

The Relation Between Surgically Treated Spontaneous Intracerebral Hematomas and Mortality: Retrospective Evaluation of 72 Cases

Cerrahi Tedavi Uygulanan Spontan İntraserebral Hematomlar ve Mortalite İlişkisi: 72 Olgunun Retrospektif Değerlendirilmesi

Mete Karatay, Reyhan Mehmetoğlu, Yavuz Erdem, Bülent Gülensoy, Tuncer Taşcıoğlu, İdris Sertbaş, Mehmet Akif Bayar Ministry of Health Ankara Training and Research Hospital, Department of Neurosurgery, Ankara, Turkey

Summary

Objective: In this study we investigated the relationship between mortality and various factors discovered during the surgical treatment of spontaneous intracerebral hematoma in cases presented to our clinic.

Material and Method: Between 1997 and 2009, there were 72 cases of spontaneous intracerebral hemorrhage presenting with non-traumatic intracerebral hemorrhage who underwent surgical treatment at our clinic and were evaluated retrospectively.

Results: Among the 72 cases, 41 (57%) were males, and 31 (43%) were females. The etiology in 52 (72%) of the cases was hypertension. Regarding the location of the hematoma, 27 (37%) of the cases were lobar, 30 cases (41%) were thalamic, 13 (18%) were cerebellar and 2 cases (4%) were in the putaminal location. The mortality rate in cases that received surgery within the first 8 hours was 41% (18 cases), and the rate was 61% (14 cases) in patients that received surgery between 8-24 hours. If surgery was performed between 24-48 hours following the initial hematoma, the mortality rate was 80% (4 cases).

Discussion: In spontaneous intracerebral hematoma, surgical treatment performed as quickly as possible following consideration of the initial neurological examination, age, volume of hematoma and its localization reduces mortality. Clinically there is no advantage in using surgical treatment over medical treatment in cases with a poor neurological condition. (*Turkish Journal of Neurology 2012; 18:83-7*)

Key Words: Intracerebral hematoma, surgical treatment, GCS (Glasgow Coma Scale), mortality

Özet

Amaç: Bu çalışmada kliniğimizde spontan intraserebral hematom nedeniyle cerrahi tedavi uygulanan olgularda yer alan faktörlerin mortaliteyle olan ilişkileri araştırıldı.

Gereç ve Yöntem: 1997 ile 2009 yılları arasında nontravmatik intraserebral hemoraji nedeni ile başvuran ve cerrahi yöntemler ile tedavi edilmiş 72 spontan intraserebral hemoraji olgusu retrospektif olarak incelenmiştir.

Bulgular: İncelenen 72 olgunun 41'i (%57) erkek, 31'i (%43) kadın'dı. Elli iki olguda (%72) etiyoloji hipertansiyondu. Hematom lokalizasyonu olarak 27 olgu (%37) lober, 30 olgu (%41) talamik, 13 olgu (%18) serebellar, 2 olgu (%4) putaminal yerleşimliydi. İlk 8 saatte cerrahiye alınan olgularda mortalite oranı %61 (14 olgu), 8 ile 24 saat arasında cerrahiye alınan olgularda mortalite oranı %61 (14 olgu), 24 ile 48 saat arasında cerrahiye alınan olgularda mortalite oranı %80 (4 olgu) olarak bulundu.

Sonuç: Spontan intraserebral hematom olgularında ilk nörolojik muayene, yaş, hematom volümü, hematomun lokalizasyonu ve cerrahiye alınma süresi göz önüne alınarak yapılacak olan cerrahi tedavi mortaliteyi azaltır. Klinik olarak kötü nörolojik durumdaki olgularda uygulanacak cerrahi tedavinin medikal tedaviye üstünlüğü yoktur. (*Türk Nöroloji Dergisi 2012; 18:83-7*)

Anahtar Kelimeler: İntraserebral hematom, cerrahi tedavi, GKS (Glaskow Koma Skalası), mortalite

Address for Correspondence/Yazışma Adresi: Mete Karatay MD, Ministry of Health Ankara Training and Research Hospital, Department of Neurosurgery, Ankara, Turkey Phone.: +90 312 595 36 00 E-mail: lexel26@hotmail.com

