Journal of Multidisciplinary Healthcare

Dovepress
Taylor & Francis Group

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ORIGINAL RESEARCH

# Risk Factors for Cognitive Impairment Following Angiographically Negative Subarachnoid Haemorrhage Around the Midbrain

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**Objective:** This study aimed to explore the risk factors for cognitive impairment caused by angiographically negative subarachnoid haemorrhage (SAH).

**Methods:** This retrospective study employed a convenience sampling method to select patients with negative SAH in the midbrain who were admitted to the neurosurgery department of our hospital between September 2018 and September 2023. A total of 69 patients with angiographically negative SAH were enrolled and divided into the cognitive impairment group (n = 16) and the non-cognitive impairment group (n = 53). General demographic and clinical data were collected, and patients' cognitive function was assessed using the Montreal Cognitive Assessment scale. The risk factors of the cognitive impairment caused by angiographically negative SAH were identified by logistic regression analysis.

**Results:** The results of the univariate analysis showed that there were statistically significant differences ($p < 0.05$) between the two groups of patients in terms of age, consciousness disorders, history of hypertension, ventricular haemorrhage, concurrent hydrocephalus, Glasgow Coma Scale score, Hunt–Hess grading (≥3) and Fisher grading (≥3). The logistic regression results showed that age ($p = 0.031$), degree of consciousness impairment ($p = 0.023$), Hunt–Hess grading ($p = 0.019$), presence of hydrocephalus ($p = 0.002$) and presence of ventricular haemorrhage ($p = 0.021$) were independent risk factors for cognitive impairment after angiographically negative SAH ($p < 0.05$).

**Conclusion:** Age, degree of consciousness impairment, Hunt–Hess grade (≥3), concomitant ventricular haemorrhage and hydrocephalus are risk factors for cognitive function after angiographically negative SAH.

**Keywords:** subarachnoid haemorrhage, computed tomography angiography, digital subtraction angiography, cognitive impairment

## Introduction

Subarachnoid haemorrhage (SAH) refers to a clinical syndrome in which blood flows into the subarachnoid space directly due to the rupture of diseased blood vessels at the bottom or surface of the brain; it accounts for approximately 10% of acute stroke. It is a very serious and common disease.[1] Approximately 61.7% of cases of spontaneous SAH are caused by ruptured intracranial aneurysms, which have a high mortality and poor prognosis.[2] Negative SAH refers to SAH cases in which no bleeding source (such as an aneurysm or arteriovenous malformation) is identified on the computed tomography angiography (CTA) or digital subtraction angiography (DSA) imaging, and hence SAH is ruled out in the patient. Negative SAH around the midbrain accounts for approximately 15% of spontaneous SAH. Until now, there has been no evidence from various diagnostic examinations to identify the cause of bleeding.[3] Cognitive impairment is one of the main clinical complications in surviving patients with SAH, characterised by impaired memory, learning ability, attention, executive ability, orientation and language function. Both cognitive and neurological impairments affect the prognosis of patients with SAH. However, some patients cannot return to normal work and life because their abilities have been impaired due to cognitive dysfunction, even though they may have good recovery of neurologic

Received: 17 July 2024
Accepted: 23 January 2025
Published: 12 March 2025

function.[4] Negative SAH around the midbrain is generally considered to have a good prognosis. However, approximately 25% of patients may experience cognitive dysfunction after the treatment,[5] which can seriously affect their quality of life and ability to reintegrate into society.

Although the specific mechanisms underlying cognitive dysfunction following SAH remain unclear, numerous studies have identified factors associated with cognitive impairment in patients with SAH. These factors include global brain oedema, lateral hemisphere infarction, low admission Glasgow Coma Scale (GCS) score (Hunt and Hess grade ≥ 3, Fisher grade ≥ 3), aneurysms in the anterior communicating and anterior cerebral arteries, extensive bleeding in the anterior and lateral longitudinal fissures, epilepsy, advanced age and limited education levels[6,7.] However, the precise cause of cognitive impairment linked to negative SAH near the midbrain is still unknown.

This study retrospectively analyses 69 cases of midbrain negative SAH in the The First People's Hospital of Tianshui between September 2018 and September 2023 and explores the risk factors for cognitive impairment caused by midbrain negative SAH. The study aims to provide suggestions for the clinical treatment of negative SAH in the midbrain.

