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Characteristics and outcomes of subarachnoid hemorrhage from vertebral artery dissection: A comparative study with other non-traumatic etiologies

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Abstract

Aim: Vertebral artery dissection (VAD) is a rare cause of non-traumatic subarachnoid hemorrhage (SAH) with significant clinical implications. This study compared the clinical characteristics and outcomes of SAH from intracranial VAD rupture to those from other etiologies, primarily aneurysmal rupture.

Methods: This single-center retrospective cohort study at Okayama University Hospital included patients with non-traumatic SAH diagnosed between 2019 and 2023. Patients were categorized into "VAD rupture" and "other etiologies" groups. The main outcome was clinical presentation and symptoms. Additional outcomes included ICU mortality, in-hospital mortality, and unfavorable outcomes at discharge and 6 months, defined as a modified Rankin Scale score of 3–6.

Results: A total of 66 patients were included, with 14 in the VAD rupture group and 52 in the other etiologies group. The VAD rupture group was younger (median age 49 vs. 64 years, $p = 0.003$) and had a higher incidence of out-of-hospital cardiac arrest (42.9% vs. 9.6%, $p = 0.011$). Preceding headache was more common in the VAD rupture group (78.6% vs. 11.5%, $p < 0.001$), with a median duration of 36 h before presentation. ICU and in-hospital mortality was higher in the VAD rupture group (both 50.0% vs. 19.3%, $p = 0.019$). No significant differences were found in unfavorable neurological outcomes at hospital discharge and 6 months.

Conclusions: VAD-related SAH often presents with prodromal headaches, severe symptoms like out-of-hospital cardiac arrest, and higher ICU and in-hospital mortality than other SAH causes, though long-term outcomes are similar. Larger, prospective studies are needed to refine interventions.

KEYWORDS
headache, intracranial aneurysm, prodromal symptoms, subarachnoid hemorrhage, vertebral artery dissection

INTRODUCTION

Accurate diagnosis of subarachnoid hemorrhage (SAH) poses a significant challenge, especially when patients present with mild, non-specific, or transient symptoms, or in cases of minimal hemorrhage or severe anemia at the time of computed tomography (CT) scanning in the emergency department.[1–4] These factors contribute to an

increased risk of misdiagnosis, potentially leading to fatal outcomes.

Aneurysmal rupture stands as the predominant cause of non-traumatic SAH[5]; however, intracranial vertebral artery dissection (VAD) also serves as an important etiological factor, though VAD is more commonly associated with ischemic strokes than hemorrhagic ones.[4] SAH resulting from VAD rupture is generally associated with a less favorable prognosis compared to saccular aneurysms, but this remains uncertain without thorough research[6,7]. Clinical manifestations of VAD often include head and/or neck pain similar to thunderclap headaches, leading to initial evaluation via plain head CT scans, which may not reveal any abnormalities. A previous large study of spontaneous VAD patients showed that 67% (114/169) presented with ischemic stroke, and only 2% (3/169) presented with SAH, with a median time interval from symptom onset to diagnosis of 4 days (range 2 h–88 days).[8]

Prior prospective investigations have primarily focused on identifying high-risk clinical features of SAH in patients presenting with acute headache, limiting their scope to aneurysmal or arteriovenous malformation causes identified through cerebral angiography,[9] potentially overlooking a broader spectrum of non-traumatic SAH etiologies. To date, there has been a paucity of research examining the symptomatology and history of present illness of SAH across different etiologies, including aneurysmal ruptures and VAD. A more thorough understanding of these etiology-specific symptoms could lead to earlier, etiology-based diagnoses and management. Moreover, this understanding might enable both patients and healthcare professionals to seek timely medical consultation and diagnostic evaluation for VAD, potentially preventing the catastrophic sequelae associated with its rupture. The aim of this study was to compare the clinical characteristics of non-traumatic SAH caused by intracranial VAD rupture with other etiologies, primarily aneurysmal rupture, focusing on their clinical presentation and outcomes.

METHODS

Study design, setting, and ethics

This was a single-center retrospective cohort study conducted at Okayama University Hospital, which is a tertiary facility that exclusively accepts emergency patients transported by emergency vehicles, without accepting walk-in patients. In 2023, the hospital accepted 4228 emergency vehicles.