Received/Geliş Tarihi: 20.02.2012 Accepted/Kabul Tarihi: 26.08.2012

Introduction

Spontaneous intracerebral hemorrhage is defined as a hemorrhage without a history of trauma. Spontaneous intracerebral bleeding is the third most common cause of a stroke, which is often due to hypertension. The mortality rate resulting from these hemorrhages is high. There is a possibility of early and easy diagnosis with the use of computerized brain tomography (CBT). By using early diagnosis there have been significant changes in treatment approaches and prognoses. Accurate selection of cases for surgical treatment is the most important factor in post-surgical prognosis. Although there have been many studies on this subject, deficient data comparing medical and surgical treatments published from the same source, insufficient follow-up on the cases, and the absence of a common classification of this type hematomas adopted by all authors has made the indications for surgical treatment a controversial topic (1,2). In this study the relationship between etiology, clinical symptoms, surgical treatment indications, treatment approaches, and mortalities of 72 cases with spontaneous intracerebral hematomas treated between 1997-2009 were reviewed and evaluated.

Materials and Methods

We retrospectively evaluated 72 cases of non-traumatic intracerebral hemorrhage who were surgically treated. The cases in which the etiology was determined to be trauma, tumor, aneurysm and arteriovenous malformation were not included in the study. Our study only included intracerebral hematoma cases with hypertension, bleeding disorders, systemic disease, or idiopathic origin in their etiology. The effects of age, etiology, neurological evaluation, localization, the volume of bleeding, and the timing of the surgery on mortality were investigated. Among the cases included in the study, 31 (43%) were females, while 41 (57%) were males. The youngest case was 8 months old, while the oldest was 85 years old (Table 1).

All of our patients were admitted to our hospital within the first 48 hours after the onset of symptoms. A detailed neurological examination was performed and followed by an assessment of the level of consciousness based on Glasgow Coma Scale (GCS). After the first neurological examination, computerized brain tomography (CBT) scans were taken for all cases included in the study. Controlling hypertension was the first step in treatment. To investigate the etiology, routine blood tests, CBT, MRI and cerebral angiography were performed. Intracerebral hematomas were classified according to their localization as follows: lobar, putaminal, thalamic and cerebellar. In addition, they were classified according to the presence of intraventricular extension. Based on the location, it was determined that 27 cases (37%) were lobar, 2 cases (3%) were putaminal, 30 (42%) were thalamic, and 13 (18%) were cerebellar (Table 2). Furthermore, in 21 cases (30%) the hematoma extended into the ventricular system.

Results

The etiological evaluation of cases with intracerebral hematomas showed hypertension was the most frequent cause. Systemic disorders were second, idiopathic was third, and bleeding disorders were the fourth most frequent etiology. In all cases, blood pressure exceeding 140/90mmHg was accepted as hypertensive. Fifty-two cases (72%) were previously diagnosed with hypertension. From the patient histories of cases with diagnosed hypertension where hypertension treatment was started, 23 cases (44%) were determined to have used their treatment irregularly. In 7 cases (9%), a bleeding disorder was found, while in 5 cases (7%) a systemic disorder was observed. In 8 cases (12%) the cause of the hematoma could not be detected and they were classified as idiopathic (Table 3).

The mortality rate of cases where intracerebral hematoma was diagnosed and surgery was performed within the first 8 hours after diagnosis was 41% (18 cases); the rate was 61% (14 cases) in cases where the surgery was done between 8-24 hours after diagnosis. In cases where the surgical treatments were done between 24-48 hours, the mortality rate was 80% (4 cases) (Table 4).

The mortality rate in 18 cases with a hematoma volume less than 25 cm³ was found to be 50% (9 cases). In 19 cases with a hematoma volume of 25-50 cm³, the mortality rate was 36.8%

Table 1. The age distribution of 72 cases diagnosed with spontaneous intracerebral hematoma				
Age (years)	Number of cases			
<40	5			
40-49	3			
50-59	17			
60-69	16			
>70	31			
TOTAL	72			

Localization Number of cases			
Lobar	27		
utaminal	2		
Thalamic	30		
erebellar	13		
OTAL	72		

tiology Number of case		
52		
7		
8		
5		
72		

(7 cases), and in 35 cases with a hematoma volume larger than 50 cm³, the mortality rate was 57.1% (20 cases). The calculation of the hematoma volume was done by multiplying the width of the hematoma's mass (A) and its length (B) with its height (C). Width, length and height were determined by using the number of the observed cross-sections on the CBT scan and then dividing them by two (AxBxC/2) (Table 5).

