Journal of Experimental and Clinical Medicine
https://dergipark.org.tr/omujecm

Research Article

J Exp Clin Med
2024; 41(1): 177-184
doi: 10.52142/omujecm.41.1.30

Aneurysmal subarachnoid haemorrhage: A retrospective study of epidemiological, clinical and radiological findings

Halit ŞENSOY[1], Recai ENGİN [2]*, Fatih TOMAKİN [3], Durmuş Emre KARATOPRAK [2] Bora TETİK [4]

[1]Department of Neurosurgery, Ministry of Health Gaziantep City Hospital, Gaziantep, Türkiye
[2]Department of Neurosurgery, Kahramanmaraş Necip Fazıl City Hospital, Kahramanmaraş, Türkiye
[3]Department of Neurosurgery, Unye State Hospital, Ordu, Türkiye
[4]Department of Neurosurgery, Faculty of Medicine, Inönü University, Malatya, Türkiye

Received: 08.02.2024 • **Accepted/Published Online:** 18.03.2024 • **Final Version:** 29.03.2024

Abstract

In our study, we aimed to evaluate the data obtained by retrospective examination of the epidemiological, clinical, and radiological findings of the patients with aneurysmal subarachnoid hemorrhage. A group of 589 patients with spontaneous subarachnoid hemorrhage was evaluated retrospectively. The epidemiological, clinical, and radiological results of the patients were analyzed. It was found that the most common presentation to the emergency department with spontaneous subarachnoid hemorrhage was in the 6th decade, the female-to-male sex ratio was 1.5/1, the most common presentation time was winter-spring seasons, especially December, and the mortality rate was 39%. Headache was the most common presenting complaint. The epileptic seizure and loss of consciousness affected the prognosis negatively and increased mortality significantly. Hypertension was the most common chronic disease and was found to increase mortality. In FISHER and WFNS classifications, mortality increased as grade and class progressed. Spontaneous SAH and SA is a life-threatening pathology with high morbidity and mortality, difficult to treat and follow-up and significantly increasing healthcare costs.

Keywords: spontaneous subarachnoid hemorrhage, cerebral aneurysm, epidemiology

1. Introduction

Hemorrhage into the subarachnoid space of the brain from arterial or venous vessels is called subarachnoid hemorrhage (SAH) (1). It accounts for 6-8% of hemorrhagic stroke cases. While the most common cause of SAH is trauma, the most common cause of spontaneous SAH is aneurysmatic hemorrhage (75-80%). Each year, 6-28 aneurysmal SAHs occur in approximately 100,000 people (2), 10% of whom die within the first hours of hemorrhage and 25% die within the first 24 hours (3). 50% of the patients die within the first 3 months and the remaining 50% have deficits (4). Despite successful surgical intervention, patients' personal, social, and professional lives are significantly affected and they cannot reach their previous physical performance (5). Subarachnoid hemorrhages are mostly caused by trauma (6). Spontaneous subarachnoid hemorrhages are most commonly caused by rupture of aneurysms in intracerebral vascular structures, but may also develop due to other vascular malformations (7). However, no pathology is detected in approximately 15-20% of spontaneous SAHs. There are many risk factors for spontaneous subarachnoid hemorrhage (8). Female gender, Japanese or Finnish nationality, and possibly African-American or Hispanic ethnicity are non-modifiable risks for spontaneous SAH (9). Hypertension, smoking, and heavy alcohol use are modifiable risks for spontaneous SAH (10). The most prominent clinical symptom of acute spontaneous SAH is a sudden onset of severe headache. It is often described as the most severe headache in the patient's life. Both the severity and the acute onset of headache are highly characteristic features of spontaneous SAH. In addition to headache, symptoms such as nausea-vomiting, epileptic seizure, altered consciousness, and photophobia may accompany (11).

The primary aim in patients presenting with spontaneous subarachnoid hemorrhage is to stabilize the respiratory and circulatory mechanisms. Then, the cause of spontaneous SAH should be determined by examination and imaging methods and the diagnosed pathology should be treated as soon as possible by surgical or endovascular intervention. The choice of surgical or endovascular intervention treatment for aneurysmal spontaneous SAHs is based on parameters such as the patient's age, clinical condition, location, size, and shape of the aneurysm. After the treatment of the existing pathology, follow-up and treatment of complications such as vasospasm, hydrocephalus and hemodynamic disturbance in intensive care conditions are also very important in terms of the prognosis and clinical course of spontaneous SAH (12).

The most preferred initial diagnostic method for spontaneous SAHs to demonstrate hemorrhage in the subarachnoid space is computed tomography (CT) of the brain without intravenous

*Correspondence: r.engin5552@gmail.com

contrast material. CT reveals hemorrhage as a hyperdense signal in the basal cisterns and ventricular system, if present. CT performed within the first 12 hours after acute spontaneous SAH has a sensitivity and specificity of 98-100% for the detection of blood in the subarachnoid space (13).

