ORIGINAL ARTICLES

UDC: 616.832.94-005.1
DOI: https://doi.org/10.2298/VSP230208023S

Clinical and radiological characteristics of patients with spontaneous and post-traumatic subarachnoid hemorrhage: a retrospective observational study

Kliničke i radiološke karakteristike bolesnika sa spontanim i posttraumatskim subarahnoidalnim krvarenjem: retrospektivna opservaciona studija

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Abstract

Background/Aim. Several serious complications can accompany both spontaneous and post-traumatic subarachnoid hemorrhage (SAH) such as the development of intracranial hypertension, hydrocephalus, rebleeding, cerebral hypoxia, cerebral vasospasm, impaired pituitary function, electrolyte imbalance, and electrocardiographic (ECG) abnormalities. Although there is a declining trend in mortality, the treatment of SAH and its complications represents a challenge even in imposing neurosurgical centers. The aim of the study was to compare some clinical characteristics and complications between spontaneous and post-traumatic SAH. **Methods.** The retrospective study included 138 patients treated at the Department of Neurosurgery from January 2018 to January 2023. There were 71 patients with spontaneous and 67 patients with post-traumatic SAH. **Results.** A predominance of spontaneous SAH in female and post-traumatic SAH in male patients ($p < 0.001$) was found. There was a statistically significant difference in the frequency of hydrocephalus between groups of spontaneous and post-traumatic SAH patients ($p = 0.013$). Cerebral vasospasm was significantly more prevalent in patients with spontaneous SAH ($p < 0.001$). A statistically significant association was also obtained between the thickness of the coagulum in these two groups ($p < 0.001$). Patients with spontaneous SAH were significantly more likely to have a negative T wave in ECG findings ($p < 0.001$). Furthermore, there was no statistically significant difference regarding electrolyte imbalance in these two groups of patients with SAH. **Conclusion.** There were statistically significant differences between gender distribution, the frequency of abnormal ECG findings in the form of a negative T wave, greater coagulum thickness, vasospasm occurrence, and a higher rate of hydrocephalus in patients with spontaneous SAH compared to patients with post-traumatic SAH.

Key words:
brain injuries; electrocardiography; hydrocephalus; intracranial aneurysm; sex factors; subarachnoid hemorrhage; vasospasm intracranial.

Apstrakt

Uvod/Cilj. Kod bolesnika sa spontanim i posttraumatskim subarahnoidalnim krvarenjem (SAK), moguća je pojava teških komplikacija kao što su razvoj intrakranijalne hipertenzije, hidrocefalus, ponovno krvarenje, cerebralna hipoksija, cerebralni vazospazam, poremećena funkcija hipofize, poremećaj ravnoteže elektrolita i elektrokardiografske (EKG) abnormalnosti. Iako je trend mortaliteta u opadanju, lečenje bolesnika sa SAK i njenim komplikacijama predstavlja izazov čak i u velikim neurohirurškim centrima. Cilj rada bio je da se uporede učestalost nekih kliničkih karakteristika i komplikacija između bolesnika sa spontanim i posttraumatskim SAK. **Metode.** Retrospektivnom studijom obuhvaćeno je 138 bolesnika lečenih u Odeljenju za neurohirurgiju u periodu od januara 2018. do januara 2023. godine. U analizu je bio uključen 71 bolesnik sa spontanim i 67 bolesnika sa posttraumatskim SAK. **Rezultati.** Utvrđena je prevaga spontanog SAK kod žena, a posttraumatskog SAK kod muškaraca ($p < 0,001$). Utvrđena je statistički značajna razlika u učestalosti hidrocefalusa između bolesnika sa spontanim i posttraumatskim SAK ($p = 0,013$). Cerebralni vazospazam bio je značajno češći kod bolesnika sa spontanim SAK ($p < 0,001$). Utvrđena je statistički značajna povezanost u pogledu debljine koaguluma između te dve grupe ($p < 0,001$). Bolesnici

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sa spontanim SAK su značajno češće imali negativan T talas u EKG nalazu ($p < 0,001$). Takođe, nije bilo statistički značajne razlike u pogledu poremećaja ravnoteže elktrolita između te dve grupe bolesnika sa SAK. **Zaključak.** Rezultati našeg istraživanja ukazuju na postojanje statistički značajne razlike vezane za pol bolesnika, učestalost abnormalnog EKG nalaza u vidu negativnog T talasa, veće debljine koaguluma, pojave vazospazma i veće stope hidrocefalusa kod bolesnika sa spontanim SAK, u odnosu na bolesnike sa posttraumatskim SAK.