# Participants and Method

## Research Participants

This single-centre study adopted a retrospective design and the convenience sampling method to select patients with negative SAH in the midbrain who were admitted to The First People's Hospital of Tianshui neurosurgery department between September 2018 and September 2023 as the participants. The inclusion criteria were as follows: (1) aged 30–75 years; (2) head computed tomography (CT) scan indicating SAH; (3) no source of bleeding detected on CTA or DSA examinations; (3) time from bleeding onset to hospital admission <72 hours; (4) admission to the neurosurgery department. The exclusion criteria were as follows: (1) history of traumatic brain injury; (2) previous cognitive impairment, language or hearing disabilities; (3) inability to complete neuropsychological scale assessments; (4) history of severe stroke (eg cerebral infarction, cerebral haemorrhage, SAH), traumatic brain injury, surgery, psychiatric disorders or epilepsy; (5) findings of aneurysms, moyamoya disease or cerebral vascular malformations during cerebrovascular examination.

Screening was conducted by a team of evaluators, with each case reviewed independently by at least two evaluators to ensure consistency. An inter-rater reliability check was performed to confirm agreement across evaluators, helping to maintain a homogeneous sample by minimising subjective variance in applying the inclusion and exclusion criteria. A total of 69 patients with angiographically negative SAH were enrolled and divided into the cognitive impairment positive group (n = 16) and the cognitive impairment negative group (n = 53). This study was approved by the hospital's ethics approval committee. Patients and their families were provided with detailed information regarding the research procedures and potential risks, ensuring that they fully understood the implications of their participation. Informed consent was obtained from both patients and their families, particularly for those with cognitive impairment, ensuring that appropriate measures were taken to assess their capacity to consent.

## Data Collection

### General and Clinical Data Collection

General information of the patients (including age, gender, smoking history, drinking history and hypertension history) and clinical data (including past history, onset time, Fisher grade, haematoma size, GCS score, Hunt–Hess grade, whether puncture and drainage surgery was performed, whether ventricular haemorrhage was complicated [using the Verma classification system] and laboratory testing indicators) were collected from the hospital's electronic medical record system. The flowchart is shown in Figure 1. "Complex ventricular haemorrhage" refers to a pathological state of intraventricular haemorrhage that involves multiple regions of the ventricular system and is associated with a larger volume of bleeding and often accompanies severe complications and a high degree of treatment difficulty. This type of haemorrhage is not limited to a single ventricle but may involve multiple areas, such as the lateral, third and fourth ventricles, and may even accompany intraparenchymal haemorrhage. Compared with general intraventricular haemorrhage, complex ventricular haemorrhage is often accompanied by a greater volume of bleeding, leading to significant expansion of the ventricular system, compression of surrounding brain