Magnetic resonance imaging (MRI) and lumbar puncture (LP) are also included in the diagnostic methods for spontaneous SAHs in addition to CT. Although MR angiography and CT angiography are also used to detect vascular pathologies in the differential diagnosis, the gold standard method is accepted as Digital Subtraction Angiography (DSA) (14).

While aneurysmal features such as location, size, and morphology govern the hemorrhage tendency of the intracranial aneurysm (15), the outcome following aneurysm rupture is determined by factors including the age of the patient and the severity of hemorrhage (16). The severity of ASAH is assessed by a series of grading scales that provide an objective measurement of the clinical and radiological status of the patient at presentation. In routine clinical practice, ASAH-specific indices are used in making treatment decisions and prognostic evaluation (17). One of these is the Glasgow Coma Scale (18).

In our study, we retrospectively analyzed the patients who presented to the emergency department with spontaneous SAH and whose radiological imaging and DSA results showed the presence of an intracerebral aneurysm. It is aimed to evaluate the epidemiological and demographic characteristics of the patients, the location of the aneurysm, the Glasgow Coma Scale (GCS), duration of hospital stay, clinical course and prognosis, and the treatment methods applied in comparison with the data in national and international literature.

2. Materials and Methods

2.1. Research Group

In our study, 998 patients who came to the emergency department of Inonu University Turgut Özal Medical Centre with subarachnoid hemorrhage between 2011 and 2020 were scanned from the hospital data system. We excluded 408 patients with traumatic SAH, Digital Subtraction Angiography (DSA) with no pathology, Arteriovenous Malformation (AVM) or subarachnoid hemorrhage caused by other causes mentioned in the etiology. In our study, a group of 590 patients who came to the emergency department with spontaneous subarachnoid hemorrhage and whose DSA results revealed cerebral aneurysm were retrospectively evaluated.

2.2. Data Collection

The data was obtained from the ENLIL system, the data system of Inonu University Turgut Özal Medical Centre, and the PACS programme, the radiological imaging programme. The following data were analysed: age at presentation, female-male sex ratios, months and seasons of presentation, complaints and clinical findings at presentation, known concomitant chronic diseases, GCS values at presentation, distribution according to FISHER and WFNS classifications, prognosis, length of hospital stay, and treatment modalities in patients who presented to our emergency department with spontaneous SAH. Male-female gender ratios and their effects on mortality were investigated. In addition, the locations, sizes, and shapes of the aneurysms detected after DSA were evaluated. In patients with aneurysms, the presence of multiple aneurysms, its relation with mortality and female-male sex ratios were evaluated. In patients in whom DSA was not performed, CT angiography or MR angiography findings were utilized.

2.3. Statistical Analysis

The data were analysed using SPSS 21.0 software. Mean, standard deviation, and percentage distributions were given as descriptive statistics. Chi-square analysis was used to determine the relationship between categorical variables and Mann Whitney U test was used to determine whether there was a difference in Time until interventional treatment is applied according to two-parameter variables. The results were evaluated at a 95% ($p<0.05$) significance level

3. Results

In this study, we retrospectively analyzed 589 patients who presented to the emergency department of our hospital with non-traumatic spontaneous SAH in the last 10 years and were found to have a cerebral aneurysm as a result of DSA.

The ages of the patients who came to the emergency department with spontaneous SAH ranged between 6-91 years and the mean age was 56.29±15.13 years. 28.4% of the patients were in the 6th decade and 0.2% (n = 1) were in the 1st decade. Female patients constituted 60.4% of the patients included in the study. 10.2% of the patients came to the emergency department in November and 7.1% in October. In this study, the incidence of patients who presented to the emergency department with spontaneous SAH and had cerebral aneurysm as a result of DSA was analyzed according to the aneurysm shape as saccular, fusiform, and dissecting aneurysms. The most common shape of cerebral aneurysms as a result of DSA was a saccular aneurysm in 96.6% (569 patients). The fusiform aneurysm was found in 2% (12 patients) and dissecting aneurysm in 0.3% (2 patients) of the patients. In the study group of 590 patients, the rate of patients with spontaneous saccular aneurysm detected on CT but who could not undergo DSA for various reasons was 1% (6 patients). The aneurysm size varied between 2-35 mm and the mean aneurysm size was 7.02±4.04 mm. Comorbidity was present in 60.4% of patients. Hypertension was present in 54.3%, coronary artery disease in 12.1%, Diabetes mellitus in 11.2%, and asthma in 2.9% of the patients. Surgery was performed in 52.6% and coil embolization in 36.8% of the patients. The hospitalization period of the patients ranged between 1-152 days and the mean hospitalization period was 13.95±14.90 days. Multiple aneurysms were present in 20.5% of patients. The mortality rate was 39% in the patient group included in the study (Table 1).