Ključne reči:
mozak, povrede; elektrokardiografija; hidrocefalus; aneurizma, intrakranijalna; pol, faktor; krvarenje, subarahnoidno; vazospazam, intrakranijalni.

Introduction

Nontraumatic subarachnoid hemorrhage (SAH) represents a type of hemorrhagic stroke that most often arises spontaneously due to aneurysm rupture, occurs through the penetration of blood into the subarachnoid space (Figure 1), and is thought to be responsible for about 3% of all strokes, with an estimated annual incidence of approximately 600,000 cases worldwide [1]. Spontaneous SAH affects a disproportionately large number of people under the age of 65 compared to ischemic stroke and represents a major burden on the healthcare system and society [2]. The development of delayed cerebral ischemia due to vasospasm (Figure 2) significantly worsens the prognosis of patients [3]. It is considered that most intracranial aneurysms are not congenital but develop gradually during life. Furthermore, it is estimated that an average of 2% to 3% of adults without risk factors have brain aneurysms, but this number increases proportionally with age [4]. The most common place where saccular aneurysms occur is the branches of the intracranial arteries of the base of the brain (Figure 3), especially in the area of the circle of Willis [4, 5]. Although most intracranial aneurysms will never rupture, the risk for hemorrhage increases with increasing aneurysm size, although paradoxically, most ruptured aneurysms are less than 1 cm in diameter [4].

In 10% of patients with spontaneous SAH, the cause cannot be detected, while a smaller (5%) number of cases arises due to other vascular pathology (arteriovenous malformations, arteriovenous fistula, reversible cerebral vasoconstriction syndrome) [6]. On the other hand, traumatic brain injuries (TBI) are the most common cause of SAH (Figure 4). Furthermore, post-traumatic SAH occurs in approximately 33–60% of cases of severe and moderate TBI,

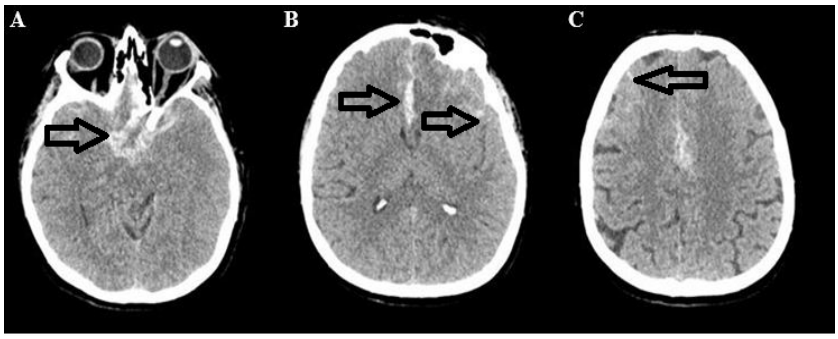

Fig. 1 – Non-contrast axial computed tomography indicates spontaneous subarachnoid hemorrhage (marked by arrows) extending into the basal and suprasellar cisterns (A), into the proximal Sylvian fissure and the anterior part of the interhemispheric fissure (B), reaching the right frontal convexity (C).

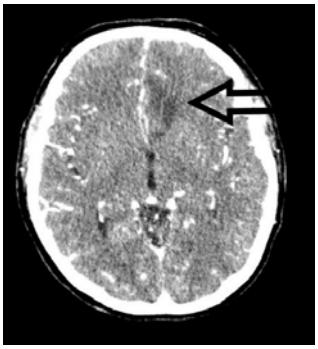

Fig. 2 – Computed tomography angiography in the axial plane demonstrates a subacute ischemic zone on the frontal parasagittal left (indicated by arrow) as a consequence of cerebral vasospasm.