In 17 cases where the GCS was between 13-15 there was no mortality observed post surgically. In 34 cases with a GCS of 7-12 the mortality rate was 45% (15 cases), while all 21 cases with GCS of 6-7 died (Table 6).

The indication for surgical treatment in cases that underwent surgery were obtained by the decline of the neurological status despite intensive medical treatment, and/or by the presence of the mass effect due to hematoma volume of the lobar, putaminal and thalamic hematomas. External ventricular drainage was performed in cases where the bleeding extended in the ventricles, creating a progressive worsening of the neurological status (GCS) along with the development of hydrocephalus. In cerebellar hematomas the brain stem compression findings were taken into account. The surgical methods applied in these cases were craniotomy, decompressive craniotomy or external ventricular drainage.

Discussion

Spontaneous intracerebral hematomas often occur deep in the gray matter. The hemorrhage formed in the cerebral hemispheres tends to spread to the white matter by dissecting the fiber fascicules. Generally the hemorrhage reaches its final size in the first 15-20 minutes. This bleeding is controlled by the brain's elastic resistance and cranial perfusion pressure. Fujitsu et al. showed that in most of the 180 cases of putaminal bleeding observed in the first 3 hours and followed by serial CBT scans, then treated medically and surgically, the

hematomas reached their maximum size within the first 6 hours (3). The most frequent cause of spontaneous intracerebral hematomas is hypertension. The cause of bleeding due to hypertension is still controversial. In 1868, Charcot and Bouchart asserted that the bleeding due to hypertension is due to rupturing of microaneurysms in intracerebral arteries and this opinion was supported for a century. This explains the frequency of hypertensive bleeding in relatively thin-walled lenticulostriate and paramedian arteries (4,5). Benes et al., in their postmortem study of cases with spontaneous intracerebral hemorrhage, described two types of spontaneous hemorrhages. The first type is massive acute bleeding, which damages peripheral brain tissue and extends to the ventricles and obstructs the ventricular circulation. This bleeding may cause a fatal intracranial pressure increase and does not give a good response to either medical or surgical treatment. The second type of hemorrhage that starts as a large hematoma, progresses slowly and acts as a peripheral mass lesion. In these cases, surgical drainage of the hematoma gives good results. Generally, it is the perforated artery that bleeds (6). The hematoma mass causes pressure on the surrounding tissue and results in damage to the tissue. At the same time, the increase in the intracranial pressure affects the cerebral perfusion and venous drainage. For this reason, treatment of the hematoma should be done quickly. Also, when deciding on surgery, these same reasons should be carefully considered if the hematoma mass could have a lifethreatening effect; regarding the hematoma drainage, it should also be considered whether damage of the brain tissue surrounding the hematoma can be prevented (6-8). Craniotomy, decompressive craniectomy and external ventricular drainage systems can be applied as surgical treatment (9-11). The mortality rate is higher in intracerebral hematomas compared to other types of stroke (12). The first aim of surgical treatment is

Surgical Timing	Number of cases	Number of deaths	Mortality rate
The first 8 hours	44 (L:17, P:1, T:18, C:8)	18 (L:5, P:1, T:9, C:3)	41
8-24 hours	23 (L:9, P:2, T:9, C:3)	14 (L:3, P:2, T:7, C:2)	61
24-48 hours	5 (L:0, P:0, T:3, C:2)	4 (L:0, P:0, T:3, C:1)	80
TOTAL	72 (L:26, P:3, T:30, C:13)	36 (L:8, P:3, T:19, C:6)	50