Table 1. Demographic and clinical characteristics of the participants

Demographic characteristics	
Age (x̄±SD)	56.29±15.13
1. Decade	1 (0.2%)
2. Decade	10 (1.7%)
3. Decade	14 (2.4%)
4. Decade	58 (9.8%)
5. Decade	116 (19.7%)
6. Decade	167 (28.4%)
7. Decade	120 (20.4%)
8. Decade	76 (12.9%)
9. Decade	24 (4.1%)
10. Decade	3 (0.5%)
Gender	
Female	356 (60.4%)
Male	233 (39.6%)
Month	
January	52 (8.8%)
February	51 (8.7%)
March	51 (8.7%)
April	44 (7.5%)
May	46 (7.8%)
June	44 (7.5%)
July	59 (10%)
August	46 (7.8%)
September	49 (8.3%)
October	42 (7.1)%
November	45 (7.6%)
December	60 (10.2%)
Aneurysm Type	
Saccular	569 (96.6%)
Fusiform	12 (2.0%)
Dissectant	2 (0.3%)
Digital Subtraction Angiography None	6 (1%)
Aneurysm Size (x̄±SD)	7.02±4.04
Comorbidity	
No	233 (39.6%)
Yes	356 (60.4%)
Hypertension	
No	269 (45.7%)
Yes	320 (54.3%)
Diabetes mellitus	
No	523 (88.8%)
Yes	66 (11.2%)
Asthma	
No	572 (97.1%)
Yes	17 (2.9%)
Coronary Artery Disease	
No	518 (87.9%)
Yes	71 (12.1%)
Treatment	
Surgery	310 (52.6%)
Coil	217 (36.8%)
Coil+Surgery	1 (0.2%)
Coil+Stents	1 (0.2%)
Follow-up	52 (8.8%)
Follow-Up+Extraventricular Drainage	2 (0.3%)
Rejection	6 (1%)
Hospitalization Duration (x̄±SD)	13.95±14.90
Multipl Aneurysm	
No	468 (79.5%)
Yes	121 (20.5%)
Prognosis	
Exitus	230 (39%)
Discharged	359 (61%)

As a result of our study, it was observed that the highest mortality rate was in the 10th decade with a rate of 100%, followed by the 9th decade with a rate of 79.2%, the 8th decade with a rate of 64.5%, and the 2nd decade with a rate of 60%, and the relationship between decade and mortality was found to be significant (Table 2).

Table 2. The relationship between decade and the prognosis

Decade	Survival	Mortality	p
1st Decade	100% (n = 1)	0% (n = 0)	.000
2nd Decade	40% (n =4)	60% (n = 6)	
3rd Decade	78,6% (n = 11)	21,4% (n = 3)	
4th Decade	75,9% (n = 44)	24,1% (n = 14)	
5th Decade	81% (n = 94)	19% (n = 22)	
6th Decade	65,9% (n = 110)	34,1% (n = 57)	
7th Decade	52,5% (n = 63)	47,5% (n = 57)	
8th Decade	35,5% (n = 27)	64,5% (n = 49)	
9th Decade	20,8% (n = 5)	79,2% (n = 19)	
10th Decade	0% (n = 0)	100% (n = 3)	

The aneurysm was located in the anterior communicating artery (A.Com) in 177 patients (30.2%), in the right arteria cerebri media in 20.4%, in the right arteria carotis interna in 11.4%, and in the left ophthalmic artery in 0.2% (n = 1). The relationship between SA location and mortality was also analysed. Accordingly, in the group with more than 1 patient, the localisation with the highest mortality rate was Arteria Comminican Posterior with a rate of 61.9% (13 patients). The localisation with the lowest mortality rate was Left Arteria Cerebellaris Posterior Inferior with a rate of 27.3% (12 patients) and the relationship between the localisation of the aneurysm and prognosis was found to be significant ($p<0.05$) (Table 3).

Table 3. Relationship between survival-mortality rates according to aneurysm location

Localization of the Aneurysm	Survival	Mortality	P
Anterior Comminican Artery	50,3% (n = 89)	49,7% (n = 78)	.016
Left Middle Cerebral Artery	66,2% (n = 43)	33,8% (n = 22)	
Right Middle Cerebral Artery	67,5% (n = 81)	32,5% (n = 39)	
Left Internal Carotid Artery+Basilar	72,7% (n = 32)	27,3% (n = 12)	
Right Internal Carotid Artery	65,7% (n = 44)	34,3% (n = 23)	
Left Anterior Cerebral Artery	63% (n = 17)	37% (n = 10)	
Right Anterior Cerebral Artery	59,1% (n = 13)	40,9% (n = 9)	
Posterior Communicate Artery	38,1% (n = 8)	61,9% (n = 13)	
Basilar Artery	71% (n =22)	29% (n = 9)	
Left Vertebral Artery	0% (n = 0)	100% (n = 1)	
Left Posterior Inferior Cerebellar Artery	72,7% (n = 8)	27,3% (n = 3)	
Left Ophthalmic Artery	100% (n = 1)	0% (n = 0)	

In patients with FISHER grade 2, the survival rate was 87.3% (151 patients) and the mortality rate was 12.7% (22 patients). In patients with FISHER grade 3, the survival rate was 66.3% (187 patients) and the mortality rate was 33.7% (95 patients). In patients with FISHER grade 4, the survival rate was 15.7% (21 patients) and the mortality rate was 84.3% (113 patients). As seen in the study, it was observed that the mortality rate increased from FISHER grade 1 to 4 (Table 4).

