Original Research

Bagcilar Medical Bulletin 2016;1(1):12-17
DOI: 10.5350/BMB2016010103

Factors Affecting the Outcome in Traumatic Subarachnoid Hemorrhage

Feyza Karagoz Guzey[1], Burak Eren[1], Ibrahim Alatas[2], Erhan Emel[3], N.Serdar Bas[4], M. Hakan Seyithanoglu[5], Nezih Ozkan[6], Baris Sel[7]

ABSTRACT

Objective: To define risk factors affecting the outcome in traumatic subarachnoid hemorrhage.

Material and Methods: Forty-four patients with traumatic subarachnoid hemorrhage were evaluated retrospectively. They were divided into three groups according to their age: elderly (≥65 years), adult (16-64 years), and children (<16 years).

The clinical picture on admission was evaluated using the Glasgow Coma Scale. The patients were also divided into three groups according to their coma grading on admission: mild injury (Glasgow Coma Scale score 13-15), moderate injury (8-12), and severe injury (3-7). The amount of subarachnoid blood shown in computerized tomography was evaluated according to the Fisher index, and additional tomography findings were recorded. At last follow-up, presence of headache and neurological deficits as well as return to work or school were investigated, and the last clinical picture was evaluated with the Glasgow Outcome Scale.

Results: There were 11 children, 23 adults and 10 elderly patients. Twelve patients died between 1-49 days after trauma; the others were followed for a mean of 14.6 months (from 10 to 30 months).

In the children group, Glasgow Coma Scale score was significantly higher (p=0.004), subarachnoid blood amount was significantly lesser, and Glasgow Outcome Scale score was significantly better compared to the other groups.

For all groups, higher trauma severity on admission was associated with higher Fisher index (p=0.016). Most important factors affecting clinical results were severity of head injury on admission (p=0.0001), Fisher index (p=0.003), and presence of additional findings on computerized tomography (p=0.0001).

Conclusion: Traumatic subarachnoid hemorrhage usually has a good clinical outcome in children; however, in elderly patients, the outcome is worse, and there are usually additional intracranial traumatic lesions. Most important factors affecting outcome are blood amount on first computerized tomography, head trauma severity, and presence of additional intracranial traumatic lesions.

Keywords: Aged, head injuries, traumatic subarachnoid hemorrhage

ÖZET

Travmatik subaraknoid kanamada son durumu etkileyen faktörler

Amaç: Travmatik subaraknoid kanamada son durumu etkileyen faktörlerin belirlenmesi.

Yöntem ve Gereçler: Kırk dört travmatik subaraknoid kanamalı olgu retrospektif olarak değerlendirildi. Hastalar yaşlarına göre yaşlı (65 yaş ve üstü), erişkin (16-64 yaş) ve çocuk (<16 yaş) olarak üç gruba ayrıldı. Başvurudaki klinik tablo Glasgow Koma Skalası ile değerlendirildi. Buna göre olgular ayrıca 3 gruba ayrıldı: Hafif kafa travması (Glasgow Koma Skalası skoru 13-15), orta dereceli kafa travması (8-12) ve ağır kafa travması (3-7).

Bilgisayarlı tomografideki subaraknoid kanama miktarı Fisher indeksiyle değerlendirildi. Ek bulgular kaydedildi. Son izlemde baş ağrısı olup olmaması ve hastanın işe ya da okula dönüp dönmediği sorgulandı.

Bulgular: Olguların 11'i çocuk, 10'u yaşlı, 23'ü erişkin gruptaydı. Olguların 12'si 1-49 günler arasında öldü. Diğerleri ortalama 14,6 ay (10-30) izlendi.

Çocuk grubunda başvurudaki travma şiddeti anlamlı olarak daha iyi (p=0.004), subaraknoid kanama derecesi daha düşük ve klinik sonuçlar daha iyiydi.