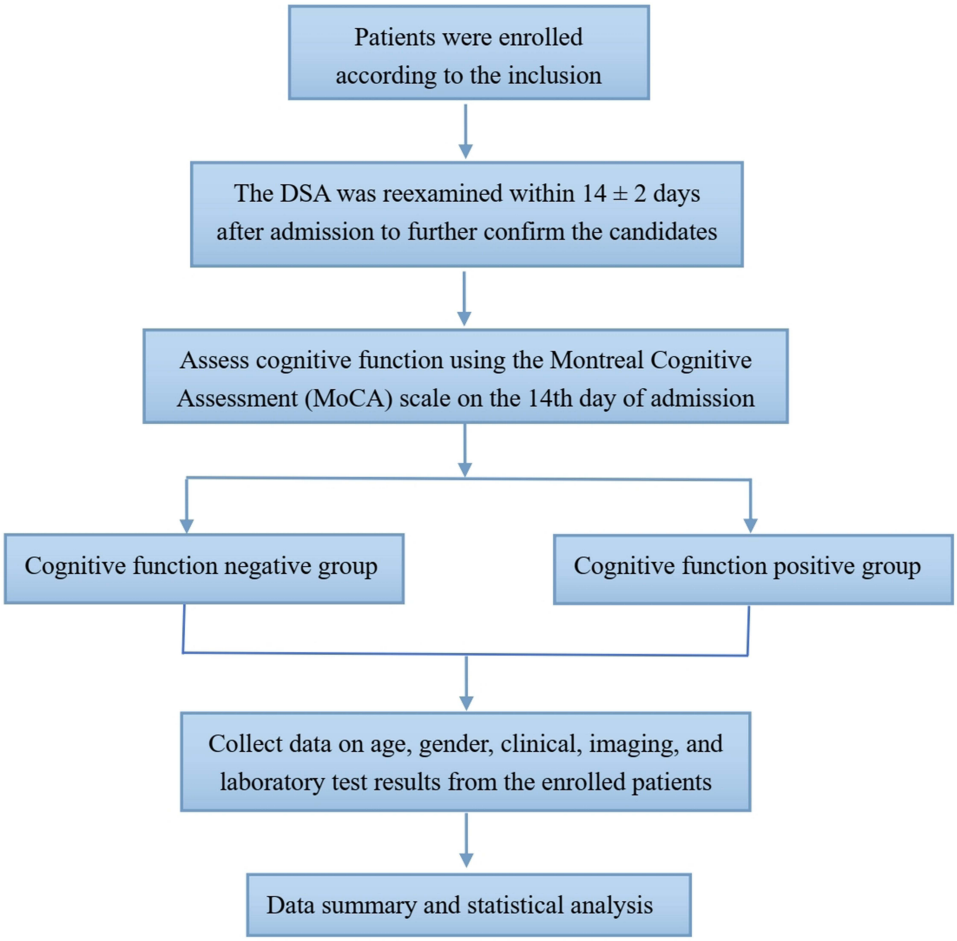


**Figure 1** The flowchart.

tissue and disruption of normal cerebrospinal fluid circulation. Patients with this condition are more prone to severe complications, such as hydrocephalus, ventriculitis and intracranial hypertension.

## Cognitive Impairment

During the fourth week of treatment, all patients' cognitive function was assessed using the Montreal Cognitive Assessment (MoCA) scale,[8] a tool widely recognised for its effectiveness in detecting mild cognitive impairment (MCI). The MoCA was chosen over other cognitive scales, such as the Mini-Mental State Examination, due to its comprehensive evaluation of cognitive domains relevant to patients with SAH, including attention, concentration, executive function, memory, language, visuospatial skills, abstract reasoning and both computational and directional abilities.

The MoCA scale has a maximum score of 30 points, with scores ≥26 indicating normal function and scores <26 suggesting impairment A team of trained neuropsychologists administered the assessment to ensure consistency. Additionally, to minimise inter-observer variability, an inter-rater reliability check was conducted, and all evaluators underwent standardised training sessions on administering and interpreting MoCA scores.

### Evaluation of Consciousness Disorders

According to the GCS, which includes three aspects (eye-opening response, language response and limb movement), the degree of consciousness impairment can be divided into drowsiness, coma, light coma, moderate coma and deep coma. The total score range of the GCS scale is 3–15 points, with 15 points indicating clear consciousness, 13–14 points indicating mild consciousness disorder, 9–12 points indicating moderate consciousness impairment, 3–8 points indicating severe consciousness disorder, <8 indicating a comatose state and <3 indicating deep coma or brain death.


https://doi.org/10.2147/JMDH.S487479

### Fisher Classification

The Fisher classification grades SAH into four levels, mainly based on CT performance, to evaluate the occurrence of symptomatic vasospasm. The levels are as follows: level 1 – there is no bleeding observed on CT, and the incidence of vascular spasm is up to 21%; level 2 – there is diffuse bleeding observed on CT without blood clots and the incidence of vascular spasm is up to 25%; level 3 – there are blood clots or thick blood accumulation, the vertical thickness is >1cm (cerebral longitudinal fissure, insular cistern, annular cistern) or the horizontal length and width is >(5 × 3 mm) (lateral fissure cistern, plantar cistern), and the incidence of vascular spasm is up to 37%; level 4 – there are intracerebral haematoma or intraventricular haemorrhage in the brain, but there is no or a small amount of diffuse bleeding in the basal cistern, which shows the incidence of vascular spasm is up to 31%.