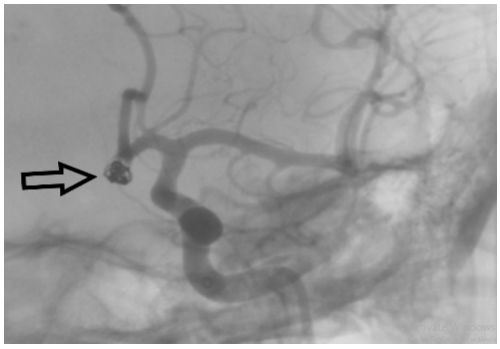

Fig. 3 – Digital subtraction angiogram shows an embolized bleeding aneurysm at the A1/A2 junction of the left anterior cerebral artery (indicated by arrow).

such as brain contusion and subdural hematoma [7]. The leading mechanisms of injury are injuries in traffic accidents, violence, and falls, while the patients are mostly males between 15 and 44 years of age. Brain trauma associated with post-traumatic SAH is a prognostic factor of poor outcomes in patients because it is associated with numerous complications [7, 8].

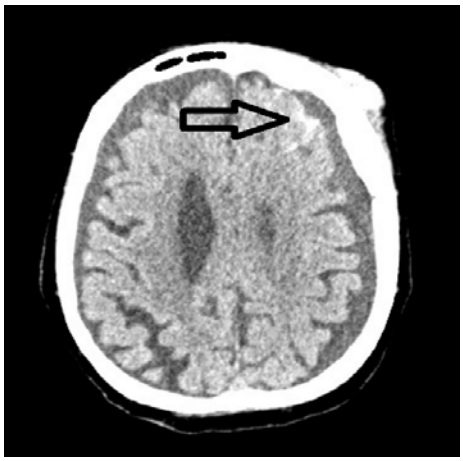

Fig. 4 – Non-contrast axial computed tomography demonstrates post-traumatic subarachnoid hemorrhage with cerebral contusion in the region of the left superior frontal gyrus (marked by arrow). A subcutaneous hematoma can be seen frontally on the left side.

Approximately 11% of patients with SAH die before receiving medical care, in addition to approximately 40% of patients who die within four weeks of admission to the hospital [9]. The mortality rate is estimated to be about 50% in the population, with a declining trend [10]. Moreover, several serious complications can accompany both spontaneous and post-traumatic SAH, such as the development of intracranial hypertension, hydrocephalus, re-bleeding (recurrence of SAH), cerebral hypoxia, cerebral vasospasm, impaired pituitary function, electrolyte imbalance, and electrocardiographic (ECG) abnormalities. Although there is a declining trend in mortality, the treatment of SAH and its complications represents a challenge even for experienced neurosurgical centers [7, 8, 10].

Methods

This retrospective study included 138 patients treated at the Department of Neurosurgery, University Clinical Center Niš, Serbia from January 2018 to January 2023.

The data processed in this research was collected from the available medical records. In all patients included, computed tomography (CT) with CT angiography (CTA) of the brain was performed initially upon admission, as well as control CT during hospitalization.

Based on the etiology of SAH, patients were divided into the following groups: Group I – patients with spontaneous SAH and Group II – patients with post-traumatic SAH.

According to the protocol of our Neurosurgery Department, the control CT scan was performed three days after the initial one or even earlier in cases of clinical deterioration of the patient. Furthermore, CTA was not performed in patients with chronic renal failure, allergy to iodinated contrast media, decompensated heart failure, and type 2 diabetes mellitus with metformin as regular therapy. Digital subtraction angiography (DSA) of the brain was performed in all of the patients with spontaneous SAH in order to exclude or confirm the presence of aneurysms, arteriovenous malformations, or other malformations of the brain blood vessels as a potential source of bleeding. In patients with spontaneous SAH and a positive CTA finding, DSA was urgently performed. Moreover, if the presence of a brain aneurysm was confirmed, subsequent endovascular embolization and stent graft placement or microsurgical clipping of the aneurysm was performed.

In patients with post-traumatic SAH, DSA was performed only in cases where there was a doubt about whether spontaneous SAH preceded head trauma and caused additional post-traumatic SAH, more precisely in six patients.

Follow-up brain CT reports were used to determine the presence of hydrocephalus and re-hemorrhage after SAH. The radiological characteristics of SAH were graded by using the modified Fisher scale (mFS), while independently of the mFS grade, the thickness of the coagulum was also measured, where a coagulum with a thickness of more than 1 mm was considered thick. Neurological status and clinical presentation of the patients were evaluated in order to suspect the presence of vasospasm after SAH. Consequently, CTA and DSA were performed to confirm vasospasm and were reviewed by radiologists and the neurosurgical council. The criteria for excluding patients from the research were the following: patients with incomplete documentation, patients with an initial brain CT performed at a local hospital that was not available for analysis, and patients in whom an associated brain injury was the dominant cause of the patient's clinical deterioration, such as traumatic epidural hematoma, subdural hematoma, and massive brain contusions. Besides, patients under 18 years of age were not included in our analysis.