In patients with WFNS class 1, the survival rate was 87.3% (185 patients) and the mortality rate was 12.7% (27 patients). In patients with WFNS class 2, the survival rate was 83.3% (75 patients) and the mortality rate was 16.7% (15 patients). In patients with WFNS class 3, the survival rate was 72.1% (44 patients) and the mortality rate was 27.9% (17 patients). In patients with WFNS class 4, the survival rate was 32.4% (47 patients) and the mortality rate was 67.6% (98 patients). In patients with WFNS class 5, the survival rate was 9.9% (8 patients) and the mortality rate was 90.1% (73 patients). As seen in the study, it was observed that the mortality rate increased from WFNS class 1 to 5 (Table 4).

While the survival rate was 11.1% (4 patients) in patients with GCS 3, the mortality rate was 88.9% (32 patients). The survival rate was 87.3% (185 patients) and the mortality rate was 12.7% (27 patients) in patients with GCS 15. The survival rate was 83% (93 patients) and the mortality rate was 17% (19 patients) in patients with GCS 14. In patients with GCS 7 and below, the survival rate was 11.5% (13 patients) and the mortality rate was 88.5% (100 patients). In patients with GCS above 7, the survival rate was 73.2% (356 patients) and the mortality rate was 26.8% (130 patients) (Table 4).

Table 4. FISHER grade-mortality relationship in spontaneous SAH

	Survival	Mortality	p
FISHER			
Grade 2	87,3% (n = 151)	12,7% (n =22)	
Grade 3	66,3% (n = 187)	33,7% (n = 95)	.000
Grade 4	15,7% (n = 21)	84,3% (n =113)	
WFNS			
Class 1	87,3% (n = 185)	12,7% (n =27)	
Class 2	83,3% (n = 75)	16,7% (n = 15)	
Class 3	72,1% (n = 44)	27,9% (n =17)	.000
Class 4	32,4% (n = 47)	67,6% (n = 98)	
Class 5	9,9% (n = 8)	90,1% (n = 73)	
GCS			
15	87,3% (n = 185)	12,7% (n =27)	
14	83% (n = 93)	17% (n = 19)	
13	65,8% (n = 25)	34,2% (n = 13)	
12	45,5% (n = 5)	54,5% (n = 6)	
11	45,5% (n = 20)	54,5% (n = 24)	
10	25% (n = 3)	75% (n = 9)	
9	30,4% (n = 14)	69,4% (n = 32)	.0000
8	100% (n = 1)	0% (n = 0)	
7	15,6% (n = 5)	84,4% (n = 27)	
6	0% (n = 0)	100% (n = 10)	
5	10,3% (n = 3)	89,7% (n = 26)	
4	16,7% (n = 1)	83,3% (n = 5)	
3	11,1% (n = 4)	88,9% (n = 32)	

While 49.4% (158 patients) of the patients with a diagnosis of hypertension who came to the emergency department with spontaneous SAH were discharged, the mortality rate was 50.6% (162 patients). It was observed that 50% (33 patients) of the patients with diabetes were discharged, while the mortality rate was 50% (33 patients). While 82.4% (14 patients) of the patients with asthma were discharged, the mortality rate was 17.6% (3 patients). In the patient group with chronic arterial disease, 46.5% (33 patients) were discharged, while the mortality rate was 53.5% (38 patients) (Table 5).

The mortality rate was 47.1% (57 patients) in patients with multiple aneurysms and 37% (173 patients) in patients with a single aneurysm ($p<0.05$) (Table 5).

Table 5. Chronic disease-mortality relationship in spontaneous SAH

	Survival	Mortality	P
Chronic Disease			
Hypertension	49,4% (n = 158)	50,6% (n = 162)	.000
Coronary Artery Disease	46,5% (n = 33)	53,5 (n = 38)	.008
Diabetes mellitus	50% (n = 33)	50% (n = 33)	.053
Asthma	82,4% (n = 14)	17,6% (n = 3)	.066
Multiple Aneurysm			
Yes	52,9% (n = 64)	47,1% (n = 57)	.042
No	63% (n = 295)	37% (n = 173)	

The mean time until interventional treatment was 32.42±59.50 hours in ex patients and this time was significantly shorter than that of the discharged patients ($p<0.001$) (Table 6; Fig. 1).