### Hunt–Hess Grading System

This system adopts a I–V grading scale, as follows: Grade I – asymptomatic or mild headache; Grade II – moderate-to-severe pain, meningeal irritation and cerebral nerve paralysis; Grade III – drowsiness, confusion of consciousness and mild focal neurological signs; Grade IV – coma, moderate-to-severe hemiplegia, early brain removal and rigidity or autonomic dysfunction; Grade V – coma, cerebral rigidity or other symptoms or state near death. The presence or absence of intraventricular haemorrhage and hydrocephalus should be determined based on imaging data (see Figures 2 and 3).

## Statistical Methods

The SPSS 26.0 software package (IBM, USA) was used to analyse the collected data. The figures conforming to normal distribution were analysed using mean ± standard deviation ($\bar{x} \pm s$), and the *t*-test was used for inter-group comparison. The non-parametric rank sum Z-test was used for count data, while count data were analysed using $\chi^2$ inspection. Using cognitive impairment as the dependent variable and statistically significant variables in the univariate analysis as independent variables, a multivariate logistic regression model was constructed to analyse the influencing factors of SAH around the midbrain. A *p*-value of <0.05 indicated a statistically significant difference. According to the current and sample size and statistical threshold (0.05), the statistical analysis power of this study through Gpower software was 73–77%.

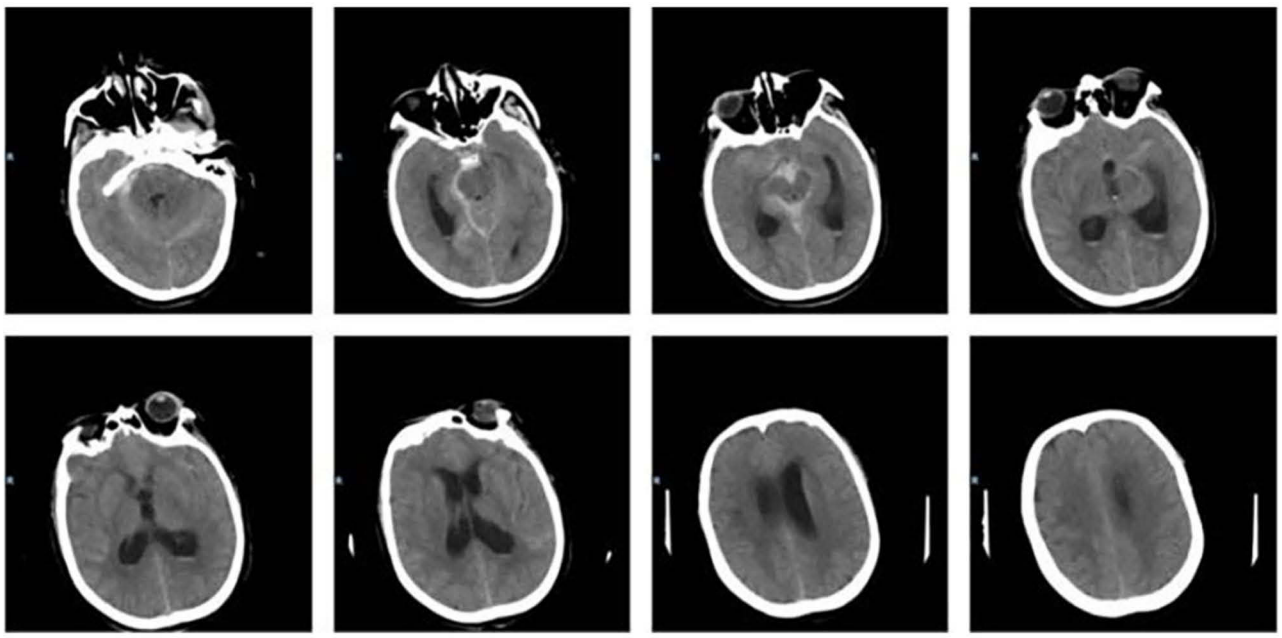

**Figure 2** Imaging data of ventricular hemorrhage. CT showed negative SAH around the midbrain, hemorrhage ruptured into the ventricles, and ventricular hematocele.

