

Clinical Course, Complications, and Treatment Approaches in Cases with Subarachnoid Hemorrhage: Single-Center Experience at a Tertiary Care Facility

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ABSTRACT

Objective: This study aimed to evaluate the demographic, clinical, and radiological characteristics, treatment approaches, and outcomes of patients diagnosed with subarachnoid hemorrhage (SAH) at a tertiary neurosurgery center, distinguishing between traumatic and spontaneous (aneurysmal and non-aneurysmal) etiologies.

Materials and Methods: A total of 188 patients diagnosed with SAH between September 2023 and September 2025 were retrospectively reviewed. Patients were classified as having traumatic or spontaneous SAH based on etiology. Demographic characteristics, imaging findings, aneurysm features, complications (vasospasm, hydrocephalus), treatment modalities, and clinical outcomes were recorded and analyzed.

Results: Of the 188 patients, 126 (67.0%) had traumatic SAH and 62 (33.0%) had spontaneous SAH. The mean age was 40.6 ± 23.6 years in traumatic cases and 52.0 ± 14.1 years in spontaneous cases. Aneurysmal rupture accounted for 74.2% of spontaneous SAH cases, most frequently involving the anterior communicating artery (27.4%) and the middle cerebral artery (21.0%). Vasospasm occurred in 19.3% and hydrocephalus in 14.5% of spontaneous cases. The overall mortality rate was 1.6%, and favorable neurological recovery ($mRS \leq 3$) was achieved in 83.9% of patients.

Conclusion: Traumatic SAH occurs predominantly in younger males, whereas spontaneous SAH most commonly results from aneurysm rupture. Early diagnosis, multidisciplinary management, vasospasm monitoring, and the widespread use of endovascular therapy are key factors in reducing mortality and improving neurological recovery.

Keywords: Aneurysm, endovascular treatment, subarachnoid hemorrhage, traumatic brain injury, vasospasm

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INTRODUCTION

Subarachnoid hemorrhage (SAH) represents one of the most life-threatening neurological emergencies, caused by bleeding into the space between the pia mater and the arachnoid membrane surrounding the brain and spinal cord.^[1] The presence of blood in this anatomical compartment, where cerebrospinal fluid circulates, triggers severe pathophysiological processes, such as increased intracranial pressure, meningeal irritation, cerebral edema, vasospasm, and secondary ischemic injury.^[1,2]

SAH is classified into two main categories based on etiology: traumatic and non-traumatic (spontaneous).^[3] The most common cause of all SAH cases is trauma; traumatic subarachnoid hemorrhage (tSAH) is frequently observed in patients with moderate to severe head injury and significantly increases the mortality associated with traumatic brain injury.^[3,4] Post-traumatic subarachnoid bleeding typically appears over cortical surfaces and cerebral convexities and may be accompanied by diffuse axonal injury, contusions, or skull base fractures.^[3,4]

Non-traumatic or spontaneous SAH most often results from the rupture of aneurysms that develop due to structural



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weaknesses in the walls of intracranial arteries.^[1,5] Aneurysms are the most frequent cause of non-traumatic subarachnoid hemorrhage, accounting for approximately 80% of all spontaneous SAH cases.^[5] However, other causes, such as arteriovenous malformations (AVM), arterial dissections, vasculitis, moyamoya disease, brain tumors, coagulopathies, or anticoagulant use, may also lead to non-aneurysmal spontaneous SAH.^[2,5] Among these, cerebral dissection and vasculitis are particularly significant etiological factors in young patients.^[5]

An aneurysm is defined as a localized or segmental dilation of an artery resulting from a disruption of its normal wall structure. Intracranial aneurysms form when degenerative changes occur in the elastic lamina and tunica media of the vessel wall, causing the lumen to balloon outward.^[6] The most common type is the saccular (berry) aneurysm, which typically develops at arterial bifurcation points within the Circle of Willis and has a distinct neck-and-dome configuration. Saccular aneurysms are most frequently found at the anterior communicating artery (ACom), posterior communicating artery (PCom), middle cerebral artery (MCA), and internal carotid artery (ICA) bifurcations; less commonly, they occur in the posterior circulation, especially at the basilar and vertebral artery levels.^[6,7]

