

Shunt-Dependent Hydrocephalus After Aneurysm Subarachnoid Hemorrhage: Predictors and its Relationship with Lumbar Puncture

Kaiwen Shi

Fujian Medical University Union Hospital

Zuan Yu

Fujian Medical University Union Hospital

Songsheng Shi

Fujian Medical University Union Hospital

Jin Chen (✉ xhswcj@163.com)

Fujian Medical University Union Hospital

Research Article

Keywords: Aneurysm, Hydrocephalus, Lumbar puncture, Subarachnoid hemorrhage, Shunt dependence

Posted Date: July 6th, 2021

DOI: <https://doi.org/10.21203/rs.3.rs-671484/v1>

License:  This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Abstract

Objective

To identify predictors of shunt-dependent hydrocephalus (SDHC) after aneurysmal subarachnoid hemorrhage (aSAH) and determine the effect of postoperative lumbar puncture (LP) on the incidence of SDHC.

Methods

We conducted a retrospective review of consecutive patients with aSAH who underwent surgical clipping or endovascular embolization for cerebral aneurysm in our hospital from October 2009 to May 2019. Univariate and multivariate analyses were performed to characterize risk factors for SDHC after aSAH. The incidence of SDHC in the LP and No LP groups were compared after propensity score matching analysis.

Results

A total of 414 patients were enrolled in this study. SDHC developed in 59 patients (14.3%). On multivariate analysis, age > 55 years ($P = 0.004$), intraventricular hemorrhage (IVH) ($P = 0.038$), Fisher grade ≥ 3 ($P = 0.043$), Hunt and Hess grade (H&H) ≥ 3 ($P = 0.027$), acute hydrocephalus ($P = 0.001$), central nervous system infection (CNSI) ($P = 0.021$), placement of external ventricular drainage (EVD) ($P = 0.044$), and a history of postoperative LP ($P < 0.016$) were independently associated with SDHC after aSAH. Propensity score matched cohorts exhibited a significantly higher incidence of SDHC after LP treatment than that without LP (20.7% vs 12.1%, $P = 0.019$). Receiver operating characteristic analysis revealed an area under the curve of 0.818 for the final regression model.

Conclusion

Evaluating risk factors can help identify patients at high risk of developing SDHC. Postoperative LP may increase the incidence of SDHC after aSAH. Strategies for treatment of ruptured aneurysms should be used to mitigate SDHC and minimize poor outcomes.

Introduction

Spontaneous subarachnoid hemorrhage (SAH) accounts for 5–10% of all stroke cases and is mainly caused by an intracranial aneurysm rupture hemorrhage (70–80%)[1–3]. This phenomenon is called an aneurysmal subarachnoid hemorrhage (aSAH), and it has a high incidence and mortality of the nervous system, affecting about 2 to 16 per 100000 people each year[4]. Most survivors face a higher risk of complications, including delayed cerebral ischemia, epilepsy, nosocomial infections, cognitive

impairment, SDHC, and shunt-related complications. As many as 6–87% of aSAH patients develop acute hydrocephalus[5]. Permanent CSF diversion, such as through ventriculoperitoneal (VP) shunting, is required in 9–31% of aSAH patients due to chronic hydrocephalus[6–8]. Shunting procedures can be associated with high rates of revision (13–32%) due to complications such as infection, mechanical obstruction, and under or overdrainage[9–11]. Increasing data suggest that early aneurysm repair, together with aggressive management of complications such as hydrocephalus and delayed cerebral ischemia, leads to improved functional outcomes[12]. We need to continually reassess which interventions provide the greatest benefit to patients. Theoretically, the release of CSF containing erythrocytes and inflammatory cytokines by external drainage can reduce the stimulation to the subarachnoid space, decrease intracranial pressure and promote the circulation of CSF, thereby reducing the incidence of chronic hydrocephalus. So far, in clinical practice, serial lumbar puncture has sometimes been used to prevent hydrocephalus, but its effects have not been sufficiently proven. According to our years of clinical observation, this does not appear to be the case. Therefore, the aims of this study were to (1) define the predictors of SDHC after aSAH and (2) identify the effect of postoperative LP on SDHC.