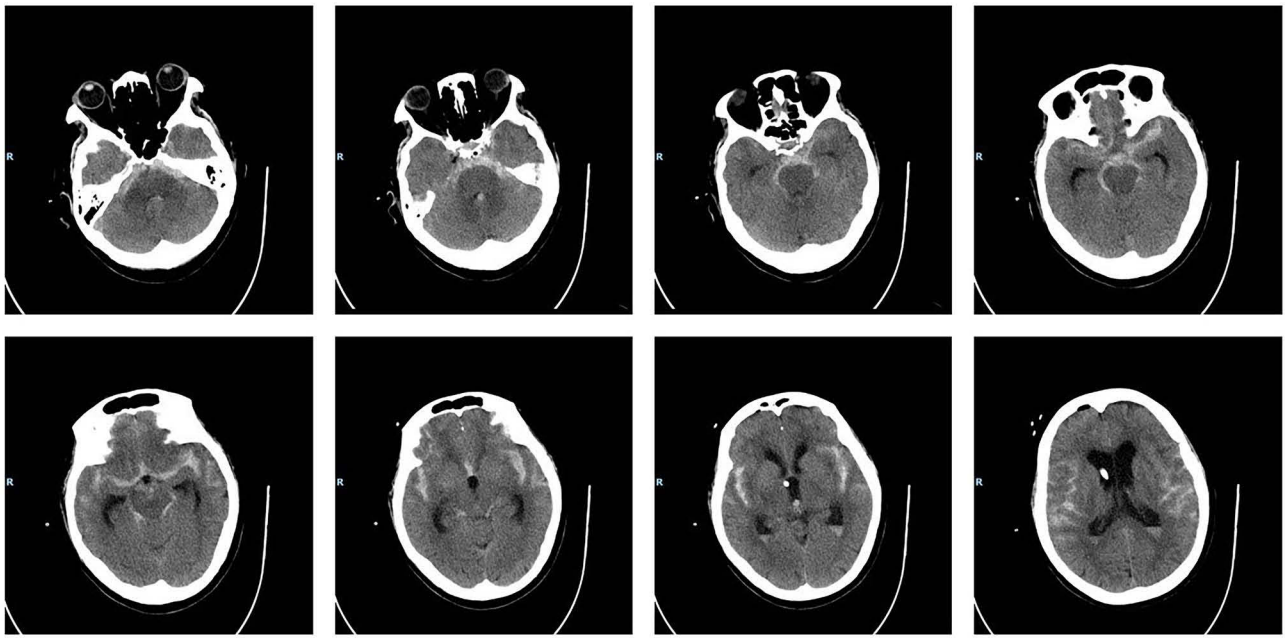

**Figure 3** Imaging data of hydrocephalus. CT showed SAH around the midbrain, in the lateral fissure, hemorrhage ruptured into the ventricles, ventriculomegaly, and obstructive hydrocephalus formation.

# Results

## The Situation of Cognitive Dysfunction in Patients with Negative Subarachnoid Haemorrhage Around the Midbrain

Among the total 69 cases of negative SAH, 16 cases were assessed to have cognitive impairment by the Montreal Scale in the fourth week of treatment, and the rate of cognitive impairment was 23.1% (with 16 cases in the positive group; the score was 21.0 ± 0.6).

## Univariate Analysis of Cognitive Impairment in Patients with Negative Subarachnoid Haemorrhage Around the Midbrain

The majority of the patients in the negative group and positive group were female, accounting for 52.8% and 56.2%, respectively. The average age of patients in the negative group was 54.68 ± 16.31 years, and in the positive group, it was 57.54 ± 19.35 years. The GCS scores of the two groups were 14.27 ± 1.02 and 9.78 ± 1.24, respectively. In the negative group, the proportion of drowsiness as the main consciousness disorder was 77.4%, while in the positive group, the proportion was 43.8%. The proportion of patients in the negative group who did not experience ventricular haemorrhage was the highest (88.7%), while the proportion of patients in the positive group who experienced ventricular haemorrhage was the highest (56.2%). The patients in the negative group did not experience concurrent hydrocephalus; the assessment results showed Hunt–Hess grade <3 and Fisher grade <3. In the positive group, approximately 50% of patients had concurrent hydrocephalus, almost 12.5% of patients had a Hunt–Hess grade ≥3 and 31.2% had a Fisher grade ≥3. There were statistically significant differences ($p < 0.05$) between the two groups of patients in terms of age, consciousness disorders, history of hypertension, ventricular haemorrhage, concurrent hydrocephalus, GCS score, Hunt–Hess grading (≥3) and Fisher grading (≥3). There was no statistically significant difference between the two groups in terms of gender, puncture and drainage surgery, history of cerebral haemorrhage or smoking history ($p > 0.05$) (see Table 1).