Rarer fusiform aneurysms are characterized by a diffuse dilation involving an entire arterial segment. In this type of aneurysm, all layers of the vessel wall are affected, often as a result of atherosclerotic degeneration, dissection, or structural wall weakness. Fusiform aneurysms lack a defined neck, making surgical or endovascular treatment more difficult, and are most often located in the basilar artery, vertebral artery, or distal segments of the internal carotid artery.^[5,6]

Aneurysm rupture causes the sudden discharge of high-pressure arterial blood into the subarachnoid space. This leads to a rapid increase in intracranial pressure, a decrease in global cerebral perfusion, and diffuse neurological deterioration.^[1,2,6] Following rupture, blood spreads into the basal cisterns and sulci, resulting in diffuse cerebral edema, vasospasm, ischemic injury, and secondary complications.^[1,4] Posterior circulation aneurysms and advanced age are associated with higher mortality rates.^[6]

Global epidemiological studies report the incidence of SAH as 7.9 per 100,000 person-years, occurring more frequently in women.^[2] Geographical differences are notable; although incidence has decreased in developed countries in recent years, mortality remains between 20–40% (2,6).

Hypertension, smoking, advanced age, and female sex are identified as major risk factors for aneurysm rupture.^[2,5]

In approximately 15–20% of spontaneous SAH cases, angiographic examinations do not detect an aneurysm, and this group is called non-aneurysmal (angiogram-negative) SAH.^[5] In these patients, possible causes include perimesencephalic venous bleeding, vasculitis, dissection, or microscopically ruptured small aneurysms. However, in some cases, digital subtraction angiography (DSA) initially negative may reveal small or thrombosed aneurysms in follow-up investigations.^[5]

In conclusion, subarachnoid hemorrhage is a cerebrovascular disease with high mortality, which can develop due to traumatic or non-traumatic causes. Mechanical vascular injury predominates in traumatic cases, while aneurysm rupture is mostly responsible for the etiology in spontaneous cases. Early recognition of SAH, proper etiological differentiation, and appropriate treatment choices are crucial for reducing mortality and morbidity.

The aim of this study was to classify all subarachnoid hemorrhage cases followed in our center over the last two years as traumatic and spontaneous (aneurysmal and non-aneurysmal) and to comparatively evaluate the demographic, clinical, and radiological features, treatment approaches, and short-term outcomes of both groups. Moreover, we aim to contribute to the clinical management of this group by determining the rate of aneurysms detected later in control DSA examinations of non-aneurysmal cases.

MATERIALS and METHODS

Study Design and Ethical Approval

This study was designed as a retrospective, cross-sectional research conducted at the neurosurgery clinic of our tertiary care hospital. All patients admitted and followed with the diagnosis of subarachnoid hemorrhage (SAH) between September 2023 and September 2025 were included in the study. Ethical approval was obtained from the Kanuni Sultan Süleyman Training and Research Hospital Ethics Committee (Approval No: 2025.10.294, Date: 23.10.2025). Patient data were evaluated based on confidentiality principles, and the study was conducted in accordance with the Declaration of Helsinki principles.

Patient Population

All consecutive SAH cases were retrospectively reviewed through the hospital information system and radiological archive records. Patients were divided into two main groups

according to their etiology: traumatic and spontaneous (non-traumatic). The traumatic SAH (tSAH) group consisted of patients with subarachnoid hemorrhage detected by brain computed tomography (CT) performed after head trauma. The non-traumatic (spontaneous) SAH group was further evaluated in two subgroups based on underlying vascular pathology: aneurysmal SAH and non-aneurysmal (angiogram-negative) SAH.

In traumatic cases, diagnosis was made by demonstrating the presence of blood in the basal cisterns or convexity sulci on brain computed tomography (CT) scans performed within the first 48 hours after trauma. In spontaneous cases, diagnosis was confirmed with non-contrast CT, and all cases were directed to brain angiography for further evaluation. Cases with saccular or fusiform aneurysms detected on angiography were classified as the "aneurysmal group," while those without any aneurysm detected were classified as the "non-aneurysmal group." All patients in the non-aneurysmal group underwent follow-up digital subtraction angiography (DSA) or magnetic resonance angiography (MRA) between days 7 and 14 or weeks 4 and 6. Cases with new aneurysm development detected on follow-up DSA were also recorded.