Travma şiddeti artıkça kanama derecesi arttı (p=0.016). Klinik sonucu etkileyen en önemli faktörler başvurudaki travma şiddeti (p=0.0001), Fisher indeksi (p=0.003) ve tomografide ek bulgu olup olmaması (p=0.0001) idi. Son izlemde baş ağrısı olması ile Fisher indeksi arasında anlamlı bir ilişki bulundu (p=0.0001).

Sonuç: Travmatik subaraknoid kanama çocuk yaş grubunda genellikle iyi gidiş gösterirken, yaşlılarda subaraknoid kanamaya genellikle başka kafa içi lezyonlar da eşlik eder ve klinik gidiş kötüdür. Sonucu etkileyen önemli etkenler ilk BT'deki kanama miktarı, eşlik eden bulgu varlığı ve travmanın şiddetidir.

Anahtar kelimeler: Yaşlılık, kafa travması, travmatik subaraknoid kanama

[1]Bagcilar Training and Research Hospital, Neurosurgery Clinic, Istanbul, Turkey
[2]Bilim University, Florence Nightingale Hospital, Department of Neurosurgery, Istanbul, Turkey
[3]Bakirkoy Mazhar Osman Training and Research Hospital, Neurosurgery Clinic, Istanbul, Turkey
[4]Private Basari Hospital, Neurosurgery Clinic, Istanbul, Turkey
[5]Bezmialem University Faculty of Medicine, Department of Neurosurgery, Istanbul, Turkey
[6]Acibadem University, Faculty of Medicine, Department of Neurosurgery, Istanbul, Turkey
[7]Private Cevre Hospital, Neurosurgery Clinic, Istanbul, Turkey

Corresponding author:
Feyza Karagoz Guzey,
Topkapi Mahallesi, Kahal Basi Sokak, 46/1, Fatih 34093 Istanbul, Turkey
Phone: +90-212-440-4000
E-mail address: fkarag@yahoo.com

Date of submission: October 01, 2016
Date of acceptance: November 7, 2016

Introduction

In autopsy studies, the most frequent pathologic lesion in fatal head injuries is traumatic subarachnoid hemorrhage (tSAH) (1). Clinical studies reported that incidence of tSAH increased with severity of head injury. In severe head injury, it was shown in 12-39% of patients (2,3), whereas in mild head injury it was seen in 2-4% (4,5). Levi et al. (6) found presence of tSAH in 20% of patients with diffuse axonal injury. Although tSAH is rarely seen in mild head injury, Jeret et al. (7) reported that the most frequent computerized tomography (CT) finding in these patients was tSAH.

It is well known that the presence of tSAH causes significant worsening of the outcome of patients with head injury (2,8,9). This may be due to vasospasm after tSAH, or tSAH may be an important factor initiating secondary insult after trauma.

In this study, 44 consecutive patients with tSAH that had been treated and followed in our clinic were retrospectively evaluated to define risk factors causing worsening of clinical outcome.

Material and Methods

Patients and Groups

In this study, patients with tSAH that had been admitted to the emergency department within 24 hours after trauma and treated in the neurosurgery clinic or intensive care unit of our hospital were evaluated retrospectively. Patients with serious systemic multitrauma, patients whose CT examination within 24 hours after trauma was not available, patients whose follow-up period was shorter than 6 months, and patients whose hospital records were not adequate were excluded. A total of 44 patients met the inclusion criteria.

Scales

Neurological state on admission was evaluated using the Glasgow Coma Scale (GCS) (10) (Table 1), and at last follow-up, with the Glasgow Outcome Scale (GOS) (11) (Table 2). The patients were divided into 3 groups according to their GCS scores on admission: Mild head injury (GCS 13-15), moderate injury (GCS 8-12), and severe injury (GCS<8). In addition, patients were divided according to their ages into other 3 groups: children (<16 years), adults (16-64 years), and elderly (≥65 years). Clinical results after 6 months were categorized as good if GOS score was 1 and 2, and worse if it was grade 3, 4, or 5.