Methods

Study Population

The study was approved by Ethics Committee of Union Hospital of Fujian Medical University and performed according to the Declaration of Helsinki guidelines, and the need for informed consent was waived. The retrospective analysis was performed using the clinical and radiological data of patients underwent surgical clipping or endovascular treatment for ruptured cerebral aneurysms in the Department of Neurosurgery, Fujian Medical University Union Hospital, from October 2009 to May 2019. The inclusion criteria were as follows: (1) SAH from an aneurysm, confirmed by preoperative computed tomography (CT), computed tomography angiography (CTA) or digital subtraction angiography (DSA); (2) history of repair by clipping or coiling the ruptured cerebral aneurysms within 72 hours; (3) survival beyond the initial hospitalization and adequate follow up. Finally, a total of 414 patients were enrolled in this study (Fig. 1). Approval from the institutional review board of Fujian Medical University Union Hospital was obtained before the medical records were accessed. And our work had been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki).

Management of Aneurysmal Subarachnoid Hemorrhage

Patients were managed according to the most recent Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage, published by the American Heart Association[12]. An emphasis was placed on prompt aneurysm treatment, treatment of acute hydrocephalus, maintenance of euvolemia, and anti-vasospasm treatment. Treatment choice, surgical clipping or endovascular coiling, was determined according to multidisciplinary discussions involving both surgical and endovascular teams. Radiological studies, such as CT and CTA/DSA, were routinely performed within 24 hours, 3 days, 1 week, and 1 month

after treatment to confirm successful obliteration of the aneurysm and to assess for any complications, such as vasospasm, cerebral infarction, and hydrocephalus.

Based on the clinical decisions of neurosurgeons and the willingness of patients or their families, regular LP was performed within 48–72 h following the operation. A total of 84 patients underwent regular LP therapy and 330 patients who did not undergo a postoperative LP were used as the control group. LP was performed once a day, and the CSF of approximately 30–40 ml was slowly drained each time. Generally, an LP was performed 6 to 8 times (average 7.15) until the biochemical protein content in the CSF was less than 0.45 g/L.

Management of Hydrocephalus

The diagnosis of hydrocephalus are based on clinical symptoms and radiological evidence. Clinical symptoms include decreased consciousness, chronic headache, walking difficulty, pupil changes, and incontinence. Radiological evidence includes enlarged ventricles, periventricular edema, rounding of the frontal horns, and obliteration of the cerebral sulci. The acute hydrocephalus is defined as hydrocephalus that occurs within 7 days of admission and it can be self-limiting in some patients, whereas others will require external ventricular drainage to alleviate hydrocephalus symptoms. EVD was removed within 14 days to prevent secondary CNSI. Shunt-dependent hydrocephalus was defined as persistent, symptomatic hydrocephalus presenting after aSAH that required permanent CSF diversion surgery. Patients who did not tolerate EVD weaning, demonstrated progressive ventriculomegaly on CT, or had persistently elevated opening pressures on LP underwent CSF diversion surgery, typically a ventriculoperitoneal shunt.

Data Collection

Through a review of electronic medical records, the variables collected in the study cohort comprised patient demographic information (age, sex, hypertension, diabetes), habits (smoking and alcohol intake), clinical and imaging features, which included a GCS score on admission, World Federation of Neurological Surgeons (WFNS) grade on admission, H&H on admission, presence of IVH, presence of intracerebral hemorrhage (ICH), occurrence of early cerebral infarction, occurrence of angiographic cerebral vasospasm, Fisher's grade and Barrow Neurological Institute (BNI) score for evaluating the volume of subarachnoid hemorrhage, aneurysm characteristics such as aneurysm size (maximum diameter) and location (anterior circulation (internal carotid artery, anterior cerebral artery, anterior communicating artery, middle cerebral artery) vs posterior circulation (posterior communicating artery, posterior cerebral artery, basilar artery)), presence of acute hydrocephalus, presence of CNSI, a history of EVD placement and a history of postoperative LP.