Table 6. Time until interventional treatment is applied compared to prognosis

	Prognosis	n	x̄±SD	P
Time until interventional treatment is applied	Exitus	195	32,42±59,50	.000
	Discharged	334	54,14±81,36	

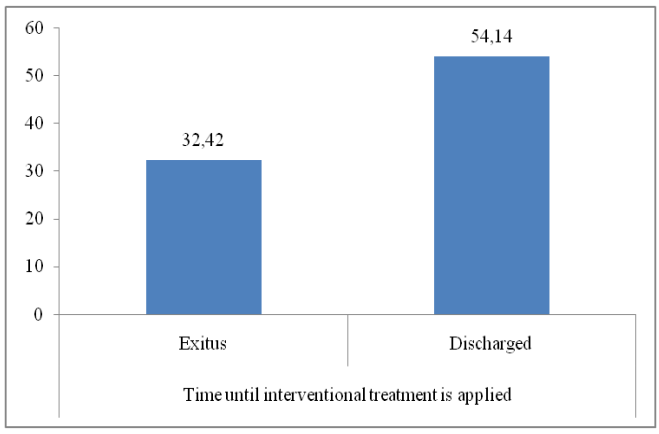

Fig. 1. Time until interventional treatment is applied compared to prognosis

4. Discussion

The study included 589 patients who presented to the emergency department of Inonu University Turgut Özal Medical Centre with spontaneous subarachnoid hemorrhage

between January 2011 and January 2020 and were diagnosed with cerebral aneurysm as a result of Digital Subtraction Angiography (DSA). During this period, 998 patients who were admitted to the intensive care unit with a diagnosis of SAH were analysed, while a group of 408 posttraumatic patients who were diagnosed with SAH due to arteriovenous malformation (AVM) and all other causes mentioned in the aetiology were excluded from the study.

The female sex ratio was 60.5% and the male sex ratio was 39.5%. The female-male sex ratio, which was reported as 1.4-1.6/1 in the literature, was found to be approximately 1.5/1 in the study. It was observed that the study was compatible with the literature in these data (19,20).

Spontaneous SAH is most commonly observed between the ages of 40-60 years. In many studies it has been reported that spontaneous SAHs are most frequently observed in the 6th decade and then in the 5th decade (19,21,22). In a study by Bozkuş (23) different from the literature, it was found that the incidence of spontaneous SAH was higher with increasing age, especially in the 7th and 8th decades. In our study, the most common decade with a rate of 29.5% was the 6th decade and the second most common decade with a rate of 20.4% was the 5th decade.

The most common times of spontaneous SAH vary according to geography, genetics, and climate characteristics. In the study by Clarke et al (19) it was reported that spontaneous SAH was mostly observed in winter and spring, whereas in the study by Chayette et al (24), it was found that the most frequent presentation was in spring and autumn seasons. In the present study, we analysed the frequency of monthly presentations and thus the seasonal distribution of spontaneous SAH. In our study, the most common month of presentation was found to be December with a rate of 10.2%. On a seasonal basis, winter was found to be the most common season in which spontaneous SAH was admitted to our emergency department with a rate of 27.7%. In this sense, it was observed that our study was compatible with the study by Clarke et al (19). The season with the least number of admissions was autumn with a rate of 23%. In fact, there were no significant differences in the frequency of spontaneous SAH admissions between seasons in our series.

The overall mortality rate was found to be 39%. In our study, the mortality rate in the female sex was found to be 37.6% and in the male sex 41.4%. Since mortality rates were reported to be approximately 30-50% in the literature, the results of the study were found to be compatible with the literature (25,26).

In our study, the mortality rate in the 7th decade was 49.1%, while the mortality rate in the 10th decade was found to be 100%. In the literature, it has been shown that advanced age is one of the most important reasons that adversely affects the prognosis of spontaneous SAH and increases the mortality rate (23,27).

Many studies in the literature have shown that the most common SA localisation is ACoM22. In another study in the literature, it was reported that MCA aneurysm was the most common localisation with a rate of 34% (28). In the present study, the most common SA localisation was MCA with a rate of 31.5% (185 patients). In the patient series in the study, it was found that SAs were located in the anterior circulation in 89% and in the posterior circulation in 11%. In the literature, the aneurysm rate in the anterior circulation was reported to be 90% and 10% in the posterior circulation. In this sense, our study was found to be compatible with the literature (29,30).

In some studies, the presence of multiple aneurysms was reported to be 6.5%, while in different studies it was reported to be between 15-45% (29,31,32). The presence of multiple aneurysms was found to be 20.4% in our patient group. In this sense, the study was compatible with the literature.

In our study, the mortality rate was 46.7% in patients with multiple aneurysms and 37% in patients without multiple aneurysms. In the light of these data, it can be said that being female gender is an additional risk in terms of the presence of multiple aneurysms and the presence of multiple aneurysms affects the prognosis in a bad way.

Headache is known to be the most common clinical symptom in patients with spontaneous SAH. In the literature, headache was observed in 70-76% of patients with spontaneous SAH (28,33). In the present study, headache was found to be present in 72.7% of patients presenting to the emergency department with spontaneous SAH.

Patients with spontaneous SAH may present to the emergency department with focal neurological deficits. In the literature, 15-20% of patients with SAH have focal neurological deficits. In our study, focal neurological deficit was found in 21.4% of the patients presenting to the emergency department. In this sense, the study was found to be compatible with the literature (34,35).

In the literature, the rate of nausea-vomiting in patients with SAH is reported to be between 34-48%, whereas, in some studies, it has been reported to be quite frequent with a rate of 70% (34,36). The rate of nausea-vomiting was found to be 22.9% in our patient group. This may be explained by the decrease or disappearance of nausea-vomiting complaints due to the antiemetic agents used by the patients until they reach the emergency department.