A case was considered as tSAH if there was localized or diffuse blood in the subarachnoid space. Tentorial hyperdensity was not accepted as tSAH if there was no accompanying blood in the adjacent subarachnoid space. tSAH was graded with the Fisher index (12) (Table 1). Accompanying CT findings such as epidural hematoma, subdural hematoma, intracerebral hematoma, contusions, or cerebral edema were also recorded.

Table 1: Fisher index

1	No subarachnoid blood;
2	Diffuse SAH, but thickness <1 mm, no hematoma;
3	SAH, thickness>1 mm;
4	SAH and intraventricular hemorrhage

At last follow-up, for adult patients return to work and for children return to school, respectively, and presence of headache were recorded. For a housewife, her capability to do her housework being similar to that before the trauma was accepted as return to work.

Statistical Evaluation

To compare nominal data, chi-square test or Fisher's exact test were used according to the number of the groups and for ordinary data Mann-Whitney test was applied. Results were accepted as significant if $p<0.05$.

Results

There were 13 female and 31 male patients with a mean age of 40.2 years (3 months to 79 years). There were 11 children, 23 adults, and 10 elderly patients. Female/male ratio was not significantly different in the three age groups (p=0.827) (Table 2).

On admission, 30 patients had mild, 9 had moderate, and 5 had severe head injury. The rate of mild injury was 90.9% in children, 30.4% in adults and 20% in the elderly (p=0.004) (Table 2). In accordance with these results, the Fisher index score was 2 in 81.8% of children, in 60.9% of adults, and in 40% of elderly patients. However, Fisher index scores were not significantly different in the three age groups (p=0.09) (Table 2). Trauma severity was not different between female and male patients (p=0.879). On the other hand, there was a significant relationship between Fisher index and clinical grade on admission (p=0.016) (Table 3).

There were additional CT findings in 12 patients: contusion in 8 cases, intracerebral hematoma in 5, subdural hematoma in

Table 2: Gender, head injury severity, Fisher index, and presence of accompanying lesions by age group

		<65 years (n/%)		16-64 years (n/%)		0-15 years (n/%)		p
Gender	Male	7	70%	17	73.9%	7	63.6%	χ^2:0.379
	Female	3	30%	6	26.1%	4	36.4%	p=0.827
Head Injury severity	Mild	2	20%	7	30.4%	10	90.9%	
	Moderate	4	40%	11	47.8%	1	9.1%	χ^2:15.16
	Severe	4	40%	5	21.7%	0	0.0%	***p=0.004***
Fisher Index	2	4	40%	14	60.9%	9	81.8%	χ^2:8.93
	3 and 4	6	60%	9	39.1%	2	18.2%	p=0.09
Additional findings	(-)	5	50%	19	82.6%	8	72.7%	χ^2:3.86
	(+)	5	50%	4	17.4%	3	27.3%	p=0.145
	Good	4	40%	18	78.3%	10	90.9%	
Clinical outcome	Worse	1	10%	0	0.0%	0	0.0%	χ^2:3.73
	Ex	5	50%	5	21.7%	1	9.1%	p=0.154
Complaints at fu	Headache (-)	2	50%	12	66.7%	10	100.0%	χ^2:9.18
	Headache (+)	2	50%	6	33.3%	0	0.0%	p=0.058

fu: follow-up; n: number
Significant p values are shown in bold and italic.