Statistical Analysis

The data were presented as the mean \pm standard deviation or median (interquartile range (IQR)) for continuous variables and partial categorical variables (include GCS score, Fisher grade, H&H, WFNS grade and BNI score) which were compared using Student's t-test and Mann-Whitney U test. And the data were

presented as frequency and percentage for other categorical variables which were compared using Pearson's Chi-square Test and Fisher's exact test. Receiver operating characteristic (ROC) curve analysis was used to determine the optimal cutoff values for age, GCS score, Fisher grade, H&H, WFNS grade and BNI score to predict SDHC. Variables with $p < 0.1$ in the univariate analysis were entered into a multivariable logistic regression analysis to determine independent predictors of SDHC. ROC analysis was performed to measure the predictive power of the final model. Because the patients in this study were not randomized to the LP group, in order to ensure the balance of other confounding factors between the LP and no LP groups, propensity score matching was performed to compare the difference in the incidence of SDHC between the two groups. All analyses were conducted with IBM SPSS (version 23, IBM Corp., Armonk, NY, USA). A p -value < 0.05 was considered statistically significant.

Results

414 patients with aSAH who met the inclusion criteria underwent open surgical or endovascular treatment of a ruptured aneurysm at the Fujian Medical University Union Hospital. Overall, SDHC developed in 59 patients (14.3%) in our cohort. The majority of patients were female (60.4%) with a mean age of 55.96 ± 9.75 years; 43.0% ($n = 178$) of the patients had hypertension. Furthermore, 61.6% ($n = 255$) of the patient cohort had anterior circulation aneurysms. A total of 84 patients (20.3%) received an LP after the operation.

Table 1 summarizes the comparison of characteristics between aSAH patients with and without SDHC. On univariate analysis, patients with SDHC were older (mean age 58.55 vs 55.46 years, $P = 0.002$); had lower GCS scores (mean 11.03 vs 13.45, $P < 0.001$), higher WFNS grades (mean 2.72 vs 1.95, $P < 0.001$), higher H&H (mean 2.94 vs 2.22, $P < 0.001$); higher Fisher grades (mean 3.86 vs 3.29, $P < 0.001$), and higher BNI score (mean 3.03 vs 2.68, $P = 0.009$); were more likely to have acute hydrocephalus (42.4% vs 22.5%, $P < 0.001$); had a greater proportion with surgical clipping (80.0% vs 62.2%, $P = 0.011$); were more likely to have an angiographic vasospasm (25.4% vs 14.1%, $P = 0.008$); were more likely to have CNSI (10.2% vs 2.3%, $P < 0.001$); were more likely to have an IVH (50.8% vs 33.5%, $P < 0.001$) and ICH (18.6% vs 14.9%, $P = 0.038$); were more likely to receive EVD placement (54.2% vs 20.0%, $P < 0.001$) and had a greater proportion with a postoperative LP (35.6% vs 17.7%, $P = 0.005$).

To reduce the potential confusion surrounding the differences in baseline characteristics between the two groups, we carried out a multivariate logistic regression analysis of the variables with $p < 0.1$ in univariate analysis. Table 2 details the logistic regression analyses for predictors of SDHC after aSAH. In the multivariable analysis, age > 55 years ($P = 0.004$), the presence of IVH ($P = 0.038$), a Fisher grade ≥ 3 ($P = 0.043$), a H&H ≥ 3 ($P = 0.027$), a history of acute hydrocephalus ($P = 0.038$), a history of CNSI ($P = 0.021$), a history of EVD placement ($P = 0.044$) and postoperative LP ($P = 0.016$) were independent predictors of SDHC. ROC analysis revealed an area under the curve (AUC) of 0.842 for the final regression model (Fig. 2).