The present study received approval from the Okayama University Hospital Ethics Committee (reference number K2407-014) and was conducted in accordance with the principles described in the Declaration of Helsinki. Given the retrospective nature of the study and the use of anonymous data, the ethics committee waived the requirement for written informed consent.

Participants

Patients were included if they were diagnosed with non-traumatic SAH in the emergency department at our hospital or if they were transferred from another hospital after being diagnosed with non-traumatic SAH between January 1, 2019 and December 31, 2023. Diagnoses were identified using the International Classification of Diseases, Tenth Revision (ICD-10) codes I600 to I609, which categorize non-traumatic SAH. Data were extracted from the Japanese Diagnosis Procedure Combination inpatient database. Patients were excluded if the diagnosis was not consistent with non-traumatic SAH following a thorough medical record and CT image check, if the etiologies were not identified due to cardiac arrest without stable return of spontaneous circulation, or if the etiology remained unknown after evaluation by CT angiography and subsequent digital subtraction angiography.

SAH diagnosis and clinical management

Once SAH was diagnosed with plain head CT, CT angiography was immediately performed to determine the etiology. Patients whose etiologies were identified were diagnosed within an hour of hospital admission. Endovascular or neurosurgical treatment was promptly initiated at the earliest feasible time. If a patient had a Hunt & Hess classification of grade 5 with dilated pupils, definitive treatment was withheld following thorough discussion among emergency and critical care physicians and neurosurgeons. Vasospasm was assessed using CT angiography, magnetic resonance angiography, or digital subtraction angiography during the spasm window. In principle, patients remained in the ICU for up to 14 days, during which time vasospasm could occur. After this period, they were either discharged to a general ward or transferred to another hospital, based on the discretion of the emergency and critical care physicians and neurosurgeons. However, patients who did not receive aggressive treatments receive end-of-life care or were discharged from the ICU earlier than 14 days.

Data collection

We collected the following data: age, sex, past medical history, hospital transfer (direct admission vs. transfer), presenting symptoms, prior hospital visits due to headache before presenting to our hospital, vital signs both upon emergency medical services arrival and hospital arrival, Hunt & Hess classification,[10] the modified Fisher scale,[11] etiologies of SAH, treatment modality (endovascular or neurosurgical treatment), complications (spasm), and outcomes. Information on the onset, clinical presentation, or chief complaint was retrieved through a thorough review of not only hospital medical records but also emergency

medical services records and referral letters if the patients were transferred from another hospital. Preceding headache was defined in this study as a headache persisting for over 1 h before seeking emergency medical services. The level of consciousness was recorded as the Japan Coma Scale score in prehospital settings and the Glasgow Coma Scale score upon hospital arrival.[12] The Japan Coma Scale score was then converted to the Glasgow Coma Scale score using a validated method.[13]

Grouping and outcomes

Study subjects were categorized based on the etiologies of SAH into a "VAD rupture" group and an "other etiologies" group. Definitive diagnoses and their etiologies were determined by an attending neurosurgeon and radiologist, based on findings from CT and CT angiography, and subsequent digital subtraction angiography if performed. The main outcome was the clinical presentation and symptoms. Additional outcomes included ICU mortality, in-hospital mortality, and modified Rankin Scale (mRS) scores at hospital discharge and at 6 months. An unfavorable outcome was defined as a mRS score of 3–6, indicating dependence or death.[14]

Statistical analyses

Continuous variables are presented as medians with interquartile ranges (IQRs), whereas categorical variables are reported as counts with percentages. The Mann–Whitney *U*-test was used to compare continuous variables between the two groups, and the Chi-squared test was employed for categorical variables. All tests were two-tailed, and a p value of <0.05 was deemed statistically significant. Analyses were performed using Prism 10.3.0 (GraphPad, San Diego, CA).

RESULTS

Over a 5-year period, of the 87 patients coded as having non-traumatic SAH, 66 were included in the study population, with 14 patients in the VAD rupture group and 52 patients in the other etiologies group (Figure 1).

Clinical characteristics, symptoms, and presentation

Table 1 demonstrates clinical characteristics, symptoms, and presentation of the study population. The VAD rupture group had a younger median age (49 vs. 64 years, $p = 0.003$). Three patients in the other etiologies group had a surgical history for SAH, all with a pre-stroke mRS of 0. A significantly higher proportion of patients in the VAD rupture group experienced out-of-hospital cardiac arrest (42.9% vs. 9.6%, $p = 0.011$). The GCS scores were significantly lower in the VAD rupture group, both at emergency medical services arrival (3 vs. 6, $p = 0.001$) and on hospital arrival (3 vs. 9, $p = 0.002$), corresponding with a worse Hunt & Hess grade in the VAD rupture group ($p = 0.018$).