Patients with aneurysmal SAH were treated after the neurosurgical and radiological intervention by maintaining the mean arterial pressure close to the upper limit of 100 mmHg, oral administration of nimodipine (60 mg every 4 hrs) or intravenously (2 mg/h), as well as with hemodynamic optimization.

A venous blood sample was taken from each patient upon admission. Routine biochemical parameters, such as sodium and potassium values, were determined immediately using standard biochemical methods on an AU680 Clinical Chemistry Analyzer (Beckman Coulter, Brea, CA, USA). The electrolyte reference values were for sodium 135–148 mmol/L and potassium 3.5–5.5 mmol/L.

Each patient underwent a standard ECG examination on admission with six precordial leads, which was interpreted by a cardiologist. The cardiologist assessed dynamic ST-

segment abnormalities, as well as the presence of negative T waves and abnormal U waves.

Statistical analysis

Data entry and tabulation were performed using the MS Office 2016 Excel program. Statistical calculations were performed with the program SPSS (IBM SPSS Statistics – version 23). Standard and basic statistical methods were used for qualitative and quantitative assessment of the results. The normality of the distribution of individual values was assessed by the Kolmogorov-Smirnov test. The Chi-squared (χ^2) test was used to test whether the distributions of categorical variables differ from each other. The statistical hypothesis was tested at the level of significance for the risk of $\alpha = 0.05$, i.e., the difference between the samples was considered significant if $p < 0.05$. The Mann-Whitney *U* test was used when the assumptions of the *t*-test were not met, and independence within the samples and mutual independence was assumed.

Results

In the whole group of patients, there were 78 male and 60 female patients. In the group of patients with spontaneous SAH, there were 46 (64.79%) females and 25 (35.22%) males, while in the group of patients with post-traumatic SAH, there were 14 (20.90%) female and 53 (79.10%) male patients. Our results indicated a statistically significant difference in the frequency of spontaneous and post-traumatic SAH according to gender (χ^2 statistic was 27.0247 and $p < 0.001$). The mean age of patients in the whole group was 61.53 ± 14.82 years. In the group of patients with spontaneous SAH, the mean age was 59.94 ± 11.48 years, while in the group of patients with post-traumatic SAH, it was 59.42 ± 20.45 years. There was no statistically significant age difference between patients with spontaneous and post-traumatic SAH (the *Z*-score was -1.79925 and $p = 0.072$) (Table 1).

The incidence of hydrocephalus in both groups of patients with SAH is shown in Table 2. There was a statistically significant difference in the frequency of

Table 1

Demographic characteristics of the patients with spontaneous and post-traumatic subarachnoid hemorrhage (SAH)

Variable	Spontaneous SAH	Post-traumatic SAH	*p*
Gender			
male	25 (35.22)	53 (79.10)	< 0.00001*
female	46 (64.79)	14 (20.90)	
Age (years)	59.94 ± 11.48	59.42 ± 20.45	0.072†

All values are expressed as numbers (percentages) or mean ± standard deviation.
***Chi-squared test; †Mann-Whitney *U*-test.**

Table 2

Clinical and radiological characteristics in patients with spontaneous and post-traumatic subarachnoid hemorrhage (SAH)

Variable	Spontaneous SAH n = 71	Post-traumatic SAH n = 67	*p**
Hydrocephalus			
with	19 (26.76)	6 (8.95)	0.013
without	52 (73.24)	61 (91.05)	
Recurrence of SAH			
yes	7 (9.86)	2 (2.99)	0.197
no	64 (90.14)	65 (97.01)	
Vasospasm			
with	17 (23.94)	1 (1.49)	0.00009
without	54 (76.06)	66 (98.51)	
Coagulum thickness			
thick	48 (67.59)	24 (35.82)	0.0002
thin	23 (32.39)	43 (64.18)	
mFS			
I	18 (25.35)	23 (34.32)	0.110
II	32 (45.07)	19 (28.36)	
III	14 (19.71)	21 (31.34)	
IV	7 (9.86)	4 (5.97)	

mFS – modified Fisher scale. All values are expressed as numbers (percentages).
***Chi-squared test.**

hydrocephalus between these two groups (χ^2 statistic with Yates correction was 6.2157 and $p = 0.013$). Regarding the incidence of re-bleeding in both groups of patients with SAH, it was found that there was no statistically significant difference in the frequency of re-bleeding between these two groups (χ^2 statistic with Yates correction was 1.6632 and $p = 0.197$). A statistically significant association was also determined between the thickness of the coagulum and spontaneous and post-traumatic SAH (χ^2 statistic was 13.9564 and $p = 0.0002$), where a thick coagulum was considered a coagulum with a thickness of 1 mm or more (Table 2).