Of course, the more frequent incidence of hydrocephalus in the puncture group could be due to selection bias, as this group was more burdened with other risk factors. To account for patient nonrandomization in their choice of postoperative LP treatment, we performed propensity score matching with the aim of minimizing the impact of selection bias on the results of the study. Before the propensity score was matched, the analysis of the baseline characteristics of the patients showed that the patients who received an LP after the operation were younger (mean age 53.19 vs 56.41 years, $P = 0.037$), comprised fewer women (50.0% vs 63.0%, $P = 0.031$), had a higher GCS score (mean 13.30 vs 11.21, $P = 0.061$), had a higher Hunt-Hess grade (mean 2.46 vs 2.24, $P = 0.036$), had a IVH (50.0% vs 34.5%, $P = 0.007$), had a less proportion with acute hydrocephalus (15.4% vs 27.9%, $P = 0.018$) and EVD placement (15.5% vs 27.3%, $P = 0.001$). It is worth noting that all patients with postoperative CNSI received LP treatment. The standard deviation of the propensity score was 0.21, and the match tolerance was 0.02. After 1:1 tendency score matching, the number of patients in the LP group was equal to that in the No LP group ($n = 58/\text{group}$), and the matching factors were balanced and comparable between the two groups. The propensity score matched cohorts showed that the incidence of SDHC in patients who received an LP after the operation was significantly higher than that in patients who did not receive an LP (20.0% vs 12.1%, $P = 0.019$) (Table 3).

Discussion

We have identified several independent predictors for shunt-dependent hydrocephalus in a consecutive cohort of patients with SAH. Patients with age > 55 years, presence of intraventricular hemorrhage, a Fisher grade ≥ 3 , a H&H ≥ 3 , a history of acute hydrocephalus, a history of CNSI, a history of EVD placement and a history of postoperative LP had higher odds of developing shunt-dependent hydrocephalus. Many of these risk factors have been found in prior studies[5, 6, 13, 14]. Our 14.3% rate of shunt dependence is consistent with past reports[8, 14]. Our study showed that elderly patients had a higher incidence of shunt dependency than younger patients. The susceptibility of elderly patients to SDHC may be related to the mechanism; with age, the enlargement of the ventricle and subarachnoid space caused by brain atrophy allows the subarachnoid space to accommodate more blood, thus promoting meningeal inflammation and fibrosis. CSF absorption decreased and CSF circulation in the ventricular system slowed[15]. However, these factors, including angiographic vasospasm, ICH, GCS score, WFNS grade, BNI score, surgical clipping and posterior circulation aneurysm, were found to be significant in the univariate analysis, but not in the multivariate analysis. Several reports also documented the other risk factors for SDHC, including hypertension, smoking and alcohol abuse[12, 16]. In our study, these factors were more frequently present in the SDHC group, but the differences were not significant. Fisher grade and BNI score reflect the volume of hemorrhage in the ventricle and subarachnoid space. It was reported that a higher Fisher grade is positively correlated with SDHC[8]. Likewise, our study found this correlation. Treatment choice, surgical clipping or endovascular coiling, was determined according to multidisciplinary discussions involving both surgical and endovascular teams. Surgical clipping was the preferred modality of treatment in our study. Two-thirds of aSAH patients were treated using surgical clipping. Furthermore, our study findings can be consistent with those

from previous studies that have shown no significant differences in the incidences of SDHC between surgical clipping and endovascular treatment[6, 8]. However, A large registration study involving 1448 patients, which showed that coil embolization, when compared with clipping, is associated with significantly lower risks for development[7].