Table 5. Volume of the hematoma, localization and relation to mortality							
Volume of the hematoma	Lobar	Putaminal	Thalamic	Cerebellar	Total	Deaths	Mortality rate
<25 cm ³	0	1	13	4	18	9	50
25-50 cm ³	8	0	2	9	19	7	37
>50 cm ³	18	2	15	0	35	20	57
TOTAL	26	3	30	13	72	36	50

to prevent mortality and the second aim is to reduce any remaining sequelae. In the Kanaya et al. study, which is the largest study to date consisting of 5255 cases, they reported a 22% post-operative mortality (10). Kaneko et al. reported 7 cases of mortality in their study of 100 cases operated in emergency situations (11). The most important risk factors leading to spontaneous intracerebral bleeding are advanced age, acute and chronic hypertension. Two-thirds of the patients in our study were between the ages of 45-75, and spontaneous intracerebral bleeding was seen 5-20% more often in males than females (13). Fifty-nine percent of the cases were of patients older than 50 with an age distribution peaking at around 70. This finding is consistent with the literature. A past medical history of hypertension is present in 72-78% of all intracerebral hemorrhage cases (10,14). In our study, 52 cases (72 %) were hypertensive. This ratio was consistent with the literature. In cases with increasing volume of intracerebral hemorrhage, distinctive hypertension may be associated with the early stages of bleeding. Whether this distinctive hypertension is a secondary effect due to intracranial mass or the reason for the ongoing bleeding is a subject of discussion (1). Intracerebral hemorrhages associated with bleeding disorders constitute only 2.1-13.3% of all intracerebral hemorrhages (15). Moreover, in cases treated with anticoagulants, the intracerebral hemorrhage ratio is 1.6-3.1% (16) for a variety of reasons. The risk of intracerebral hemorrhage increases 7.6-11% in cases receiving oral anticoagulant treatment compared to other forms of treatment (17-19). In our study, there were 7 cases (9%) with a determined bleeding disorder. Surgical treatment was performed after the clotting tests were brought to normal levels. In the literature, the idiopathic intracerebral hemorrhage ratio varies between 6-12% (20,21). In our study we found this ratio to be 12% (8 cases), which is consistent with the literature. Surgical treatment was suggested in cases with GCS of 6-12, or in cases in which the initial GCS was 13-15 but decreased over time (9,14). The prognosis was worse in cases with GCS below 7 (3). In our study, surgical treatment was applied to cases with GCS of 6-12. We did not observe mortality in cases with GCS 13 and above (17 cases). In 21 cases with GCS below 7 the mortality rate was 100%. The width and volume of the intracerebral hematoma also affects the prognosis, and it may be discerned that the larger volume and deeper placement of the hematoma results in a worse prognosis (22). All three patients

Table 6. The relationship between GCS and mortality				
GCS	Number of cases	Number of deaths	Mortality rates	
GCS: 13-1	5 17	0	0	
GCS: 7-12	34	15	45	
GCS: 6-7	21	21	100	
TOTAL	72	36	50	
GCS: Glascow Coma Scale				

with putaminal bleeding in our study died. 63% of cases (19 out of 30 cases) with hematomas located in the thalamus and 31% of cases (8 out of 26 cases) with lobar-subcortical localization of hematomas died. We found a higher mortality rate in cases with deeper placement of the hematoma, which was also observed in the literature. 6 patients (46%) among 13 patients with cerebellar hemorrhage died. In 4 of those cases the volume of the hematoma was more than 25 cm³. The mortality rate in cases with a hematoma volume less than 25 cm³, 25-50 cm³, and more than 50 cm³ was 50% (9 cases), 37% (7 cases) and 57% (20 cases) respectively. The literature reports that there is not much benefit for surgeries performed after 24 hours from the onset of the stroke symptoms (11,14). In the cases discussed in this paper, when surgery was performed within the first 8 hours without looking for the location of the hematoma, the mortality rate was 41% (18 cases). When surgery was done within 8-24 hours, the mortality rate was 61% (14 cases). The mortality rate was 80% (4 cases) when the surgery was performed after 24-48 hours. These findings were consistent with the literature.

Conclusion

In cases with spontaneous intracerebral hematomas, surgical treatments done as quickly as possible following consideration of the initial neurological examination, age, volume of the hematoma and location of the hematoma reduces mortality. Clinically, there is no advantage in using surgical treatment over medical treatment in cases with poor neurological status.