The incidence of vasospasm was also considered in Table 2. Cerebral vasospasm was significantly more prevalent in patients with spontaneous SAH ($p = 0.00009$). On the other hand, there was no statistically significant association between the grades of the mFS and spontaneous and post-traumatic SAH (χ^2 statistic was 6.0308 and $p = 0.110$) (Table 2).

Initial mean values of serum sodium, the incidence of sodium level disturbances (hyper- or hyponatremia), and mean values of serum potassium, hypo- or hyperkalemia in both groups of examined SAH patients are shown in Table 3. There was no statistically significant difference regarding electrolyte imbalance in these two groups of patients with SAH. Elevation of the ST segment was observed in 5 (7.04%) patients with spontaneous and 1 (1.49%) with post-traumatic SAH, while ST depression was present in 7 (9.85%) patients with spontaneous and 1 (1.49 %) with post-traumatic SAH. No statistically significant difference was observed between the presence of abnormal U wave, elevation, and depression of the ST segment in two groups of patients with SAH ($p = 0.689$). Patients with spontaneous SAH were significantly more likely to have a negative T wave (χ^2 statistic was 12.9772 and $p = 0.0003$) (Table 3).

Discussion

According to the relevant scientific literature, the most common age of patients with ruptured aneurysms is between 40 and 65 years, which coincides with the results from our study [11, 12]. The mean age of patients with spontaneous SAH in our research was 59.94 ± 11.48 years, and in the group with post-traumatic SAH, it was 59.42 ± 20.45 years. The frequency of females in patients with spontaneous SAH was 1.8 times higher than in males, which is in accordance with the results of other studies where women were found to have about 1.7 times greater risk for spontaneous SAH than men, but this difference was evident only in patients older than 50 years [13]. A possible explanation for the higher incidence of spontaneous SAH in females after the age of 50 could be a decrease in the protective hormones estrogen and progesterone after reaching menopause [14–16]. Frontera et al. [15] found in their study of 580 patients with spontaneous SAH that 68% of patients were female, which corresponds to the results of our study. In contrast to the spontaneous SAH, the results of our study indicate that the frequency of men was significantly higher in post-traumatic SAH, which could be explained by the fact that men are more often involved in traffic accidents and more often suffer from serious injuries at work [11–16].

SAH is known to be associated with ECG abnormalities [17]. The reported prevalence of ECG changes in patients with SAH ranges from 27% to 100% [17]. Abnormal U wave is one of the most common abnormalities that occur in patients with SAH, with a frequency between 50–60%, and after that,

Table 3

Electrolyte and electrocardiographic abnormalities in patients with spontaneous and post-traumatic subarachnoid hemorrhage (SAH)

Variable	Spontaneous SAH	Post-traumatic SAH	*p*
Sodium values			
serum natrium disorders	135.79 ± 6.84	136.42 ± 3.24	0.072†
hypernatremia	2 (2.81)	1 (1.49)	0.917*
hyponatremia	28 (39.43)	6 (8.95)	
Potassium values			
serum potassium disorder	3.40 ± 0.71	4.03 ± 0.56	0.960†
hyperkalemia	2 (2.82)	1 (1.49)	0.865*
hypokalemia	15 (21.12)	6 (8.95)	
ST segment changes			
ST elevation	5 (7.04)	1 (1.49)	
ST depression	7 (9.85)	1 (1.49)	0.689*
Abnormal U wave			
T wave shapes	36 (50.70)	12 (17.91)	
negative T wave	26 (36.62)	7 (10.48)	0.0003*
normal T wave	45 (63.38)	60 (89.52)	