Clinical and Radiological Evaluation

Patient age, gender, trauma mechanism, Glasgow Coma Scale (GCS), Fisher score, complications (vasospasm, hydrocephalus), surgical intervention status, length of hospital stay, and neurological outcomes at discharge were recorded. A favorable outcome was defined as a modified Rankin Scale (mRS) score of 0–3 or a Glasgow Outcome Scale–Extended (GOSE) score of 5–8 at discharge.

All CT scans were performed using the Philips Ingenuity Core 128 (Best, Netherlands) device. Imaging parameters were standardized to 120 kVp tube voltage, automatic tube current modulation, 1 mm slice thickness, and a bone algorithm. A fixed head position and a standard brain window (WW 80, WL 40) were used throughout all CT examinations.

Angiographic examinations were conducted using the Siemens Artis zee biplane (Siemens Healthcare, Erlangen, Germany) device. DSA procedures were performed by an experienced interventional radiologist through a 5F femoral catheter. Selective imaging of all cerebral arteries was ensured (bilateral ICA, vertebral artery, and basilar artery). Three-dimensional rotational angiography technique was used when needed. For patients with aneurysms detected, localization (ACom, PCom, MCA, ICA, BA, VA, etc.) and

morphological features (size, neck width, dome/neck ratio) were recorded.

Aneurysm treatment was performed by either endovascular coil embolization or surgical clipping. Treatment choice was determined based on the patient's clinical status, aneurysm morphology, and decisions of the neurovascular surgery council.

Statistical Analysis

The data were analyzed using SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean \pm standard deviation (SD) or median (interquartile range, IQR); categorical variables were expressed as number and percentage (%).

RESULTS

A total of 188 subarachnoid hemorrhage (SAH) patients were evaluated. The etiological distribution of traumatic and spontaneous subarachnoid hemorrhage cases is illustrated in Figure 1. Of the cases, 126 (67.0%) were traumatic, and 62 (33.0%) were spontaneous in origin. The mean age of the traumatic SAH group was 40.6 ± 23.6 years, while that of the spontaneous group was 52.0 ± 14.1 years. Traumatic cases were significantly younger. Gender distribution showed a male predominance in the traumatic group (96 males, 76.2%), while the spontaneous group had a more balanced distribution (30 males, 48.3%; 32 females, 51.7%) (Table 1).

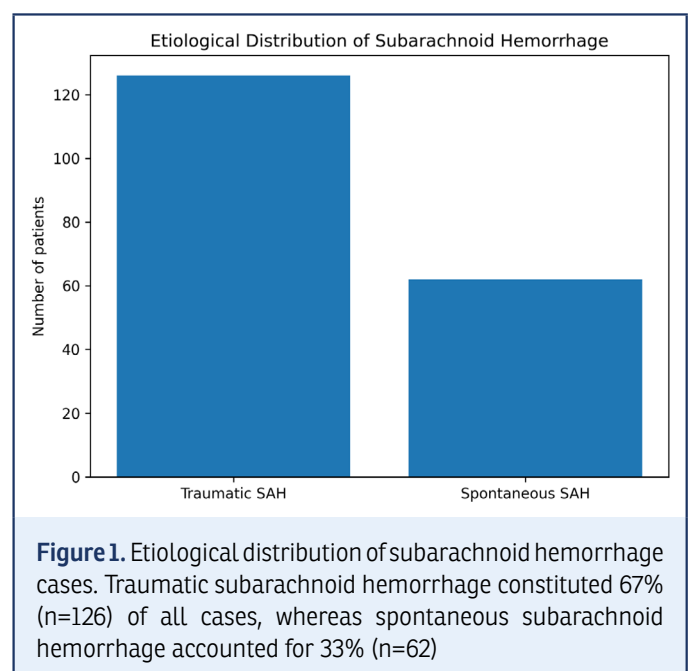


Table 1. Baseline demographic and clinical characteristics of patients with subarachnoid hemorrhage (SAH)