In the literature, the rate of epileptic seizure observed in the first 24 hours after spontaneous SAH has been shown to be between 6% and 20% (34,35,37). In our study, the rate of epileptic seizure seen after spontaneous SAH was found to be 10%, which was compatible with the literature.

In the literature, the rate of transient loss of consciousness in spontaneous SAHs was observed to be 19-40% (27,34). The

rate of patients presenting with transient loss of consciousness or altered consciousness was 25.6%. The study was found to be compatible with the literature in this respect.

The mortality rate in the patient group with GCS:7 and below was 88%. In a study conducted in a group of 1200 patients diagnosed with SAH, it was determined that GCS was one of the main factors affecting mortality (38).

In the study, spontaneous SAH patients were evaluated according to the FISHER classification. In a study by Bonilha et al (39) in which 100 patients with aneurysmal spontaneous SAH were analysed, FISHER grade 3 was the most common grade. In another study of 328 patients by Ilhan et al28 FISHER grade 2 was the most common grade.

In this study, spontaneous SAH patients were analysed according to the WFNS classification. In the study by Drake et al (40), it was reported that mortality decreased as the WFNS class decreased and the relationship between WFNS classification and the prognosis was significant. In our study, the highest mortality rate was observed in WFNS class 5 with 90.1%, while the lowest mortality rate was found in WFNS class 1 with 12.7%. It was determined that the mortality rate gradually increased as the WFNS progressed from class 1 to class 5. In the light of these data, the study was found to be compatible with the literature. In addition, it was observed that the clinical findings of the patients at the first presentation to the emergency department seriously affected the prognosis.

Hypertension has been shown to be one of the most important modifiable risk factors for spontaneous SAH in both sexes in all studies (11,41,42). It is known in the literature that chronic diseases, especially hypertension, are important factors in mortality in spontaneous SAH (10,41,42). In our study, mortality was observed at a rate of 50.6% in the patient group with hypertension.

In the literature, findings indicating that coronary artery diseases increase the risk of spontaneous SAH development have been reported (43). In our study, the mortality rate was 53.5% in patients with coronary artery disease.

In the literature, there is information that there is a relative relationship between diabetes and SAH in addition to major risk factors including hypertension, alcohol and smoking in the development of spontaneous SAH (44,45). The mortality rate in our patient group with diabetes was found as 50%.

Both treatment modalities have advantages and disadvantages. In the literature, it has been reported that the surgical method is advantageous compared to the endovascular method because it reduces the risk of vasospasm development by clearing the clot, provides effective and rapid intervention during intraoperative aneurysm hemorrhage, evacuation of the hematoma in the presence of a large parenchymal hematoma, and the aneurysm sac and the perforating branches emerging from its periphery can be protected and clipped. In addition, the advantages of the endovascular interventional method over the surgical method such as the absence of craniotomy, shorter duration of the procedure, and better postoperative patient comfort have also been emphasised in the literature. In a large meta-analysis study conducted in the literature, the results and risks of surgical and endovascular interventional methods were found to be statistically similar (46). In our study, the mortality rate was 38.1% in patients treated with surgical methods and 35.3% in patients treated with endovascular interventional methods. In the light of these data, no significant superiority of the two methods in terms of prognosis was observed in the study in accordance with the literature.

The mean hospitalisation time of 589 patients was found to be approximately 14 days after treatment. Although different hospitalisation times have been reported in the literature, it has been shown that this period varies between approximately 7-18 days (47). In this sense, the results of the series were compatible with the literature.

In conclusion, spontaneous SAH and SA is a life-threatening pathology with high morbidity and mortality, difficult to treat and follow-up, requiring serious experience and experienced personnel, and significantly increasing healthcare costs. In recent years, with the innovations in radiological imaging, surgical techniques, and endovascular interventional methods, follow-up and treatment have started to be performed more effectively. It is thought that morbidity and mortality in aneurysmal spontaneous subarachnoid hemorrhage will reach lower levels with the increase in experience, development of diagnostic methods, and innovations in treatment methods.

Conflict of interest

The authors declared no conflict of interest.

Funding

No funding was used for the study.

Acknowledgments

None to declare.

Authors' contributions

Concept: H.S., R.E., R.E., Design: H.S., R.E., D.E.K., F.T., B.T., Data Collection or Processing: H.S., Analysis or Interpretation: H.S., D.E.K., R.E., F.T., B.T., Literature Search: H.S., R.E., D.E.K., F.T., B.T., Writing: H.S., R.E., D.E.K., F.T., B.T

Ethical Statement

Approval was obtained from Inönü University Scientific Research and Publication Ethics Committee, the study started. The ethics committee decision date is 28/07/2020 and the number of ethical committee decisions is 2020/764.