Table 3: Fisher index, presence of accompanying lesions, outcome and presence of headache at follow-up in various head injury severity groups

Head injury severity		Mild (n/%)		Moderate (n/%)		Severe (n/%)		p
Fisher Index	2	15	78.9%	10	62.5%	2	22.2%	χ^2:8,3
	3 and 4	4	21.1%	6	37.5%	7	77.8%	***p=0.016***
Additional lesions	(-)	15	78.9%	12	75.0%	5	55.6%	χ^2:1.75
	(+)	4	21.1%	4	25.0%	4	44.4%	p=0.417
Outcome	Good	18	94.7%	13	81.3%	1	11.1%	
	Worse		0.0%	1	6.3%		0.0%	χ^2:26.52
	Ex	1	5.3%	2	12.5%	8	88.9%	***p=0.0001***
Complaint at fu	Headache (-)	15	83.3%	9	69.2%		0.0%	χ^2:3.89
	Headache (+)	3	16.7%	4	30.8%	1	100.0%	p=0.142

fu: follow-up; n: number
Significant p values are shown in bold and italic.

3, epidural hematoma in 2, and subdural effusion and hydrocephalus in one each. The rate of additional CT findings did not differ between age groups (p=0.145) (Table 2); in addition, there was no significant relationship between presence of additional CT findings and head injury severity on admission (p=0.417) (Table 3). Only one of the cases with additional findings required surgery to evacuate a subdural hematoma.

Twelve cases died between 1 and 49 days after trauma during hospitalization. Only one of them was a child. The mortality rate was 9.1% in children, 21.7% in adults, and 50% in the elderly (p=0.154).

The other 32 cases were followed for a mean of 14.6 months (6 to 26 months). During follow-up, one of them died because of myocardial infarction 10 months later, his GOS score being 3 when he died. The other 31 cases had good results. Eight of them had headache at last follow-up, and the others were neurologically normal. All living patients returned to their work or school, including 8 adult patients with headache. Children's school performance was not different from their status before suffering the trauma, according to their parents.

There was a significant relationship between presence of headache at last follow-up and Fisher index on admission. Headache was significantly more frequent in patients with a Fisher index score of 3-4 than in those with a Fisher index of 2 (71.4 and 12%, respectively; p=0.0001). Age, gender, severity of head injury on admission, and presence or absence of additional

Table 4: Presence of accompanying lesions, outcome and presence of headache at follow-up in different Fisher index groups

		Fisher index 2 (n/%)		Fisher index 3/4 (n/%)		p
Accompanying lesions	(-)	26	96.3%	6	35.3%	χ^2:19.57
	(+)	1	3.7%	11	64.7%	***p=0.0001***
Outcome	Good	24	88.9%	8	47.1%	
	Worse	1	3.7%	0	0.0%	χ^2:11.79
	Ex	2	7.4%	9	52.9%	***p=0.003***
Complaint at fu	Headache (-)	22	88.0%	2	28.6%	χ^2:10.3
	Headache (+)	3	12.0%	5	71.4%	***p=0.0001***

fu: follow-up; n: number
Significant p values are shown in bold and italic.

findings on admission CT did not correlate to the presence of headache at last follow-up (p=0.058, 0.112, 0.142, and 0.99, respectively).

The most important factors affecting clinical outcome were GCS on admission (p=0.0001), Fisher index on first CT on admission (p=0.003), and presence/absence of additional CT findings (p=0.0001) (Tables 2, 3, and 4). Although good outcome (GOS 1 and 2) was more frequent in children, the difference was not significant. Age and gender did not significantly affect the outcome (p=0.154 and 0.229, respectively).

Discussion

Despite new developments of diagnosis and treatment modalities, the sequel rate of head injury is still high. In head injury patients, especially those with severe injury, functionally deprived, but anatomically saved many neural cells may return to normal function after trauma if ideal conditions are provided. According to Adams et al. (13), secondary damage is responsible for 40% of worse outcome after head injury. There are some studies evaluating risk factors causing secondary damage and affecting outcome. One of these factors is tSAH. Results of European head injury studies (HIT I and II) and the American Traumatic Coma Data Bank showed that presence of tSAH on first CT after trauma was related to a significant increase of the rate of worse outcome (2,8,9). Therefore, patients with tSAH were recorded as a separate category in HIT 3 and 4 (14). tSAH doubles the mortality rate in patients with head injury (14). These results led to the assumption that tSAH may be an important factor initiating a secondary damage process in the traumatized brain. This may be due to tSAH-induced vasospasm, electrolyte imbalance, hypothalamic dysfunction, or hydrocephalus (14). tSAH in basal cisterns may be an indicator of vertebrobasilar artery injury (15). In some cases, delayed deterioration was reported due to the evolution of tSAH into intracerebral hematoma because of the disruption of perforating arteries (16).