Preceding headache was a common symptom in the VAD rupture group (78.6% vs. 11.5%, $p < 0.001$), with a median duration of 36 h before presentation. Additionally, more patients in the VAD rupture group reported a headache at presentation or immediately before loss of consciousness (92.9% vs. 38.5%, $p < 0.001$). The proportions of patients reporting posterior neck pain and nausea or vomiting were similar between the two groups. In the VAD rupture group, one patient underwent plane head CT at another hospital on

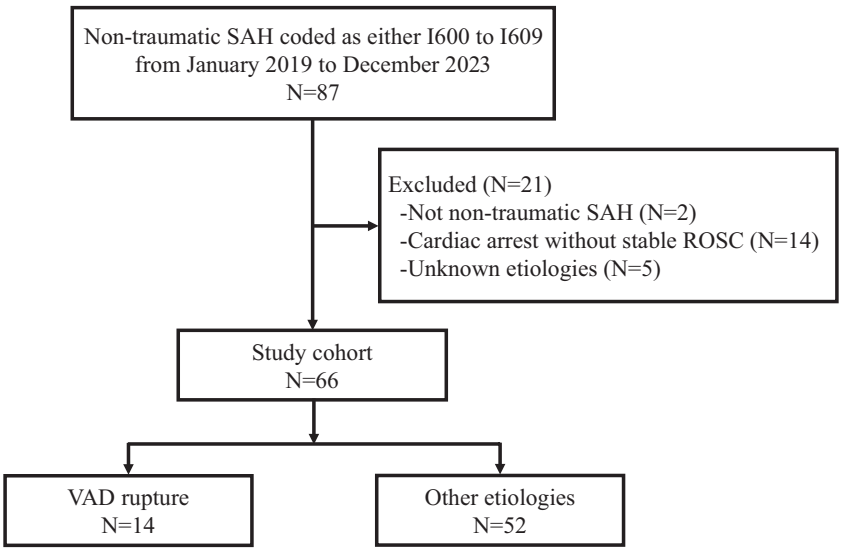

FIGURE 1 Flowchart of the study population. ROSC, return of spontaneous circulation; SAH, subarachnoid hemorrhage; VAD, vertebral artery dissection.

TABLE 1 Clinical characteristics, symptoms, and presentation of the study population.

	VAD rupture	Other etiologies	
	N = 14	*N* = 52	*p* Value
Age, median (IQR), year	49 (43, 51)	64 (49, 76)	0.003
Male sex, *n* (%)	7 (50.0)	17 (32.7)	0.38
Comorbidities or medical history, *n* (%)			
Hypertension	2 (14.3)	18 (34.6)	0.25
Diabetes mellitus	1 (7.1)	4 (7.7)	0.86
SAH	0 (0)	3 (5.8)	0.84
Unruptured intracranial aneurysm	0 (0)	8 (15.4)	0.27
Transfer from another hospital, *n* (%)	2 (14.3)	6 (11.5)	1.00
Out-of-hospital cardiac arrest, *n* (%)	6 (42.9)	5 (9.6)	0.011
Prehospital vital signs			
JCS, *n* (%)			0.040
Alert	0 (0)	3 (5.8)	
Single-digit	0 (0)	10 (19.2)	
Double-digit	0 (0)	8 (15.4)	
Triple-digit	14 (100)	31 (59.6)	
Converted GCS score, median (IQR)	3 (3, 3)	6 (3, 13)	0.001
Heart rate, median (IQR), beat/min	69 (0, 104)	76 (60, 92)	0.26
Systolic blood pressure, median (IQR), mmHg	90 (0, 173)	154 (133, 191)	0.052
Vital signs on hospital arrival			
GCS score, median (IQR)	3 (3, 6)	9 (3, 13)	0.002
Respiratory rate, median (IQR), cycle/min	10 (0, 15)	20 (15, 25)	0.001
Heart rate, median (IQR), beat/min	81 (0, 122)	79 (65, 91)	0.77
Systolic blood pressure, median (IQR), mmHg	119 (0, 158)	163 (139, 194)	0.011
GCS score change (Hospital minus EMS arrival), median (IQR)	0 (0, 3)	0 (−1, 2)	0.65
Hunt and Hess grade, *n* (%)			
1 (Mild headache)	0 (0)	3 (5.8)	0.018
2 (Moderate-to-severe headache)	1 (7.1)	8 (15.4)	
3 (Lethargy, confusion, or mild focal signs)	0 (0)	14 (26.9)	
4 (Stupor)	4 (28.6)	16 (30.8)	
5 (Coma)	9 (64.3)	11 (21.1)	
Prehospital symptoms, *n* (%)			
Preceding headache	11 (78.6)	6 (11.5)	<0.001
Duration, median (IQR), h	36 (3, 48)	10 (6, 57)	0.60
Headache	13 (92.9)	20 (38.5)	<0.001
Posterior neck pain	3 (21.4)	4 (7.7)	0.32
Nausea or vomiting	2 (14.3)	16 (30.8)	0.37
Sudden loss of consciousness, irrespective of its duration	12 (85.7)	20 (38.5)	0.005
Prior headache-related hospital visits before presentation, *n* (%)	2 (14.3)[a]	0 (0)	0.059