Parameter	Total (n=188)	Traumatic SAH (n=126)	Spontaneous SAH (n=62)
Age (years), mean±SD	44.6±21.8	40.6±23.6	52.0±14.1
Sex (male/female)	126/62	96/30	30/32
Etiology	—	Traumatic	Aneurysmal / Non-aneurysmal
Proportion of total cases	—	126 (67.0%)	62 (33.0%)

SAH: Subarachnoid hemorrhage

In cases of traumatic SAH, the most common causes were falls from a height (36 patients, 28.6%) and motor vehicle accidents (30 patients, 23.8%). Same-level falls (slips, falls from stairs, etc.) were detected in 20 patients (15.9%), and assault was found in 10 patients (7.9%). Occupational accidents or mechanical traumas were observed in 8 patients (6.3%), and falls after loss of consciousness or syncope were seen in 7 patients (5.6%). Other causes (minor traumas, head bumps of undetermined origin, etc.) were noted in 15 patients (11.9%).

In 82 patients (65.1%), the trauma was isolated head trauma, while in 44 patients (34.9%), it was polytrauma (accompanied by thoracic, pelvic, or extremity injuries). In polytrauma cases, histories of falls from height and motor vehicle accidents were particularly prominent. Upon examination of hemorrhage distribution, multifocal or diffuse SAH was detected in most cases (79 patients, 62.7%). The most frequently affected regions were the frontal (18 patients, 14.3%) and temporal/parietal (16 patients, 12.7%) lobes. In isolated head trauma cases, hemorrhage was generally limited to cortical surfaces, whereas in polytrauma patients, spread to basal cisterns or the interhemispheric fissure was more commonly observed.

Three patients (2.4%) died during intensive care follow-up; the causes of death in these cases were primarily related to multiple trauma and systemic complications (hemorrhagic shock, intrathoracic organ injuries). Additionally, decompressive craniectomy was performed in three patients (2.4%) due to increased intracranial pressure. The average hospital stay was 6.3±2.4 days (Table 2).

In 46 (74.2%) of the spontaneous SAH cases, an underlying aneurysm rupture was identified, while in 16 (25.8%), non-

Table 2. Etiological and radiological characteristics of traumatic subarachnoid hemorrhage (tSAH)

Parameter	Traumatic SAH (n=126)
Cause of trauma	Fall from height 36 (28.6%); Motor vehicle accident 30 (23.8%); Same-level fall 20 (15.9%); Assault 10 (7.9%); Occupational/mechanical 8 (6.3%); Syncope-related fall 7 (5.6%); Other 15 (11.9%)
Trauma pattern	Isolated head trauma 82 (65.1%); Polytrauma 44 (34.9%)
Hemorrhage distribution	Diffuse/multifocal 79 (62.7%); Frontal 18 (14.3%); Temporal/parietal 16 (12.7%)
Surgical treatment	Decompressive craniectomy 3 (2.4%)
ICU mortality	3 (2.4%) — related to systemic/multiple trauma
Mean hospital stay (days)	6.3±2.4

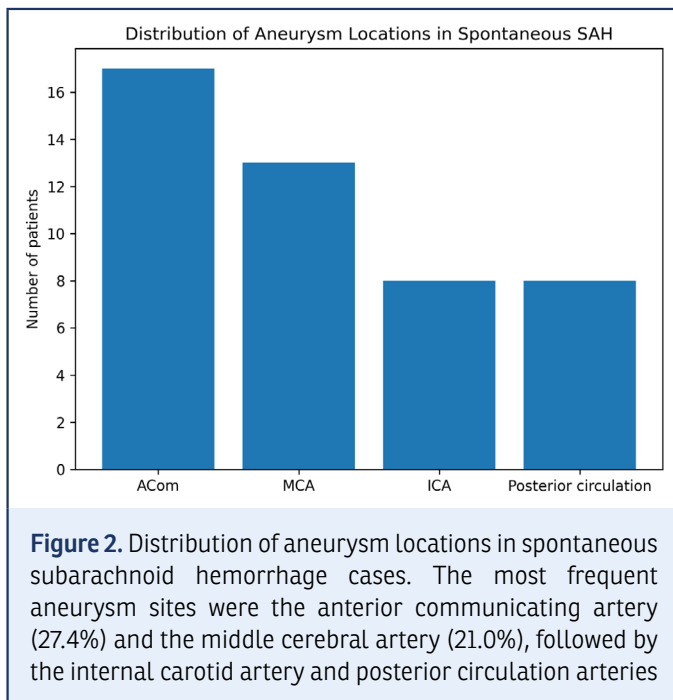
tSAH: Traumatic subarachnoid hemorrhage; ICU: Intensive care unit