Large clinical trials on tSAH such as the HIT studies were related to especially severe head injury. Regarding tSAH in patients with mild and moderate injuries, there are conflicting results in the literature. Some studies found that a presence of tSAH on the first CT after trauma predicted a worse outcome (17-19). On the contrary, Demircivi et al. (4) and Otsuka et al. (20) reported that the presence of tSAH did not affect the outcome. In our study, GOS at last follow-up in patients with tSAH was significantly related with GCS on admission, and clinical results were usually good. Especially in pediatric patients, GCS on admission was high and GOS at last follow-up was very good. In addition, the presence of tSAH did not affect school performance in this patient group at follow-up.

It was reported in the literature that clinical outcome was directly related to the amount of blood in the subarachnoid space on the first CT after trauma: a larger amount of blood caused a worse outcome (8). In our study, it was also seen that an increase in the amount of blood caused a significantly worse outcome.

Another very important factor affecting the outcome in our study was the presence of additional findings on the first CT. Demircivi et al. (4) reported that the outcome was better in patients with tSAH when not accompanied by other lesions. In their series consisting of 13 cases, only one patient died, and others returned to their normal daily activities. However, in patients where other lesions accompanied tSAH, the outcome was worse, and 46% of these patients died. Otsuka et al. (20)

also divided their patients with tSAH into two groups, with and without accompanying lesions, and they found that the outcome was significantly better in the group with only tSAH.

It is well known that the amount of subarachnoid blood on the first CT after aneurysmal subarachnoid hemorrhage (aSAH) is directly related to secondary vasospasm and, therefore, to outcome (21,22). Weber et al. (23) saw a presence of vasospasm in 40% of patients with severe head injury. Sander and Klingelhöfer (22) found that in patients with tSAH, the degree of vasospasm was correlated with the amount of blood in the subarachnoid space, and therefore, they thought that vasospasm in tSAH may develop by a mechanism similar to that observed in aSAH. However, they did not find a strong correlation between degree of vasospasm and clinical outcome. Nevertheless, it is known that posttraumatic vasospasm may cause ischemic deficits (24). In our study, it was found, contrary to the study of Sander and Klingelhöfer, that the amount of subarachnoid blood according to the Fisher index was correlated to the clinical outcome, and a larger amount of blood caused a significantly worse outcome.

Some patients with tSAH may be admitted with only mild headache and meningeal irritation findings; however, some of them display very serious conditions (4,20). In our study, especially most of the pediatric patients (>90%) and about two thirds of the adults were admitted with a very small amount of blood in the subarachnoid space and with a high GCS. Clinical outcome was also very good in these patients. However, in the elderly group, most of the patients had a much larger amount of subarachnoid blood and poor GCS on admission, and subsequently a worse outcome at last follow-up. These observations are consistent with results of experimental and clinical studies reporting worse outcomes for head injury in the elderly (25-27), and this is probably not only related to tSAH, but also with all types of lesions in head injuries.

There are several evaluation scales to classify tSAH and other intracranial traumatic lesions in the literature, such as the Fisher index (12), the Marshall CT scan classification system (15), and the Rotterdam CT grading system (28). In our study, we used the Fisher index. This choice may be criticized because the Fisher index is an old system, and the Marshall system has usually been applied and advocated in the last years. We chose the Fisher index because of its simplicity and practicality. Besides, although the Marshall system has been very popular in the last years, it has some drawbacks, such as the basis cut-off of 25 cc as volume of mass lesion, because various intracranial hematomas have different cut-off values, and the classification of traumatic intracranial mass lesion as evacuated or non-evacuated, because this knowledge can only be obtained subsequently, during the patient's hospitalization (29). The Marshall system has been developed for all lesions from head injury; however, the Fisher index only categorizes the amount of subarachnoid blood. Therefore, it is a more proper tool for our study. The Rotterdam system also classifies all head injury lesions and is not proper for the design of our study.