Abbreviations: GCS, Glasgow Coma Scale; IQR, interquartile range; JCS, Japan Coma Scale; SAH, subarachnoid hemorrhage; VAD, vertebral artery dissection.

[a]One patient underwent plane head CT at another hospital on the day of presentation, and the other on the day before presenting to our hospital.

the day of presentation, and the other on the day before presenting to our hospital; in both cases, the CT scan showed no abnormalities.

Radiologic features

Table 2 shows the modified Fisher scale and the detailed etiologies in the other etiologies group. No significant differences were observed in the modified Fisher scale between the VAD rupture and other etiologies groups. In the other etiologies group, the most common cause was the rupture of an anterior communicating artery aneurysm (28.8%), followed by middle cerebral artery aneurysm (23.1%) and internal carotid artery-posterior communicating artery aneurysm (21.2%).

Treatment and outcomes

Notably, no patients experienced rebleeding after admission. Table 3 presents the treatment and outcomes. The treatment modalities and other treatments were similar between the groups. ICU mortality and in-hospital mortality were significantly higher in the VAD rupture group compared to the other etiologies group (50.0 vs. 19.3%, $p = 0.019$). Unfavorable neurological outcomes, defined as mRS score of 3–6, and the distribution of mRS scores were similar between the groups at hospital discharge and at 6 months.

DISCUSSION

In this single-center retrospective cohort study, patients with non-traumatic SAH caused by intracranial VAD rupture more frequently experienced preceding headaches compared to those with other etiologies of non-traumatic SAH.

TABLE 2 Modified Fisher scale and details in the other etiologies group.

	VAD rupture	Other etiologies	
	N = 14	*N* = 52	*p* Value
Modified Fisher Scale, *n* (%)			
1 (Thin cisternal SAH + no IVH)	0 (0)	6 (11.5)	0.40
2 (Thin cisternal SAH + bilateral IVH)	2 (14.3)	3 (5.8)	
3 (Thick cisternal SAH + no IVH)	6 (42.9)	18 (34.6)	
4 (Thick cisternal SAH + bilateral IVH)	6 (42.9)	25 (48.1)	
Etiologies, *n* (%)			
Internal carotid artery aneurysm		5 (9.6)	N/A
Anterior communicating artery aneurysm		15 (28.8)	
Middle cerebral artery aneurysm		12 (23.1)	
Internal carotid artery-posterior communicating artery aneurysm		11 (21.2)	
Basilar artery aneurysm		6 (11.5)	
Arteriovenous malformation		3 (5.8)	

Abbreviations: IVH, intraventricular hemorrhage; SAH, subarachnoid hemorrhage; VAD, vertebral artery dissection.

They were also more likely to present with sudden loss of consciousness and to experience out-of-hospital cardiac arrest. Although ICU and in-hospital mortality was higher in the VAD rupture group, long-term neurological outcomes were similar between the two groups.