Acute hydrocephalus is a cause of early brain injury following subarachnoid hemorrhage and is often considered to be a form of noncommunicating or obstructive hydrocephalus due to the presence of intracisternal or intraventricular blood products limiting the outflow of CSF from the cranial vault[17]. The pathophysiological mechanism of chronic hydrocephalus after aSAH is not very clear. However, a generally accepted hypothesis suggests that it results from the impairment of CSF reabsorption and circulation caused by arachnoid granule adhesions and ventricular system obstruction[17–19]. There is growing evidence showing that SDHC after aSAH is a multifactorial process that involves the up-regulation of pro-inflammatory cytokines and fibroblasts, increased production of collagen, toxic effects of iron, and an increased secretion of serotonin, catecholamine, angiotensin and other hormones[20–23]. Kanat et al. suggest that both arachnoid granule inflammation and fibrosis lead to CSF reabsorption disorder rather than an increased CSF in secretion, which results in a chronic communicating hydrocephalus[24]. Tan Q, et al. believe that the dysfunction of CSF reabsorption caused by fibrosis and arachnoid granules adhesions is caused by a local inflammatory reaction after the subarachnoid hemorrhage[25]. CNSI and Requirement for EVD were independent risk factors for shunt dependency in our series, and this association has also been found in other series[10, 22]. CNSI is a risk factor that could, in principle, be prevented with comprehensive management and therefore should be the focus of prevention efforts to minimize the patient's risk for shunting. CSF diversion is generally considered as the main management approach for symptomatic acute hydrocephalus and patients with altered level of consciousness after aSAH. Drainage is required to aid CSF flow dynamics, thought to be affected by blockage caused by blood product obstructing arachnoid granulations and the ventricular and cisternal drainage pathways[22].

With that in consideration, we performed regular LPs to release hemorrhagic CSF in some patients after surgery, avoiding a large number of blood cells in the ventricular circulatory system metabolic decomposition, and then promote early recovery of the physiological state of CSF. But in fact our research shows that the incidence of SDHC in patients who underwent an LP was significantly higher than that in patients who did not undergo an LP. The possible mechanism are as follows: (1) long-term and regular LPs make the body dependent on it, and the absorption and compensation function of CSF decreases gradually; (2) excessive release of CSF reduces the pressure gradient on both sides of the arachnoid granules and slows down CSF absorption; (3) excessive CSF drainage causes the subarachnoid to collapse, which leads to the poor drainage of inflammatory factors, adhesion of subarachnoid space, and poor CSF circulation; (4) excessive drainage of CSF reflexively leads to the increased secretion of it; and (5) iatrogenic injury caused by multiple LPs leads to local coagulation, inflammation, and thickening of the dura mater or arachnoid membrane. In conclusion, the occurrence of SDHC after aSAH may be closely related to the CSF drainage volume and time during an LP; however, this finding requires further study.

Our study has several limitations: (1) all cases were from a single center, indicating that our findings were influenced by hospital and physician bias during the patient selection and treatment process; (2) selection bias exists, as patients were not randomly assigned to the LP or No LP group in our retrospective cohort study. Although propensity score matching of baseline characteristics minimized differences between LP and No LP cohorts, other unmeasured, latent variables and their interactions may have contributed to selection bias; and (3) to evaluate LP more precisely as a risk factor for the development of chronic hydrocephalus after SAH, additional analysis for the volume and time of LP is needed.

Conclusion

Postoperative LP was found to be a predictor of SDHC after aSAH even after adjustment for other factors in propensity score matching analysis. Our report reinforces the need for further study of the relationship between CSF drainage volume and time in LP and SDHC. Shunt dependency after aSAH is associated with higher morbidity and mortality. It is significant to identify and understand the factors that increase risk for shunting and to eliminate or mitigate the reversible factors that put aSAH patients at risk. And this may assist doctors in optimizing therapy and improving the outcomes of patients.