Conclusion

Traumatic subarachnoid hemorrhage may have different courses in different age groups. Whereas in pediatric patients, pure tSAH with good clinical outcome is frequently seen, in the elderly, other intracranial traumatic lesions usually accompany tSAH, as most of the cases are admitted with severe head injury and clinical outcome is worse. Other important factors affecting outcome in tSAH are amount of subarachnoid blood on first CT, presence or absence of accompanying intracranial lesions, and severity of head injury, as in other traumatic intracranial lesions.

Contribution Categories	Name of Author
Development of study idea	F.K.G., E.E., N.O.
Methodological design of the study	F.K.G., E.E., N.O.
Data acquisition and process	B.E., S.B., H.S., I.A., B.S.
Data analysis and interpretation	B.E., S.B., H.S., I.A., B.S.
Literature review	F.K.G., B.E., S.B., H.S., I.A., B.S.
Manuscript writing	F.K.G., B.E., S.B., H.S.,
Manuscript review and revisation	E.E., N.O.

Conflict of Interest: Author declared no conflict of interest.

Financial Disclosure: Authors declared no financial support.

References

1. Freytag E. Autopsy findings in head injuries from blunt forces: statistical evaluation of 1367 cases. Arch Pathol 1963;75:402-413.
2. Eisenberg HM, Gary HE Jr, Aldrich EF, Saydjary C, Turner B, Foulkes MA, et al. Initial CT findings in 753 patients with severe head injury. A report from the NIH Traumatic Coma Data Bank. J Neurosurg 1990;73(5):688-698. [CrossRef]
3. Kobayashi S, Nakazawa S, Yokota H, Isayama K, Yano M, Otsuka T. Traumatic subarachnoid hemorrhage in acute severe head injury [Japanese]. No To Shinkei 1988;40(12):1131-1135.
4. Demirçivi F, Ozkan N, Büyükkeçeci, Yurt I, Miniksar F, Tektaş S. Traumatic subarachnoid haemorrhage: analysis of 89 cases. Acta Neurochir (Wien) 1993;122(1-2):45-48. [CrossRef]
5. Merino-deVillasante J, Taveras JM. Computerized tomography (CT) in acute head trauma. AJR Am J Roentgenol 1976;126(4):765-768. [CrossRef]
6. Levi L, Guilburd JN, Lemberger A, Soustiel JF, Feinsod M. Diffuse axonal injury: analysis of 100 patients with radiological signs. Neurosurgery 1990;27(3):429-432. [CrossRef]
7. Jeret JS, Mandell M, Anziska B, Lipitz M, Vilceus AP, Ware JA, et al. Clinical predictors of abnormality disclosed by computed tomography after mild head trauma. Neurosurgery 1993;32(1):9-16. [CrossRef]
8. European Study Group on Nimodipine in Severe Head Injury. A multicenter trial on the efficacy of nimodipine on outcome after severe head injury. J Neurosurg 1994;80(5):797-804. [CrossRef]
9. Kakarieka A, Braakman R, Schakel EH. Clinical significance of the finding of subarachnoid blood on CT scan after head injury. Acta Neurochir (Wien) 1994;129(1-2):1-5. [CrossRef]
10. Teasdale G, Jennett B. Assessment of coma and impaired consciousness. Lancet 1974;2(7872):81-84. [CrossRef]
11. Jennett B, Bond M. Assessment of outcome after severe brain damage. Lancet 1975;1(7905):480-484. [CrossRef]
12. Fisher CM, Kistler JP, Davis JM. Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computed tomographic scanning. Neurosurgery 1980;6(1):1-9. [CrossRef]