https://doi.org/10.2147/JMDH.S487479

**Table 1** Characteristics of Cognitive Dysfunction in Patients with Negative SAH Around the Midbrain

| Variable | Negative Group (n = 53) | Positive Group (n = 16) | $t/X^2$ | *P* |
|---|---|---|---|---|
| Gender | | | 0.058 | 0.810 |
| Male | 25(47.2%) | 7(43.8%) | | |
| Female | 28(52.8%) | 9(56.2%) | | |
| Age(years) | 54.68±16.31 | 57.54±19.35 | 10.113 | 0.031 |
| Smoking history | | | 0.110 | 0.740 |
| Yes | 24(45.3%) | 8(50.0%) | | |
| No | 29(54.7%) | 8(50.0%) | | |
| History of hypertension | | | 6.614 | 0.013 |
| Yes | 21(39.6%) | 12(75.0%) | | |
| No | 32(60.4%) | 4 (25.0%) | | |
| GCS score | 14.27±1.02 | 9.78±1.24 | 5.152 | 0.025 |
| Disturbance of consciousness | | | 13.825 | 0.003 |
| Somnolence | 41(77.4%) | 6(37.5%) | | |
| Lethargy | 12(22.6%) | 7(43.8%) | | |
| Light coma | 0(0%) | 1(6.2%) | | |
| Coma | 0(0%) | 2(12.5%) | | |
| Deep Coma | 0(0%) | 0(0%) | | |
| Ventricular haemorrhage | | | 12.061 | 0.001 |
| Yes | 6(11.3%) | 9 (56.2%) | | |
| No | 47(88.7%) | 7 (43.8%) | | |
| Combined hydrocephalus | | | 25.296 | <0.001 |
| Yes | 0(0%) | 8(50.0%) | | |
| No | 53(100%) | 8(50.0%) | | |
| Hunt-Hess grade (≥3) | | | 6.823 | 0.009 |
| Yes | 0(0%) | 2(12.5%) | | |
| No | 53(100%) | 14(87.5%) | | |
| Puncture drainage | | | 0.660 | 0.417 |
| Yes | 27(50.9%) | 10(62.5%) | | |
| No | 26(49.1%) | 6(37.5%) | | |
| Fisher grade (≥3) | | | 6.510 | 0.010 |
| Yes | 0(0%) | 5(31.2%) | | |
| No | 53(100%) | 11(68.8%) | | |
| Erythrocyte index | | | | |
| Mean corpuscular hemoglobin (pg) | 28.68±2.58 | 27.01±0.97 | 1.383 | 0.128 |
| Mean corpuscular volume (fl) | 83.46±1.79 | 79.78±1.14 | 2.112 | 0.082 |

## Multivariate Analysis of Risk Factors for Cognitive Impairment in Patients with Negative Subarachnoid Haemorrhage Around the Midbrain

Significant variables in Table 1 were included in the multivariate logistic analysis. A multivariate logistic regression model was constructed with cognitive impairment as the dependent variable and age, consciousness impairment, ventricular haemorrhage, concomitant hydrocephalus, GCS score, Hunt–Hess grade (≥3), history of hypertension and Fisher grade (≥3) as independent variables. The results showed that age (odds ratio [OR]: 2.154, 95% confidence interval [CI]: 1.515–4.573, $p = 0.038$), degree of consciousness impairment (OR: 1.956, 95% CI: 1.106–3.872, $p = 0.023$), Hunt–Hess grade ≥3 (OR: 2.468, 95% CI: 1.004–4.139, $p = 0.019$), concomitant ventricular haemorrhage (OR: 2.139, 95% CI: 1.497–5.959, $p = 0.021$) and hydrocephalus (OR: 1.918, 95% CI: 1.013–3.374, $p = 0.002$) were all independent risk factors of cognitive impairment after negative SAH in the midbrain (see Table 2).