aneurysmal (angiogram-negative) subarachnoid hemorrhage was detected. The distribution of aneurysm locations among spontaneous subarachnoid hemorrhage cases is shown in Figure 2. Aneurysm locations were distributed as follows: anterior communicating artery (ACom, 17 patients, 27.4%), middle cerebral artery (MCA, 13 patients, 21.0%), internal carotid artery (8 patients, 12.9%), and posterior circulation (basilar, vertebral, PCA; 8 patients, 12.9%).

In the aneurysmal SAH group, treatment was performed with endovascular coil embolization in 26 patients (56.5%) and surgical treatment in 20 patients (43.5%). One patient in the surgical group, who presented with advanced brain herniation signs at admission, underwent emergency surgery and died postoperatively. Other patients generally remained neurologically stable, but 5 patients (8.0%) developed permanent neurological morbidity. Significant vasospasm was noted in most of these cases.

All spontaneous SAH patients underwent follow-up DSA on average 4–6 weeks later. Aneurysm remnants were detected on follow-up imaging in 3 of the treated aneurysmal cases (6.5%). Of 16 non-aneurysmal patients with initially negative angiograms, 2 (3.2%) developed new aneurysms detected on follow-up DSA. The remaining patients remained neurologically stable during follow-up.

According to the Fisher grading, 30 patients (48.4%) were Fisher-4, 17 (27.4%) Fisher-3, 12 (19.4%) Fisher-2, and 3 (4.8%) Fisher-1. The mean Glasgow Coma Scale (GCS) score



at admission was 11.4 ± 3.2 . Vasospasm was observed in 12 patients (19.3%) in the spontaneous SAH group; among these, 3 had no aneurysm detected. Ventriculoperitoneal (VP) shunting was performed in 9 patients (14.5%) for permanent drainage due to hydrocephalus.

The mortality rate in the spontaneous SAH group was 1.6% (1 patient), and morbidity was 8.0% (5 patients). Favorable outcomes ($mRS \leq 3$, $GOSE \geq 5$) at discharge were observed in 83.9% of survivors. The average hospital stay was 11.4 ± 4.1 days (Table 3).

Traumatic SAH cases were predominantly seen in younger age groups and males, whereas spontaneous SAH patients were older with a balanced gender distribution. The aneurysm rate in spontaneous cases was 74.2%, with the most common locations being the anterior communicating artery (ACom) and middle cerebral artery (MCA). Post-treatment remnant rate was 4.8%, and the rate of aneurysms detected later after an initially negative DSA was 3.2%. The frequency of vasospasm was 19.3%, the mortality rate was 1.6%, and the morbidity rate was 8.0%. These results demonstrate that low mortality and high neurological recovery rates can be achieved with appropriate treatment approaches and regular follow-up DSA protocols.

DISCUSSION

This study retrospectively evaluated the clinical features, etiological distributions, and short-term outcomes of traumatic and spontaneous subarachnoid hemorrhage

(SAH) cases. Our findings indicate that traumatic SAHs occur more commonly in younger age groups and males, frequently resulting from falls from height or motor vehicle accidents, while spontaneous SAH cases are mostly due to aneurysm rupture.

Traumatic SAH is a consequence of head trauma and is considered an indicator of the severity of traumatic brain injury. Literature reports indicate that SAH develops in 26% to 53% of patients who have sustained head trauma, with mortality rates ranging from 8% to 20% depending on the severity of the trauma.^[1] In the study by Parchani et al.^[3] mortality in traumatic SAH cases was 19%, with motor vehicle accidents (53%) and falls from height (35%) being the most common causes. In our series, 28.6% of traumatic cases were due to falls from height, 23.8% due to motor vehicle accidents, and the mortality rate was 2.4%. This low mortality rate may be attributed to the predominance of mild-to-moderate trauma severity and early intensive care follow-up in our cohort.