Abbreviations And Acronyms

aSAH: Aneurysmal subarachnoid hemorrhage; **AUC:** Area under the curve; **BNI:** Barrow Neurological Institute; **CNSI:** central nervous system infection; **CSF:** Cerebrospinal fluid; **CT:** Computed tomography; **CTA:** Computed tomography angiography; **DSA:** Digital subtraction angiography; **EVD:** external ventricular drain; **GCS:** Glasgow Coma Scale; **H&H:** Hunt and Hess grade; **ICH:** Intracerebral hemorrhage; **IVH:** Intraventricular hemorrhage; **LP:** Lumbar puncture; **ROC:** Receiver operating characteristic; **SAH:** Spontaneous subarachnoid hemorrhage; **SDHC:** Shunt-dependent hydrocephalus; **WFNS:** World Federation of Neurological Surgeons.

Declarations

Acknowledgements

We also want to express our gratitude to the patients and their families for participating in this study.

Authors' contributions

KS, ZY, SS and JC designed the present study, KS, ZY drafted and revised the initial manuscript for important intellectual content, and acquired and analyzed the data.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Ethics approval and consent to participate

The study was approved by Ethics Committee of Union Hospital of Fujian Medical University and performed according to the Declaration of Helsinki guidelines, and the need for informed consent was waived.

Consent for publication

Written informed consent for publication was obtained from all participants.

Competing interests

The authors declare no conflicts of interest.

References

1. Johnston SC, Mendis S, Mathers CD. Global variation in stroke burden and mortality: estimates from monitoring, surveillance, and modelling. *Lancet Neurol.* 2009;8:345–54.
2. Feigin VL, Lawes CMM, Bennett DA, Barker-Collo SL, Parag V. Worldwide stroke incidence and early case fatality reported in 56 population-based studies: a systematic review. *Lancet Neurol.* 2009;8:355–69.
3. de Rooij NK, Linn FHH, van der Plas JA, Algra A, Rinkel GJE. Incidence of subarachnoid haemorrhage: a systematic review with emphasis on region, age, gender and time trends. *J Neurol Neurosurg Psychiatry.* 2007;78:1365–72.
4. von Vogelsang A-C, Wengström Y, Svensson M, Forsberg C. Descriptive epidemiology in relation to gender differences and treatment modalities 10 years after intracranial aneurysm rupture in the Stockholm cohort 1996–1999. *World Neurosurg.* 2013;80:328–34.
5. Rincon F, Gordon E, Starke RM, Buitrago MM, Fernandez A, Schmidt JM, et al. Predictors of long-term shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage. Clinical article. *J Neurosurg.* 2010;113:774–80.
6. de Oliveira JG, Beck J, Setzer M, Gerlach R, Vatter H, Seifert V, et al. Risk of shunt-dependent hydrocephalus after occlusion of ruptured intracranial aneurysms by surgical clipping or