**Table 2** Multivariate Analysis of Risk Factors for Cognitive Dysfunction in Patients with Negative SAH Around the Midbrain

| Influencing Factors | *B* | *SE* | *OR* | *95% CI* | *P* |
|---|---|---|---|---|---|
| Age | 2.763 | 1.157 | 2.154 | 1.515~4.573 | 0.038 |
| Consciousness impairment | 4.001 | 1.359 | 1.956 | 1.106~3.872 | 0.023 |
| Hunt-Hess grade | 4.236 | 1.523 | 2.468 | 1.004~4.139 | 0.019 |
| Ventricular haemorrhage | 2.765 | 1.082 | 2.139 | 1.497~5.959 | 0.021 |
| Hydrocephalus | 3.117 | 1.549 | 1.918 | 1.013~3.374 | 0.002 |
| Hypertension | −0.214 | 1.326 | 2.011 | 0.069~1.691 | 0.116 |
| GCS score | −0.382 | 0.927 | 0.782 | 0.438~3.691 | >0.05 |
| Fisher grade | 1.827 | 1.124 | 3.674 | 0.746~2.387 | >0.05 |

## Discussion

Cognitive impairment is one of the main clinical complications of patients with SAH, which is characterised by impairment in memory, learning ability, attention, executive ability, orientation and language function. Both cognitive dysfunction and neurological dysfunction affect the prognosis of patients with SAH. Although patients with negative peripheral SAH in the midbrain have good neurological recovery, they are unable to return to work and society due to cognitive impairment; their quality of life and social abilities are impaired due to cognitive dysfunction.[9,10] Therefore, identifying risk factors of negative SAH around the midbrain and immediately intervening in these factors can greatly improve the recovery of this type of patient. This study focuses on negative SAH around the midbrain, which is an area that has received less attention in previous research. This particular type of SAH exhibits clinical symptoms even without obvious signs of bleeding, making the selection of this specific group for study significantly innovative. The study also introduces the MoCA scale to evaluate patients' cognitive function, which demonstrates a certain degree of innovation in assessing MCI. Furthermore, the study identifies multiple risk factors for cognitive dysfunction after negative SAH around the midbrain, such as age and degree of consciousness disturbance. These new findings provide a new perspective and basis for clinical treatment.

This research has shown that there is no significant difference between men and women in the effects of negative SAH around the midbrain on cognitive impairment. A neurosurgery study at the University of Helsinki Hospital also testified that there is no significant difference between men and women in the occurrence of cognitive impairment at 3 months after negative SAH around the midbrain, which is consistent with these research findings.[11] Advanced age is a risk factor for cognitive impairment after stroke.[12] The incidence of cognitive impairment in patients aged >60 years with negative subarachnoid space around the midbrain is significantly higher than in patients aged <60 years. Elderly patients with degenerative changes in brain cell activity and decreased blood flow quite possibly suffer from neurological deficits after negative SAH around the midbrain and cerebral cell ischaemia caused by intracranial hypertension. The learning and functional rehabilitation capabilities of elderly people are all decreasing, which affects the recovery of neurological function and can easily lead to cognitive dysfunction.

The consciousness disorders brought on by negative SAH around the midbrain and dysfunction of high levels of Hunt–Hess grading are mainly due to direct compression of the brain stem by haematoma and secondary hydrocephalus by bleeding.[13,14] Hunt–Hess grading is one of the commonly used methods in clinical practice to assess the severity and prognosis of SAH and is a method used to choose the time of radiography and operations and judge the therapeutic effects. At present, most studies believe that the Hunt–Hess grading can be used to foretell the patient's prognosis; the higher the grading is, the worse the patient's prognosis.[15] The negative SAH is just around the midbrain, which means the more bleeding is, the more severe the compression on the brainstem, the slower the absorption and the longer the compression on the brainstem, which leads to worse healing and more severe cognitive impairment.[16] Acute obstructive hydrocephalus can cause a state of high intracranial pressure, which affects intracranial blood perfusion and leads to ischaemic damage to brain tissue. Prolonged cerebral ischaemic perfusion causes irreversible damage to brain function, resulting in cognitive function after treatment.[17] Our study found that patients with concurrent hydrocephalus generally

experience cognitive impairment even after being treated with lateral ventricular puncture and drainage. Therefore, more attention should be paid to hydrocephalus.

