on the time elapsed between injury and onset of symptoms, and appears to be appropriate and useful in everyday practice. No preceding injury was identified in 31.6% of acute hematomas, 50% of subacute hematomas, and 61.9% of chronic hematomas. Analysis of reoperations indicates that trepanation may be superior to craniotomy as the primary surgery for subacute and chronic hematomas. Subdural hematoma surgeries take place at all times of the day, with most carried out outside the usual working hours.

Conflicts of Interest Disclosure

The authors have no personal financial or institutional interest in any of the drugs, materials, or devices in the article.

References

1) Aoki N, Oikawa A, Sakai T: Symptomatic subacute subdural haematoma associated with cerebral hemispheric swelling and ischemia. *Neurol Res* 18: 145–149, 1996
2) Drapkin AJ: Chronic subdural hematoma: Pathophysiological basis of treatment. *Br J Neurosurg* 5: 467–473, 1991
3) Flannery T, McConnell RS: Cranioplasty: why throw the bone flap out? *Br J Neurosurg* 15: 518–520, 2001
4) Greenberg MS: Head trauma. Subdural hematoma, *in* Greenberg MS (*ed*): *Handbook of Neurosurgery, sixth edition*. New York, Thieme, 2006, pp 672–679
5) Jennet B, Bond M: Assessment of outcome after severe brain damage: A practical scale. *Lancet* 480–484, 1975
6) Kawamata T, Takeshita M, Kubo O, Izawa M, Kagawa M, Takakura K: Management of intracranial hemorrhage associated with anticoagulant therapy. *Surg Neurol* 44: 438–443, 1995
7) Kaye AH: Traumatic intracranial haematomas, *in* Kaye AH (*ed*): *Essential Neurosurgery, third edition*. Melbourne, Blackwell Publishing, 2005, pp 56–63
8) Labadie EL: Fibrinolysis in the formation and growth of chronic subdural hematomas, *in* Sawaya R (*ed*): *Fibrinolysis and the Central Nervous System*. Philadelphia, Hanley & Belfus, 1990, pp 141–148
9) Lee KS: Natural history of chronic subdural haematoma. *Brain Inj* 18: 351–358, 2004
10) Lee KS, Bae WK, Doh JW, Bae HG, Yun JG: Origin of chronic subdural haematoma and relation to traumatic subdural lesions. *Brain Inj* 12: 901–910, 1998
11) Lind CR, Lind CJ, Mee EW: Reduction in the number of repeated operations for the treatment of subacute and chronic subdural hematomas by placement of subdural drains. *J Neurosurg* 99: 44–46, 2003
12) Markwalder TM, Steinsiepe KF, Rohner M, Reichenbach W, Markwalder H: The course of chronic subdural hematomas after burr-hole craniostomy and closed-system drainage. *J Neurosurg* 55: 390–393, 1981
13) Mori K, Maeda M: Surgical treatment of chronic subdural hematoma in 500 consecutive cases: clinical characteristic, surgical outcome, complications, and recurrence rate. *Neurol Med Chir (Tokyo)* 41: 371–381, 2001
14) Munro D, Merritt HH: Surgical pathology of subdural hematoma: Based on a study of one hundred and five cases. *Arch Neurol Psychiatry* 35: 64–78, 1936
15) Ogasawara K, Koshu K, Yoshimoto T, Ogawa A: Transient hyperemia immediately after rapid decompression of chronic subdural hematoma. *Neurosurgery* 45: 484–489, 1999
16) Robinson RG: Chronic subdural hematoma: Surgical management in 133 patients. *J Neurosurg* 61: 263–268, 1984
17) Sambasivan M: An overview of chronic subdural hematoma: experience with 2300 cases. *Surg Neurol* 47: 418–422, 1997
18) Seelig JM, Becker DP, Miller JD, Greenberg RP, Ward JD, Choi SC: Traumatic acute subdural hematoma: Major mortality reduction in comatose patients treated within four hours. *N Engl J Med* 304: 1511–1518, 1981
19) Tabaddor K, Shulman K: Definitive treatment of chronic subdural by twist-drill craniostomy and closed-system drainage. *J Neurosurg* 46: 220–226, 1977
20) Teasdale G, Jennett B: Assessment of coma and impaired consciousness: A practical scale. *Lancet* 2: 81–84, 1974
21) Tybor K, Fortuniak J, Komuński P, Papiez T, Andrzejak S, Jaskólski D, Shariati B, Zawirski M: [Supplementation of cranial defects by an autologous bone flap stored in the abdominal wall]. *Neurol Neurochir Pol* 39: 220–225, 2005 (Polish)
22) Wakai S, Hashimoto K, Watanabe N, Inoh S, Ochiai C, Nagai M: Efficacy of closed-system drainage in treating chronic subdural hematoma: A prospective comparative study. *Neurosurgery* 26: 771–773, 1990
23) Wilberger JE, Harris M, Diamond DL: Acute subdural hematoma: Morbidity, mortality, and operative timing. *J Neurosurg* 74: 212–218, 1991
24) Wintzen AR, Tijssen JGP: Subdural hematoma and oral anticoagulation therapy. *Ann Neurol* 39: 69–72, 1982

Address reprint requests to: Bartosz Godlewski, MD, Department of Neurosurgery and Peripheral Nerves Surgery, WAM University Hospital in Lodz, Medical University of Lodz, 113 Zeromskiego Street, 90-549 Lodz, Poland.
e-mail: bartoszgodlewski@wp.pl