13. Adams JH, Graham DI, Gennarelli TA: Head injury in man and experimental animals: neuropathology. Acta Neurochir Suppl (Wien) 1983;32:15-30. [CrossRef]
14. Modi NJ, Agrawal M, Sinha VD. Post-traumatic subarachnoid hemorrhage: A review. Neurol India 2016;64(Suppl): S8-S13. [CrossRef]
15. Marshall LF, Marshall SB, Klauber MR, Clark MV. A new classification of head injury based on computerized tomography. J Neurosurg 1991;75(Suppl 1):S14-S20.
16. Hayashi T, Karibe H, Narisawa A, Kameyama M. Delayed deterioration in isolated traumatic subarachnoid hemorrhage. World Neurosurg 2016, 511.e9-14. [CrossRef]
17. Dacey RG Jr, Alves WM, Rimel RW, Winn HR, Jane JA. Neurosurgical complications after apparently minor head injury. J Neurosurg 1986;65(2):203-210. [CrossRef]
18. Stein SC, Ross SE. The value of computed tomographic scans in patients with low risk head injuries. Neurosurgery 1990;26(4):638-640. [CrossRef]
19. Williams DH, Levin HS, Eisenberg HM. Mild head injury classification. Neurosurgery 1990;27(3):422-428. [CrossRef]
20. Otsuka S, Nakatsu S, Sato S, Motozaki T, Ban S, Yamamoto T, et al. Study on cases of traumatic subarachnoid hemorrhage. Nihon Geka Hokan 1988;57(1):84-91.
21. Hijdra A, Brouwers PJ, Vermeulen M, van Gijn J. Grading the amount of blood on computed tomograms after subarachnoid hemorrhage. Stroke 1990;21(8):1156-1161. [CrossRef]
22. Sander D, Klingelhöfer J. Cerebral vasospasm following posttraumatic subarachnoid hemorrhage evaluated by transcranial Doppler ultrasonography. J Neurol Sci 1993;119(1):1-7. [CrossRef]
23. Weber M, Grolimund P, Seiler RW. Evaluation of posttraumatic cerebral blood flow velocities by transcranial Doppler ultrasonography. Neurosurgery 1990;27(1):106-112. [CrossRef]
24. Pasqualin A, Vivenza C, Rosta L, Licata C, Carazzani P, Da Pian R. Cerebral vasospasm after head injury. Neurosurgery 1984;15(6):855-858. [CrossRef]
25. Alberico AM, Ward JD, Choi SC, Marmarou A, Young HF. Outcome after severe head injury: relationship to mass lesions, diffuse injury, and ICP course in pediatric and adult patients. J Neurosurg 1987;67(5):648-656. [CrossRef]
26. Luerssen TG, Klauber MR, Marshall LF. Outcome from head injury related to patient's age. A longitudinal prospective study of adult and pediatric head injury. J Neurosurg 1988;68(3):409-416. [CrossRef]
27. Uslu E, Guzey FK, Oguz E, Guzey D. The effects of ageing on brain tissue sialic acid contents following cold trauma. Acta Neurochir (Wien) 2004;146(12):1337-1340. [CrossRef]
28. Maas AI, Hukkelhoven CW, Marshall LF, Steyerberg EW. Prediction of outcome in traumatic brain injury with computed tomographic characteristics: a comparison between the computed tomographic classification and combinations of computed tomographic predictors. Neurosurgery 2005;57(6):1173-1182. [CrossRef]
29. Deepika A, Prabhuraj AR, Saikia A, Shukla D. Comparison of predictability of Marshall and Rotterdam CT scan scoring system in determining early mortality after traumatic brain injury. Acta Neurochir (Wien) 2015;157(11):2033-2038. [CrossRef]