In traumatic SAHs, mortality is generally associated with accompanying systemic injuries. In our series, all three cases with mortality had coexisting thoracic or abdominal trauma. Additionally, decompressive craniectomy was performed in three patients (2.4%) due to increased intracranial pressure, which is comparable to the rates of surgical requirement reported in the literature (1).

The most common etiology of spontaneous SAH cases is intracranial aneurysm rupture. Literature reports this rate as between 70% and 85%,^[2] and in our study, it was found to be 74.2%, consistent with the literature. The most frequent locations of aneurysms are the anterior communicating artery (ACom) and middle cerebral artery (MCA), a finding that aligns with the Kobe City study by Ohta and colleagues conducted in the Japanese population.^[7]

In recent years, with the advancement of endovascular techniques, mortality rates in the treatment of aneurysmal subarachnoid hemorrhage (SAH) have significantly decreased. In a 10-year series conducted by Ronne-Engström et al.^[4] it was reported that as the rates of endovascular treatment increased, mortality decreased to around 10%, while the proportion of patients with favorable neurological outcomes increased. In our study, the mortality rate was found to be 1.6%, and the favorable outcome rate was 83.9%. These findings may be explained by early intervention in selected cases and the implementation of a systematic follow-up DSA protocol.

Vasospasm is the most common secondary complication following aneurysmal SAH, reported in 20% to 30% of cases.

^[6] In our series, this rate was 19.3%, and significant vasospasm was detected in most patients who sustained morbidity. The pivotal role of delayed cerebral ischemia (DCI) and vasospasm in mortality and permanent neurological deficits has also been emphasized in previous reports.^[6,7] Additionally, the need for ventriculoperitoneal shunt placement has been reported in approximately 15% of patients who develop hydrocephalus, a rate consistent with our findings (14.5%).^[4]

In non-aneurysmal (angiogram-negative) SAH cases, the rate of aneurysms initially undetected but identified on follow-up angiography has been reported to range between 1.6% and 4.3% in the literature.^[5] In our series, two patients (3.2%) developed new aneurysms on follow-up DSA. This observation further supports the necessity of close imaging surveillance in angiogram-negative SAH cases.

The reduction in early mortality rates is associated not only with endovascular techniques but also with systematic vasospasm monitoring protocols and early medical management. In a study by Shibuya et al.^[8] the Rho-kinase inhibitor fasudil was shown to significantly reduce the incidence of cerebral vasospasm and poor outcomes. Similarly, the ISAT trial conducted by Molyneux et al.^[9] demonstrated that endovascular treatment was associated with lower mortality and morbidity compared with surgical clipping. Walcott et al.^[10] also emphasized that identifying early risk factors for shunt dependency in patients with hydrocephalus following aneurysmal SAH has a significant impact on patient prognosis.

Long-term follow-up studies have shown that survivors of subarachnoid hemorrhage have a markedly higher risk of cardiovascular events, epilepsy, and cognitive impairment compared with the general population.^[11] Therefore, not only acute but also long-term systemic follow-up is crucial in SAH patients.

In conclusion, data from our center demonstrate low mortality and favorable clinical outcomes in traumatic SAH cases, and complication and prognosis profiles consistent with the literature in spontaneous (particularly aneurysmal) SAH cases. The widespread adoption of endovascular treatment techniques, early diagnosis, systematic vasospasm monitoring protocols, and routine follow-up DSA procedures stand out as key factors in reducing mortality and improving neurological recovery.^[2,4,6,7,9,11]

CONCLUSION

In this study, the clinical courses, complication rates, and short-term outcomes of traumatic and spontaneous

subarachnoid hemorrhage (SAH) cases were comprehensively evaluated. Our findings indicate that traumatic SAH typically occurs in young male patients, follows a milder clinical course, and is associated with lower mortality, whereas spontaneous SAH largely results from aneurysmal rupture. Among aneurysmal cases, the most common locations were the anterior communicating artery (ACoM) and the middle cerebral artery (MCA), and early endovascular intervention along with routine follow-up DSA played a decisive role in reducing mortality.