- endovascular coiling: a single-institution series and meta-analysis. *Neurosurgery*. 2007;61:924–33; discussion 933–934.
7. Yamada S, Ishikawa M, Yamamoto K, Ino T, Kimura T, Kobayashi S, et al. Aneurysm location and clipping versus coiling for development of secondary normal-pressure hydrocephalus after aneurysmal subarachnoid hemorrhage: Japanese Stroke DataBank. *J Neurosurg*. 2015;123:1555–61.
 8. Zaidi HA, Montoure A, Elhadi A, Nakaji P, McDougall CG, Albuquerque FC, et al. Long-term functional outcomes and predictors of shunt-dependent hydrocephalus after treatment of ruptured intracranial aneurysms in the BRAT trial: revisiting the clip vs coil debate. *Neurosurgery*. 2015;76:608–13; discussion 613–614; quiz 614.
 9. Reddy GK, Bollam P, Shi R, Guthikonda B, Nanda A. Management of adult hydrocephalus with ventriculoperitoneal shunts: long-term single-institution experience. *Neurosurgery*. 2011;69:774–80; discussion 780–781.
 10. Lee L, King NKK, Kumar D, Ng YP, Rao J, Ng H, et al. Use of programmable versus nonprogrammable shunts in the management of hydrocephalus secondary to aneurysmal subarachnoid hemorrhage: a retrospective study with cost-benefit analysis. *J Neurosurg*. 2014;121:899–903.
 11. Reddy GK, Bollam P, Caldito G. Ventriculoperitoneal shunt surgery and the risk of shunt infection in patients with hydrocephalus: long-term single institution experience. *World Neurosurg*. 2012;78:155–63.
 12. Connolly ES, Rabinstein AA, Carhuapoma JR, Derdeyn CP, Dion J, Higashida RT, et al. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2012;43:1711–37.
 13. Xie Z, Hu X, Zan X, Lin S, Li H, You C. Predictors of Shunt-dependent Hydrocephalus After Aneurysmal Subarachnoid Hemorrhage? A Systematic Review and Meta-Analysis. *World Neurosurg*. 2017;106:844–860.e6.
 14. Di Russo P, Di Carlo DT, Lutenberg A, Morganti R, Evins AI, Perrini P. Shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage. *J Neurosurg Sci*. 2020;64:181–9.
 15. Jabbarli R, Bohrer A-M, Pierscianek D, Müller D, Wrede KH, Dammann P, et al. The CHES score: a simple tool for early prediction of shunt dependency after aneurysmal subarachnoid hemorrhage. *Eur J Neurol*. 2016;23:912–8.
 16. Beni-Adani L, Biani N, Ben-Sirah L, Constantini S. The occurrence of obstructive vs absorptive hydrocephalus in newborns and infants: relevance to treatment choices. *Childs Nerv Syst ChNS Off J Int Soc Pediatr Neurosurg*. 2006;22:1543–63.
 17. Strahle J, Garton HJL, Maher CO, Muraszko KM, Keep RF, Xi G. Mechanisms of hydrocephalus after neonatal and adult intraventricular hemorrhage. *Transl Stroke Res*. 2012;3 Suppl 1:25–38.
 18. Massicotte EM, Del Bigio MR. Human arachnoid villi response to subarachnoid hemorrhage: possible relationship to chronic hydrocephalus. *J Neurosurg*. 1999;91:80–4.

19. Dorai Z, Hynan LS, Kopitnik TA, Samson D. Factors related to hydrocephalus after aneurysmal subarachnoid hemorrhage. *Neurosurgery*. 2003;52:763–9; discussion 769–771.
20. Okubo S, Strahle J, Keep RF, Hua Y, Xi G. Subarachnoid hemorrhage-induced hydrocephalus in rats. *Stroke*. 2013;44:547–50.
21. Nakatsuka Y, Kawakita F, Yasuda R, Umeda Y, Toma N, Sakaida H, et al. Preventive effects of cilostazol against the development of shunt-dependent hydrocephalus after subarachnoid hemorrhage. *J Neurosurg*. 2017;127:319–26.
22. Jartti P, Karttunen A, Jartti A, Ukkola V, Sajanti J, Pyhtinen J. Factors related to acute hydrocephalus after subarachnoid hemorrhage. *Acta Radiol Stockh Swed* 1987. 2004;45:333–9.
23. Lenski M, Biczok A, Hüge V, Forbrig R, Briegel J, Tonn J-C, et al. Role of Cerebrospinal Fluid Markers for Predicting Shunt-Dependent Hydrocephalus in Patients with Subarachnoid Hemorrhage and External Ventricular Drain Placement. *World Neurosurg*. 2019;121:e535–42.
24. Kanat A, Turkmenoglu O, Aydin MD, Yolas C, Aydin N, Gursan N, et al. Toward changing of the pathophysiologic basis of acute hydrocephalus after subarachnoid hemorrhage: a preliminary experimental study. *World Neurosurg*. 2013;80:390–5.
25. Tan Q, Chen Q, Feng Z, Shi X, Tang J, Tao Y, et al. Cannabinoid receptor 2 activation restricts fibrosis and alleviates hydrocephalus after intraventricular hemorrhage. *Brain Res*. 2017;1654 Pt A:24–33.