Negative SAH around the midbrain combined with intraventricular haemorrhage is an important factor of cognitive dysfunction in patients with cerebral haemorrhage. In this situation, the amount of bleeding is large, and the pressure is high. When blood flows into the ventricles, it can block the ventricular system and the arachnoid particles, causing a disturbance of cerebrospinal fluid circulation. In addition, the secondary damage caused by toxic substances, inflammatory reactions, free radicals and matrix metalloproteinase leads to neuronal cell apoptosis and cognitive impairment.[18,19] Patients with negative SAH and ventricular haemorrhage around the midbrain experience cognitive impairment 4.9 times higher than those without ventricular haemorrhage. Moreover, patients with concurrent hydrocephalus also have ventricular haemorrhage, which should be a mild manifestation of hydrocephalus. Increased bleeding or blockage of the ventricular pathway can develop into hydrocephalus. Hypertension is generally regarded as a risk factor for cognitive impairment in many stroke cases, but according to our study, there is no association between cognitive impairment caused by negative SAH around the midbrain and high blood pressure.[20]

The GCS is currently the most widely used coma scoring system in the world; it is based on the patient's eye-opening, language function and limb activity and is especially used for the evaluation of consciousness level and degree of injury in patients with craniocerebral injury.

However, due to its defectiveness in the evaluation of neurological function, it is seldom used in haemorrhagic cerebrovascular diseases, such as cerebral haemorrhage and SAH. In recent years, some scholars have attempted to apply GCS assessment to the diagnosis and treatment of hypertensive intracerebral haemorrhage, which has some clinical value.[21] After SAH, the patient's intracranial pressure increases sharply, leading to a decrease in cerebral blood flow and insufficient perfusion of brain tissue, which causes structural and functional damage to the brain and leads to clinical manifestations, such as consciousness disorders. This is also one of the possible mechanisms of cognitive impairment after SAH.[22] This study also found a certain correlation between GCS scores and cognitive impairment. Therefore, patients with lower GCS scores after SAH should be prepared to have an operation and treatment of the primary disease, as well as accept the treatment of dehydration and anti-vasospasm. If necessary, methods such as lateral ventricular puncture can also be adopted to reduce intracranial hypertension and relieve nerve damage, which has positive significance in preventing and treating patient's cognitive impairment during the acute phase.

This study has some limitations. First, the study is conducted in a single centre with a small sample size, which may limit the generalisability of the findings to other populations. Second, the study uses a convenience sampling method, which could introduce selection bias. This may affect the external validity of the results, making it difficult to generalise the findings to other populations. Third, ideally, the rates of cognitive impairment in all treated patients with SAH should be reported to make a comparison with negative SAH around the midbrain group. Fourth, the multivariate analysis was not adjusted by potential confounding variables (eg educational background, baseline cognitive function). Additionally, cognitive deficits after SAH can become apparent within a few weeks to months post-injury. Longer follow-up data should be included to observe the persistence or progression of cognitive impairment. Finally, the data need to be compared with more cases from other centres.

## Conclusion

Age, degree of consciousness impairment, Hunt–Hess grade ≥3, concomitant ventricular haemorrhage and hydrocephalus are significant risk factors that influence cognitive function following negative SAH in the midbrain. Physicians must closely monitor and manage hydrocephalus and ventricular bleeding in these patients, arranging for early drainage when necessary. Special attention should be given to patients aged >60 years, as early intervention may help mitigate the occurrence and severity of cognitive impairment. Furthermore, additional research is needed to investigate whether early intervention in high-risk patients – particularly those with hydrocephalus or impaired consciousness – could further reduce the incidence or severity of cognitive impairment. Finally, it is essential to acknowledge the need to explore additional variables, such as genetic predisposition, baseline cognitive function and long-term recovery trajectories, to achieve a comprehensive understanding of the research landscape.

## Data Sharing Statement

All data generated or analysed during this study are included in this article.

## Ethics Approval and Consent to Participate

This study was conducted in accordance with the Declaration of Helsinki and approved by the ethics committee of The First People's Hospital of Tianshui. Ethic Reference Number: TYYLSK2024-09. Written informed consent was obtained from all participants.

## Funding

This research is supported by Tianshui City Science and Technology Support Plan Project Assignment (2022-SHFZKJK-5063).

## Disclosure

The authors report no conflicts of interest in this work.

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