Our findings highlight the distinct clinical presentation of SAH associated with intracranial VAD rupture. Unlike aneurysmal SAH, which often presents abruptly without prodromal symptoms, SAH due to VAD rupture frequently follows a preceding headache, with a median duration of 36 h before the presentation of SAH. This headache is likely related to the initial dissection of the vertebral artery, which may progress over days before culminating in SAH. The literature supports this finding, with previous studies indicating that the median time from VAD symptom onset to diagnosis is approximately 4 days, suggesting a critical window during which the dissection may progress to either ischemic stroke or hemorrhagic stroke.[8] In cases of VAD, common symptoms include dizziness or vertigo, headache, and neck pain. However, no single clinical symptom is sufficiently common to exclude VAD when absent.[15] VAD rupture typically occurs in younger patients compared to other causes, particularly aneurysmal SAH, as shown in previous research.[15] Our findings further emphasize the need for vigilance, as two patients showed no abnormalities on plain head CT prior to SAH onset. Therefore, CT angiography or magnetic resonance angiography may be considered to rule out VAD in such cases. Although limited studies have examined the risk factors leading to SAH following VAD, a recent study indicated that posterior communicating artery hypoplasia may be associated with an increased risk of SAH due to VAD.[16]

The occurrence of sudden loss of consciousness and the high incidence of out-of-hospital cardiac arrest in patients with VAD-related SAH underscore the catastrophic nature of this condition. SAH-related cardiac arrest is generally believed to result from a massive release of catecholamines and a surge in sympathetic activity, which can lead to cardiac stunning and/or a sudden rise in intracranial pressure, potentially causing brainstem dysfunction.[17–19] Although there is no definitive explanation for the more frequent occurrence of cardiac arrest in VAD rupture compared to other etiologies, the pathophysiology behind these differences may be related to the anatomical location of the vertebral artery, which is situated adjacent to the brainstem. This proximity could lead to a more rapid rise in intracranial pressure or heightened sympathetic nerve activity, triggering a sudden loss of cardiac function and resulting in loss of consciousness or cardiac arrest.

The higher ICU and in-hospital mortality observed in the VAD rupture group could be attributed to the more severe initial presentation, as evidenced by the more frequent occurrence of out-of-hospital cardiac arrest and higher Hunt and Hess grades in this group. These findings highlight the critical nature of VAD-related SAH during the acute phase. While early mortality is higher, the comparable long-term neurological outcomes between the VAD rupture group and other etiologies of SAH suggest that recovery potential exists for those who survive the acute phase. However, given the significant differences in patient backgrounds, recovery trajectories and rehabilitation outcomes may vary considerably, warranting a cautious interpretation of these findings. This highlights the importance of aggressive early management and individualized care for patients with VAD-related SAH to optimize outcomes.[20,21]

This study has several limitations. First, it was conducted at a single tertiary care center that exclusively admits patients transported by emergency medical services, potentially limiting the generalizability of our findings to other settings. Second, the retrospective design and reliance on medical records may introduce bias, as the quality of data collection depends on the completeness and reliability of these records. Third, the sample size was relatively small, particularly in the VAD rupture group, which may reduce the statistical power of our analyses

TABLE 3 Treatment and outcomes.

	VAD rupture	Other etiologies	
	N = 14	*N* = 52	*p* Value
Treatment, *n* (%)			
Any aneurysm treatment	6 (42.9)	37 (71.2)	0.063
Endovascular	6 (42.9)	29 (55.8)	0.76
Clipping	0 (0)	8 (15.4)	0.57
Evacuation of intracerebral hemorrhage	0 (0)	1 (1.9)	1.00
Ventricular drainage	2 (14.3)	7 (13.5)	1.00
Lumbar cerebrospinal fluid drainage	5 (35.7)	27 (51.9)	0.44
Complications, *n* (%)			
Vasospasm	1 (7.1)	6 (11.5)	1.00
Outcomes, *n* (%)			
Brain death[a]	1 (7.1)	7 (13.5)	0.52
ICU mortality	7 (50.0)	10 (19.2)	0.019
In-hospital mortality	7 (50.0)	10 (19.2)	0.019
mRS at hospital discharge			
0	0 (0)	0 (0)	0.12
1	1 (7.1)	4 (7.7)	
2	0 (0)	4 (7.7)	
3	0 (0)	11 (21.2)	
4	1 (7.1)	8 (15.4)	
5	5 (35.7)	11 (21.2)	
6	7 (50.0)	10 (21.2)	
Missing	0 (0)	3 (5.7)	
mRS 3–6 at hospital discharge	13 (92.9)	41 (83.7)[b]	0.67
mRS at 6 months			
0	0 (0)	5 (9.6)	0.32
1	1 (7.1)	3 (5.7)	
2	1 (7.1)	5 (9.6)	
3	0 (0)	4 (7.7)	
4	0 (0)	2 (3.8)	
5	0 (0)	1 (1.9)	
6	10 (71.4)	16 (30.8)	
Missing	2 (14.3)	16 (30.8)	
mRS 3–6 at 6 months	10 (83.3)[c]	23 (63.9)[d]	0.37

Abbreviations: mRS, modified Rankin Scale; VAD, vertebral artery dissection.