References

- Broderick JP, Brott TG, Tomsick T, Barsan W, Spilker J. Ultra-early evalutation of intracerebral hemorrhage. J Neurosurg 1990;72:195-9.
- Waga S, Miyazaki M, Okada M, Tochio H, Matsushima S, Tanaka Y. Hypertensive putaminal hemorrhage: analysis of 182 patients. Surg Neurol 1986;26:159-66.
- Fujitsu K, Muramoto M, Ikeda Y, Inada Y, Kim I, Kuwabara T. Indications for surgical treatment of putaminal hemorrhage. Comparative study based on serial CT and time-course analysis. J Neurosurg 1990;73:518-25.
- Franke C L, van Swieten JC, Algra A, van Gijn J. Prognostic factors in patients with intracerebral haematoma. J Neurol Neurosurg Psychiatry 1992;55:653-7.
- Giroud M, Gras P, Chadan N, Beuriat P, Milan C, Arveux P, et al. Cerebral haemorrhage in a french prospective population study. J Neurol Neurosurg Psychiatry 1991;54:595-8.
- Benes V Jr, Malý Z. Intracerebral hematoma following superficial temporal-middle cerebral artery anastomosis. Case report. Zentralbl Neurochir 1985;46:133-6.
- Anderson CS, Chakera TM, Stewart-Wynne EG, Jamrozik KD. Spectrum of prirriary intracerebral haemorrhage in Perth, VVestern. Australia, 1989-90: incidence and outcome. J Neurol Neurosurg Psychiatry 1994;57:936-40.
- Wakai S, Yamakawa K, Manaka S, Takakura K. Spontaneous intracranial hemorrhage caused by brain tumor: Its incidence and clinical significance. Neurosurgery 1982;10:437-44.
- Fernandes HM, Mendelow AD. Spontaneous intracerebral haemorrhage: a surgical dilemma. Br J Neurosurg 1999;13:389-94.
- Kanaya H. Current status of surgical therapy of hypertensive cerebral hemorrhage in Japan. Nihon Rinsho 1982;40:2775-82.

- Kaneko M, Koba T, Yokoyama T. Early surgical treatment for hypertensive intracerebral hemorrhage. J Neurosurg 1977;46:579-83.
- Sacco RL, Benjamin EJ, Broderick JP, Dyken M, Easton JD, Feinberg WM, et al. Risk Factors. Stroke 1997;28:1507-17.
- 13. Caplan LR. Intracerebral Haemorrhage. Lancet 1992;339:656-8.
- Kaneko M, Tanaka K, Shimada T, Sato K, Uemura K. Long term evaluation of ultra-early operation for hypertensive intracerebral hemorrhage in 100 cases. J Neurosurg 1983;58:838-42.
- Tatu L, Moulin T, El Mohamad R, Vuillier F, Rumbach L, Czorny A. Primary intracerebral hemorrhages in the Besancon stroke registry. Initial clinical and CT findings, early course and 30-day outcome in 350 patients. Eur Neurol 2000;43:209-14.
- Fogelholm R, Eskola K, Kiminkinen T, Kunnamo I. Anticoagulant treatment as a risk factor for primary intracerebral haemorrhage. J Neurol Neurosurg Psychiatry 1992;55:1121-4.

- 17. Forsting M, Mattle HP, Huber P. Anticoagulation-related intracerebral hemorrhage. Cerebrovasc Dis 1991;1:97-102.
- Franke CL, de Jonge J, van Swieten JC, Op de Coul AA, van Gijn J. Intracerebral hematomas during anti-coagulant treatment. Stroke 1990;21:726-30.
- Zuccarello M, Brott T, Derex L, Kothari R, Sauerbeck L, Tew J, et al. Early surgical treatment for supratentorial intracerebral hemorrhage: a randomized feasibility study. Stroke 1999;30:1833-9.
- Fogelholm R, Nuutila M, Vuorela AL. Primary intracerebral haemorrhage in the Jyvaskyla region. Central finland, 1985-89, incidence, case fatality rate, and functional outcome. J Neurol Neurosurg Psychiatry 1992;55:546-52.
- 21. Wintzen AR, de Jonge H, Loeliger EA, Bots GT. The risk of intracerebral hemorrhage during oral anticoagulant treatmant: a population study. Ann Neurol 1984;16:553-8.
- Broderick JP, Brott TG, Duldner JE, Tomsick T, Huster G. Volume of intracerebral hemorrhage: A powerful and easy-to-use predictor of 30-day mortality. Stroke 1993;24:987-93.