All values are expressed as numbers (percentages) or mean ± standard deviation.
***Chi-squared test; †Mann-Whitney *U*-test.**

changes in ST segment, T wave, and QT interval are found in about 50% of patients [17, 18]. Our results indicate that patients with spontaneous SAH were significantly more likely to have a negative T wave. The higher occurrence of negative T waves in patients with spontaneous SAH compared to the patients with post-traumatic SAH may be associated with electrolyte disturbances, especially low potassium levels, which was also observed in our study in patients after the spontaneous SAH. Electrolyte disturbances, particularly low serum potassium, are thought to cause ECG abnormalities [19, 20]. Some authors emphasize that the time when the ECG is performed in patients with SAH is of critical importance, bearing in mind that during the first 72 hrs, the most pronounced changes on the ECG could be seen in these patients [21, 22].

Moreover, SAH is also associated with electrolyte imbalance and homeostasis disturbances, such as hyponatremia occurring in 10–34% of patients with SAH [23]. Presumably, it is caused by hypothalamic dysfunction, most often due to sodium loss influenced by an increase in the concentration of brain natriuretic peptides [24, 25].

In our study, there was no statistically significant difference regarding electrolyte imbalance between post-traumatic and spontaneous SAH groups. The frequency of hyponatremia in our study coincides with some previously published research [24, 26, 27]. Several conducted studies have suggested that cerebral salt-wasting syndrome and inappropriate antidiuretic hormone syndrome are the most common causes of hyponatremia after SAH [28, 29]. Elevated brain natriuretic peptide and atrial natriuretic peptide levels after SAH are thought to lead to natriuresis causing hyponatremia [30]. Furthermore, a vasodilator peptide, adrenomedullin, is secreted into the cerebrospinal fluid from the choroid plexus and may have a natriuretic effect on the kidneys [26].

In addition to hyponatremia, in our study, there were 15 (21.12%) patients with hypokalemia in spontaneous and 6 (8.95%) patients in the post-traumatic SAH group. There was no statistically significant difference in the frequency of hypokalemia between the groups of patients with spontaneous and post-traumatic SAH. Hypokalemia is thought to occur due to the release of catecholamines after the onset of SAH. High levels of circulating catecholamines lead to excessive activation of Na/K-ATPase by stimulating beta-adrenergic receptors. This results in the displacement of the potassium ions into the intracellular space [31, 32].

Cerebral vasospasm usually occurs on the third day after SAH reaches a peak frequency between the sixth and eighth day and lasts for two to three weeks. Cerebral vasospasm can lead to decreased cerebral blood flow and impaired oxygen supply to the brain, which can cause cerebral ischemia and infarction in a number of patients [4, 33]. The incidence of cerebral vasospasm remains unknown; it is ascribed to great difficulties in reporting incidence data, both due to difficulties in diagnosing and defining the cerebral vasospasm, and ranges from 1.5% to 91%, which corresponds to the results we obtained [34, 35]. In addition, the factor that affects the occurrence of vasospasm the most is the amount and localization of SAH on brain CT in the first four days after bleeding, regardless of the way the ruptured aneurysm was previously treated [36]. The occurrence of vasospasm is significantly correlated with the amount of blood in the subarachnoid space. According to the results we obtained, cerebral vasospasm was significantly more prevalent in patients with spontaneous SAH. Vasospasm occurs most frequently and most intensively in the vicinity of a bleeding aneurysm, primarily due to the amount of blood in the proximity of the ruptured aneurysm. In patients with SAH of the non-aneurysmal etiology, the occurrence of vasospasm is significantly less frequent, which may explain the low occurrence of vasospasm in patients after traumatic SAH in our study [37].

The Fischer scale was established in 1980, assuming it was sufficient to predict the risk of developing cerebral vasospasm. This assumption was confirmed in a small sample (41 patients) in 1983 [38]. According to one study, which had a significantly higher number of patients, this correlation existed but was not statistically significant [39]. On the other hand, the mFS scale has wide application and enormous clinical significance as a grading system that correlates with vasospasm based on the amount and localization of SAH [40]. However, certain limitations were also observed with mFS in terms of different interpretations and scoring by clinical doctors, and it is considered by some authors that the criteria of this scale must be clearer [40, 41].