[a]Clinical confirmation of brain death based on the presence of deep coma, bilateral pupil dilation, absence of brainstem reflexes, and a flat electroencephalography pattern.

[b]Three patients were missing.

[c]Two patients were missing.

[d]Sixteen patients were missing.

and preclude the use of multivariable logistic regression analysis. Fourth, this study was limited by missing data regarding long-term outcomes, which may have impacted our ability to fully assess the recovery trajectory of patients. Finally, our cohort was limited to patients with a confirmed diagnosis of non-traumatic SAH, which may limit the applicability of our findings to clinical scenarios where SAH is suspected but not yet confirmed or in patients with less obvious symptoms.

Despite these limitations, this study emphasizes the unique clinical characteristics of SAH due to VAD rupture, which differs significantly from SAH caused by other etiologies, primarily aneurysmal rupture. Recognizing these differences can aid in the timely diagnosis and management of VAD-related SAH, potentially reducing the risk of fatal outcomes associated with this condition.

CONCLUSIONS

Our study highlights the distinct clinical characteristics and challenges of VAD rupture as a cause of non-traumatic SAH. Patients with VAD-related SAH often present with prodromal headaches and severe symptoms, including out-of-hospital cardiac arrest, and experience higher ICU and in-hospital mortality rates compared to those with SAH from other causes, primarily aneurysmal rupture.

Early diagnosis of unruptured intracranial VAD is crucial for preventing fatal SAH. Despite the severity of initial symptoms, long-term neurological outcomes are similar across different etiologies. Larger, prospective, multicenter studies are needed to further explore these findings and develop targeted interventions for this high-risk group.

CONFLICT OF INTEREST STATEMENT

Dr. Atsunori Nakao is an Editorial Board member of AMS Journal and a co-author of this article. To minimize bias, they were excluded from all editorial decision making related to the acceptance of this article for publication.

DATA AVAILABILITY STATEMENT

The datasets from this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

Approval of the research protocol: The present study received approval from the Okayama University Hospital Ethics Committee (reference number K2407-014) and it conforms to the provisions of the Declaration of Helsinki. Given the retrospective nature of the study and the use of anonymous data, the ethics committee waived the requirement for written informed consent.
Informed consent: N/A.
Registry and registration no. of the study/trial: N/A.
Animal studies: N/A.