References

1. Marder CP, Narla V, Fink JR, & Tozer Fink KR. Subarachnoid hemorrhage: beyond aneurysms. American Journal of Roentgenology, 2014;202(1), 25-37

2. Al-Mistarehi AH, Elsayed MA, Ibrahim RM, Elzubair TH, Badi S, Ahmed MH, et al. Clinical outcomes of primary subarachnoid

hemorrhage: an exploratory cohort study from Sudan. The Neurohospitalist, 2022;12(2),249-263

3. Kauvar DS, Lefering R, & Wade CE. Impact of hemorrhage on trauma outcome: an overview of epidemiology, clinical presentations, and therapeutic considerations. Journal of Trauma and Acute Care Surgery, 2006;60(6), S3-S11

4. Rossaint R, Bouillon B, Cerny V, Coats TJ, Duranteau J, Fernández-Mondéjar E et al. Management of haemorrhage following major trauma: an updated European guideline. Critical Care, 2010;14, 1-29.

5. Chou SHY, Feske SK, Atherton J, Konigsberg RG, De Jager PL, Du R et al. Early elevation of serum tumor necrosis factor-α is associated with poor outcome in subarachnoid hemorrhage. Journal of Investigative Medicine, 2012;60(7), 1054-1058.

6. Macdonald RL, & Schweizer TA. Spontaneous subarachnoid haemorrhage. The Lancet, 2017;389(10069), 655-666.

7. van Lieshout JH, Dibué-Adjei M, Cornelius JF, Slotty PJ, Schneider T, Restin T et al. An introduction to the pathophysiology of aneurysmal subarachnoid hemorrhage. Neurosurgical Review, 2018;41,917-930.

8. Claassen J, & Park S. Spontaneous subarachnoid haemorrhage. The Lancet, 2022;400(10355), 846-862

9. Raatikainen, E. Delayed Cerebral Ischemia and Blood Coagulation Changes After Aneurysmal Subarachnoid Hemorrhage: Tampere University Dissertations, Tampere University, Faculty of Medicine and Health Technology Tampere University Hospital, Department of Intensive Care Medicine, Finland, 2023.

10. Andreasen, TH, Bartek Jr J, Andresen M, Springborg JB, & Romner B. Modifiable risk factors for aneurysmal subarachnoid hemorrhage. Stroke, 2013;44(12),3607-3612

11. Van Gijn J, Kerr RS, & Rinkel, GJ. Subarachnoid haemorrhage. The Lancet, 2007;369(9558), 306-318.

12. de Oliveira Manoel AL, Goffi A, Zampieri FG, Turkel-Parrella D, Duggal A, Marotta TR, et al. The critical care management of spontaneous intracranial hemorrhage: a contemporary review. Critical Care, 2016;20,1-29

13. de Oliveira Manoel AL, Mansur A, Murphy A, Turkel-Parrella D, Macdonald M, Macdonald RL et al. Aneurysmal subarachnoid haemorrhage from a neuroimaging perspective. Critical Care, 2014;18(6),1-13

14. Shaban S, Huasen B, Haridas A, Killingsworth M, Worthington J, Jabbour P et al. Digital subtraction angiography in cerebrovascular disease: current practice and perspectives on diagnosis, acute treatment and prognosis. Acta Neurologica Belgica, 2021;1-18.

15. Molyneux A, Kerr R, Stratton I, Sandercock P, Clarke M, Shrimpton J et al. International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomised trial. Lancet, 2002;360(9342):1267–1274

16. Salary M, Quigley MR, Wilberger JE. Relation among aneurysm size, amount of subarachnoid blood, and clinical outcome. J Neurosurg, 2007;107(1):13–17

17. Wostrack M, Sandow N, Vajkoczy P, Schatlo B, Bijlenga P, Schaller K et al. Subarachnoid haemorrhage WFNS grade V: is maximal treatment worthwhile? Acta Neurochir, 2013;155(4):579–586

18. Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. Lancet, 1974;2(7872):81–84.

19. Clarke M. Systematic review of reviews of risk factors for intracranial aneurysms. Neuroradiology, 2008;50(8), 653-664

20. Ziemba-Davis M, Bohnstedt BN, Payner TD, Leipzig TJ, Palmer E, Cohen-Gadol AA. Incidence, Epidemiology, and Treatment of Aneurysmal Subarachnoid Hemorrhage in 12 Midwest Communities. J Stroke Cerebrovasc Dis, 2014;23(5):1073-82

21. Tong KA, Ashwal S, Holshouser BA, Shutter LA, Herigault G, Haacke EM et al. Hemorrhagic shearing lesions in children and adolescents with posttraumatic diffuse axonal injury: improved detection and initial results. Radiology, 2003;227(2):332-9

22. Devkota UP, Aryal KR. Result of surgery for ruptured intracranial aneurysms in Nepal. Br J Neurosurg, 2001;15: 13-6.

23. Bozkuş H. Subarachnoid hemorrhage in the elderly. J Neurosurg, 1993;7: 307-9.

24. Chyette D, Chen TL, Bronstein K, Brass LM. Seasonal fluctution in the incidence of intracranial aneurysm rupture and its relationship to changing climatic conditions. J Neurosurg, 1984;81: 525-30.

25. Steiner T, Juvela S, Unterberg A, Jung C, Forsting M, Rinkel G. European Stroke Organization. European Stroke Organization guidelines for the management of intracranial aneurysms and subarachnoid haemorrhage. Cerebrovasc Dis, 2013;35:93-112.