How the blood affects the appearance of vasospasm has not been completely elucidated. Theories that try to explain the mechanism of vasospasm are based on the release of vasoactive substances during the breakdown of blood. The amount and extent of SAH predict symptomatic vasospasm and delayed cerebral ischemia as a consequence of hemolysis, leading to inflammation, endothelial injury, and the release of oxygen-free radicals, which lead to vasoconstriction and promote it [42]. Contemporary research is focused on discovering new drugs that would be more effective in the treatment of vasospasm. Endothelin receptor antagonists inhibit the action of the vasoconstrictor endothelin-1, and it has been shown that endothelins play an important role in the development of cerebral vasospasm [43, 44]. On the other hand, the nonglucocorticoid 21-aminosteroid tirilazad inhibits lipid peroxidation and has a neuroprotective effect with antioxidant effects [45]. The neuroprotective effect of erythropoietin in maintaining vascular autoregulation was also investigated, which could have implications in the treatment of vasospasm after SAH [46].

Acute hydrocephalus occurs in *circa* 20% of patients with SAH [47, 48]. Hydrocephalus resolves spontaneously within 24 hrs in 30% of patients but may worsen and quickly lead to a fatal outcome [49]. The results of our research indicate that the incidence of hydrocephalus in the group with spontaneous SAH was 19 (26.76%) of 71 patients, while in the group with post-traumatic SAH, it was 6 (8.95%) of 67 patients. There was a statistically significant difference in the frequency of hydrocephalus between these two groups ($p = 0.013$). We obtained a statistically significant association between the thickness of the coagulum and spontaneous

and post-traumatic SAH ($p < 0.001$), where a thick coagulum was considered a coagulum with a thickness of 1 mm or more. In addition, thicker coagulum is more often observed after aneurysmal SAH, and in patients with a higher mFS score as a consequence of a larger amount of blood and basal localization, they lead to obstruction of the cerebrospinal fluid pathways and basal cisterns [50–52]. The results of our study are consistent with the results of some previous studies, indicating that acute hydrocephalus occurs in 15% to 58.4%, and chronic hydrocephalus develops in 4.3% to 37% after aneurysmal spontaneous SAH [50–53]. Acute hydrocephalus is most often caused by blood clots in the aqueduct of Sylvius, openings of the fourth ventricle, as well as in the subarachnoid basal cisterns, which obstruct the flow of cerebrospinal fluid [50, 54]. Chronic hydrocephalus can occur later and is caused by the formation of adhesions between the pia and arachnoid mater, which also obstructs the flow of cerebrospinal fluid [50, 51].

Should the patient survive the initial SAH, the most dangerous early complication is re-bleeding from a previously ruptured aneurysm. The frequency of this complication ranges from 8% to 23% in the first 72 hrs after the initial SAH [55]. Studies have shown that about 50% to 90% of re-bleeding episodes arise within the first 6 hrs after the primary rupture [56, 57]. The consequences of re-bleeding are serious, with a reported mortality rate of 20 to 60 [58, 59]. There was no statistically significant difference in the frequency of re-hemorrhage between the groups of spontaneous and post-traumatic SAH in our research ($p = 0.197$). In our study, the frequency of re-hemorrhage in both groups of patients was in accordance with the data from the relevant scientific literature [60–62]. The mechanism of re-bleeding is complex and influenced by many factors. Moreover, several risk factors for re-bleeding have been identified. Beck et al. [60] pointed out that large and multiple aneurysms were associated with re-hemorrhage after SAH. Large aneurysms have a more fragile wall, which explains the increased risk for re-hemorrhage. Ohkuma et al. [57] indicated that elevated systolic blood pressure (> 160 mmHg) on admission was associated with re-bleeding. Poor clinical status (e.g., high Hunt and Hess score) on admission is associated with an increased risk of re-bleeding [59, 62].

Limitations of the study

A limitation of our study is that we did not include risk factors and comorbidities that may have been associated with re-bleeding, so we do not have an adequate explanation for the results presented here.

Conclusion

There were statistically significant differences between gender distribution, the frequency of abnormal ECG findings in the form of a negative T wave, greater coagulum thickness, vasospasm occurrence, and a higher rate of hydrocephalus in patients with spontaneous SAH compared to patients with post-traumatic SAH. Additional and more comprehensive prospective studies with larger groups of patients are needed to confirm the strength of the evidence presented here.

Conflict of interest

The authors declare no conflict of interest.

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Received on February 8, 2023
Revised on March 19, 2023
Revised on March 24, 2023
Accepted on April 4, 2023
Online First April 2023