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REFERENCES

1. 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):866–9. https://doi.org/10.1001/jama.291.7.866
2. Vermeulen MJ, Schull MJ. Missed diagnosis of subarachnoid hemorrhage in the emergency department. Stroke. 2007;38(4):1216–21. https://doi.org/10.1161/01.STR.0000259661.05525.9a
3. Tarnutzer AA, Lee SH, Robinson KA, Wang Z, Edlow JA, Newman-Toker DE. ED misdiagnosis of cerebrovascular events in the era of modern neuroimaging. Neurology. 2017;88(15):1468–77. https://doi.org/10.1212/WNL.0000000000003814
4. Debette S, Leys D. Cervical-artery dissections: predisposing factors, diagnosis, and outcome. Lancet Neurol. 2009;8(7):668–78. https://doi.org/10.1016/S1474-4422(09)70084-5
5. Van Gijn J, Rinkel GJE. Subarachnoid haemorrhage: diagnosis, causes and management. Brain. 2001;124(2):249–78. https://doi.org/10.1093/brain/124.2.249
6. Santos-Franco JA, Zenteno M, Lee A. Dissecting aneurysms of the vertebrobasilar system. A comprehensive review on natural history and treatment options. Neurosurg Rev. 2008;31(2):131–40. https://doi.org/10.1007/s10143-008-0124-x
7. Chaalala C, El Hage G, Gilbert V, Martin T, Iancu D, Labidi M, et al. Spontaneous intracranial vertebral artery dissections presenting with subarachnoid hemorrhage. Neurochirurgie. 2024;70(3):101526. https://doi.org/10.1016/J.NEUCHI.2023.101526
8. Arnold M, Bousser MG, Fahrni G, Fischer U, Georgiadis D, Gandjour J, et al. Vertebral artery dissection: presenting findings and predictors of outcome. Stroke. 2006;37(10):2499–503. https://doi.org/10.1161/01.STR.0000240493.88473.39
9. Perry JJ, Stiell IG, Sivilotti MLA, Bullard MJ, Lee JS, Eisenhauer M, et al. High risk clinical characteristics for subarachnoid haemorrhage in patients with acute headache: prospective cohort study. BMJ. 2010;341(7781):1035. https://doi.org/10.1136/bmj.c5204
10. WE H, RM H. Surgical risk as related to time of intervention in the repair of intracranial aneurysms. J Neurosurg. 1968;28(1):14–20. https://doi.org/10.3171/JNS.1968.28.1.0014
11. Frontera JA, Claassen J, Schmidt JM, Wartenberg KE, Temes R, Connolly ES Jr, et al. Prediction of symptomatic vasospasm after subarachnoid hemorrhage: the modified fisher scale. Neurosurgery. 2006;59(1):21–7. https://doi.org/10.1227/01.NEU.0000243277.86222.6C
12. Yumoto T, Naito H, Yorifuji T, Aokage T, Fujisaki N, Nakao A. Association of Japan Coma Scale score on hospital arrival with in-hospital mortality among trauma patients. BMC Emerg Med. 2019;19(1):1–7. https://doi.org/10.1186/s12873-019-0282-x
13. Nakajima M, Okada Y, Sonoo T, Goto T. Development and validation of a novel method for converting the Japan Coma Scale to Glasgow coma scale. J Epidemiol. 2023;33(10):531–5. https://doi.org/10.2188/jea.JE20220147
14. 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(9488):809–17. https://doi.org/10.1016/S0140-6736(05)67214-5
15. Carpenter CR, Hussain AM, Ward MJ, Zipfel GJ, Fowler S, Pines JM, et al. Spontaneous subarachnoid hemorrhage: a systematic review and meta-analysis describing the diagnostic accuracy of history, physical examination, imaging, and lumbar puncture with an exploration of test thresholds. Acad Emerg Med. 2016;23(9):963–1003. https://doi.org/10.1111/acem.12984
16. Ariyada K, Shibahashi K, Fujika N, Sakakura Y, Hanakawa K, Murao M. Posterior communicating artery hypoplasia: a risk factor for vertebral artery dissection causing subarachnoid hemorrhage. J Stroke Cerebrovasc Dis. 2022;31(2):106224. https://doi.org/10.1016/J.JSTROKECEREBROVASDIS.2021.106224
17. Naredi S, Lambert G, Edén E, Zäll S, Runnerstam M, Rydenhag B, et al. Increased sympathetic nervous activity in patients with nontraumatic subarachnoid hemorrhage. Stroke. 2000;31(4):901–6. https://doi.org/10.1161/01.STR.31.4.901
18. Behrouz R, Sullebarger JT, Malek AR. Cardiac manifestations of subarachnoid hemorrhage. Expert Rev Cardiovasc Ther. 2011;9(3):303–7. https://doi.org/10.1586/erc.10.189
19. Zachariah J, Stanich JA, Braksick SA, Wijdicks EFM, Campbell RL, Bell MR, et al. Indicators of subarachnoid hemorrhage as a cause of sudden cardiac arrest. Clin Pract Cases Emerg Med. 2017;1(2):132–5. https://doi.org/10.5811/cpcem.2017.1.33061
20. Sugiu K, Tokunaga K, Watanabe K, Sasahara W, Ono S, Tamiya T, et al. Emergent endovascular treatment of ruptured vertebral artery dissecting aneurysms. Neuroradiology. 2005;47(2):158–64. https://doi.org/10.1007/s00234-005-1341-4
21. Haruma J, Sugiu K, Ebisudani Y, Kimura R, Edaki H, Yamaoka Y, et al. Endovascular treatment for intracranial artery dissections in posterior circulation. J Neuroendovasc Ther. 2024;18(3):92–102. https://doi.org/10.5797/jnet.ra.2023-0068

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