26. Edlow JA, Malek AM, Ogilvy CS: Aneurysmal subarachnoid hemorrhage: update for emergency physicians. J Emerg Med, 2008;34: 237- 251

27. Kassell NF, Torner JC, Haley EC Jr, Jane JA, Adams HP, Kongable GL, et al. The international cooperative study on the timing of aneurysm surgery. Part I: Overall management results. J Neurosurg, 1990;73: 37-47

28. İlhan Y, Eseoğlu M, Demirgil B. Spontaneous Subarachnoid Hemorrhage Caused By Aneurysm: The Retrospective Analysis of 328 Cases, Fırat Tıp Dergisi, 2012;156-160

29. Gambhir S, O'Grady G, Koelmeyer T. Clinical lessons and risk factors from 403 fatal cases of subarachnoid haemorrhage. J Clin Neurosci. 2009;16: 921-4.

30. Jeong YG, Jung YT, Kim MS, Eun CK, Jang SH. Size and location of ruptured intracranial aneurysms. J Korean Neurosurg Soc. 2009;45: 11-5

31. van der Wee N, Rinkel GJ, Hasan D, van Gijn J. Detection of subarachnoid haemorrhage on early CT: Is lumbar puncture still needed after a negative scan? J Neurol Neurosurg Psychiatry, 1995;58: 357–359

32. Mayberg MR, Batjer HH, Dacey R, Diringer M, Haley EC, Heros RC et al. Guidelines for the management of aneurysmal subarachnoid hemorrhage. A statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. Circulation, 1994;90(5), 2592-2605

33. Perry JJ, Stiell IG, Sivilotti ML, Bullard MJ, Hohl CM, Sutherland J, et al. Clinical decision rules to rule out subarachnoid hemorrhage for acute headache. JAMA, 2013;310(12), 1248-1255

34. Kowalski RG, Claassen J, Kreiter KT, Bates JE, Ostapkovich ND, Connolly ES, et al. Initial misdiagnosis and outcome after subarachnoid hemorrhage. Jama, 2004;291(7), 866-869.

35. McCarron MO, Alberts MJ, McCarron P. A systematic review of Terson's syndrome; frequency and prognosis after subarachnoid haemorrhage. J Neurol Neurosurg Psychiatry, 2004;75:491-493

36. Leablanc R: The minor leek preceding subarachnoid hemorrhage. J Neurosurg, 1987;66: 35-9

37. Boling W, Kore L. Subarachnoid Hemorrhage-Related Epilepsy. In: Martin, R., Boling, W., Chen, G., Zhang, J. (eds) Subarachnoid Hemorrhage. Acta Neurochirurgica Supplement, 2020;127. Springer, Cham. https://doi.org/10.1007/978-3-030-04615-6_4

38. Lantigua H, Ortega-Gutierrez S, Schmidt JM, Lee K, Badjatia N, Agarwal S, et al. Subarachnoid hemorrhage: who dies, and why?. Critical Care, 2015;19(1), 1-10

39. Bonilha L, Marques EL, Carelli EF, Fernandes YB, Cardoso AC, Maldaum MV, et al. Risk factors and outcome in 100 patients with aneurysmal subarachnoid hemorrhage. Arquivos de Neuro-Psiquiatria, 2001;59, 676-680

40. Drake CG, Hunt WE, Sano K. Report of World Federation of Neurosurgical Surgeons Committee on a Universal Subarachnoid Hemorrhage Grading Scale. J Neurosurg, 1988;68;985-986.

41. Kopitnik TA, Samson DS. Management of subarachnoid hemorrhage. J Neurol Neurosurg Psychiatry, 1993;56: 947-59.

42. Grasso G, Alafaci C, Macdonald RL. Management of aneurysmal subarachnoid hemorrhage: State of the art and future perspectives. Surg Neurol Int, 2017;8:1-10

43. Goldstein LB, Bushnell CD, Adams RJ, Chaturvedi S, Creager MA: Guidelines for the Primary Prevention of Stroke: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke, 2011;42: 517-84.

44. Lindbohm JV, Kaprio J, Korja M: Cholesterol as a risk factor for subarachoid hemorrhage: Systemic review. PLoS One,11:e0152568, 2016.

45. Zhang J, Liu G, Arima H, Li Y, Cheng G, Shiue I et al: Incidence and risks of subarachnoid hemorrhage in China. Stroke, 44:2891-2893, 2013

46. Piotin M, Spelle L, Mounayer C, Salles-Rezende MT, Giansante-Abud D, Vanzin-Santos R et al. Intracranial aneurysms: Treatment with bare platinum coils-aneurysm packing, complex coils, and angiographic recurrence. Radiology, 2007;243(2):500-508.

47. Yıldırım Ar A, Bayram T, and Turan G. The Effects of Grading Scales Used in Patients with Aneurysmal Subarachnoid Hemorrhage on Critical Care Outcomes. Kocaeli Medical Journal, 2020;9(1), 18-23.