Furthermore, our study demonstrated that vasospasm has a significant impact on morbidity, while hydrocephalus and delayed aneurysm detection markedly influence prognosis. It was also concluded that angiogram-negative cases should be re-evaluated with follow-up DSA in the near term.

In conclusion, early diagnosis, a multidisciplinary treatment approach, systematic vasospasm monitoring, and the widespread use of endovascular techniques are of critical importance in improving neurological recovery and reducing mortality in both traumatic and spontaneous SAH cases.

Disclosures

Ethics Committee Approval: This study was approved by the Kanuni Sultan Süleyman Training and Research Hospital Ethics Committee (Approval No:2025.10.294, Date:23.10.2025).

Informed Consent: Written informed consent was waived due to the retrospective nature of the study.

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REFERENCES

1. Modi NJ, Agrawal M, Sinha VD. Post-traumatic subarachnoid hemorrhage: A review. *Neurol India* 2016;64(Suppl):S8–13. [\[Crossref\]](#)
2. Etminan N, Chang HS, Hackenberg K, de Rooij NK, Vergouwen MDI, et al. Worldwide incidence of aneurysmal subarachnoid hemorrhage according to region, time period, blood pressure, and smoking prevalence in the population: a systematic review and meta-analysis. *JAMA Neurol* 2019;76:588–97. [\[Crossref\]](#)
3. Parchani A, Patra DP, Mukherjee KK, Savardekar AR, Narayan V, Srinivas D. Traumatic subarachnoid hemorrhage due to motor vehicle crash versus fall from height: a four-year epidemiologic study. *World Neurosurg* 2014;82:e639–44. [\[Crossref\]](#)
4. Ronne-Engström E, Borota L, Lenell S, Lewén A, Mahmoud E, Nyberg C, et al. Trends in incidence and treatments of spontaneous subarachnoid hemorrhage- a 10 year hospital based study. *Acta Neurochir (Wien)* 2024;166:188. [\[Crossref\]](#)
5. Mortazavi ZS, Zandifar A, Kim JU, Tierradentro-García LO, Shakarami M, Dashti Zamharir F, et al. Re-evaluating risk factors, incidence, and outcome of aneurysmal and non-aneurysmal subarachnoid hemorrhage. *World Neurosurg* 2023;175:492–504. [\[Crossref\]](#)
6. Øie LR, Solheim O, Majewska P, Nordseth T, Müller TB, Carlsen SM, et al. Incidence and case fatality of aneurysmal subarachnoid hemorrhage admitted to hospital between 2008 and 2014 in Norway. *Acta Neurochir (Wien)* 2020;162:1541–51. [\[Crossref\]](#)
7. Ohta T, Matsumoto S, Fukumitsu R, Imamura H, Adachi H, Hara Y, et al. Incidence and outcomes of aneurysmal subarachnoid hemorrhage: a multicenter retrospective registry-based descriptive trial in kobe city. *Neurol Med Chir (Tokyo)* 2023;63:519–25. [\[Crossref\]](#)
8. Shibuya M, Suzuki Y, Sugita K, Saito I, Sasaki T, Takakura K, et al. Effect of AT877 on cerebral vasospasm after aneurysmal subarachnoid hemorrhage. Results of a prospective placebo-controlled double-blind trial. *J Neurosurg* 1992;76:571–7. [\[Crossref\]](#)
9. Molyneux AJ, Kerr RS, Yu LM, Clarke M, Sneade M, Yarnold JA, et al. International subarachnoid aneurysm trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomised comparison of effects on survival, dependency, seizures, rebleeding, subgroups, and aneurysm occlusion. *Lancet* 2005;366:809–17. [\[Crossref\]](#)
10. Walcott BP, Iorgulescu JB, Stapleton CJ, Kamel H. Incidence, timing, and predictors of delayed shunting for hydrocephalus after aneurysmal subarachnoid hemorrhage. *Neurocrit Care* 2015;22:326–32. [\[Crossref\]](#)
11. Nieuwkamp DJ, Algra A, Blomqvist P, Adami J, Buskens E, Koffijberg H, et al. Excess mortality and cardiovascular events in patients surviving subarachnoid hemorrhage: a nationwide study in Sweden. *Stroke* 2011;42:902–7. [\[Crossref\]](#)