Figures

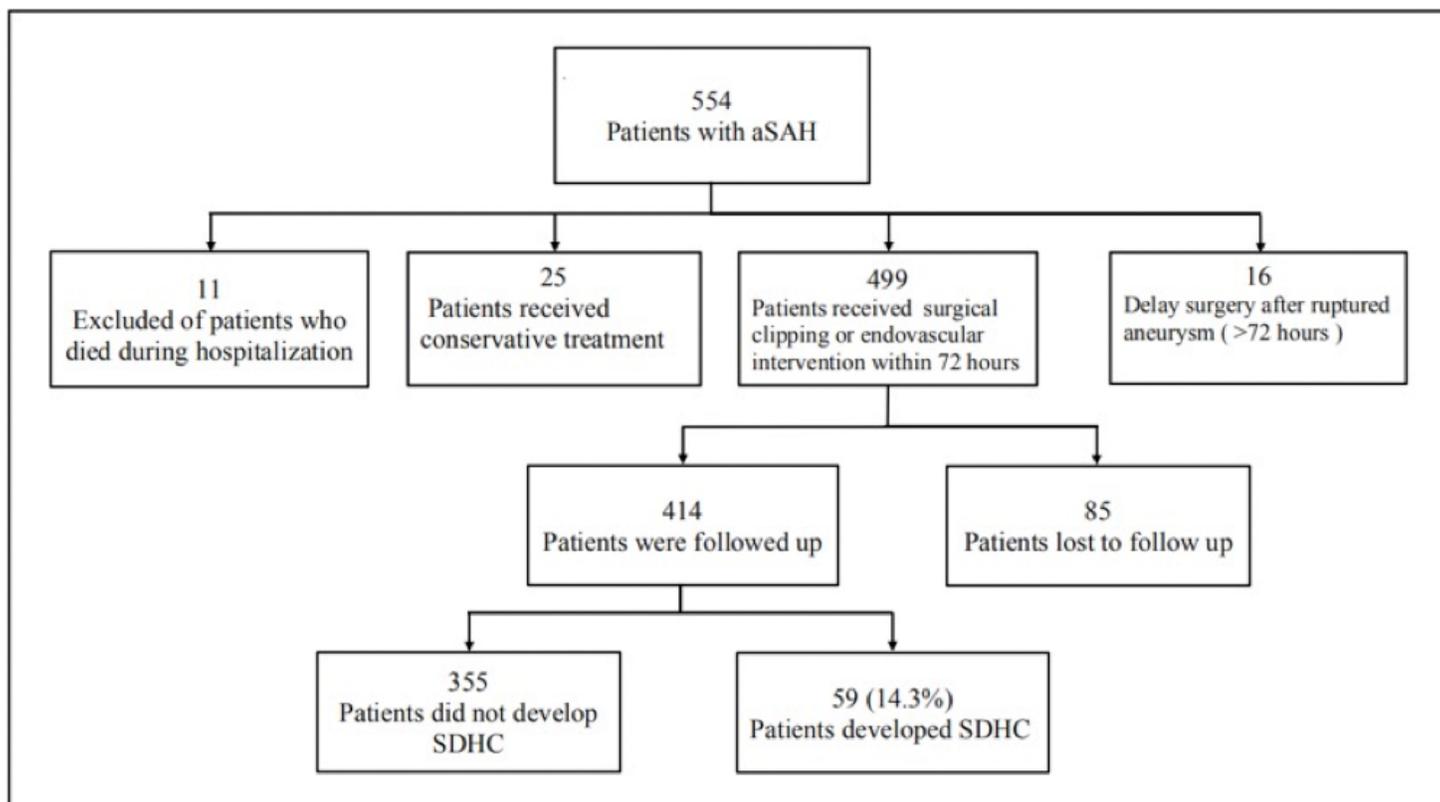


Figure 1

A flow chart illustrating the management protocol for patients with aSAH in Fujian Medical University Union Hospital, from October 2009 to May 2019.

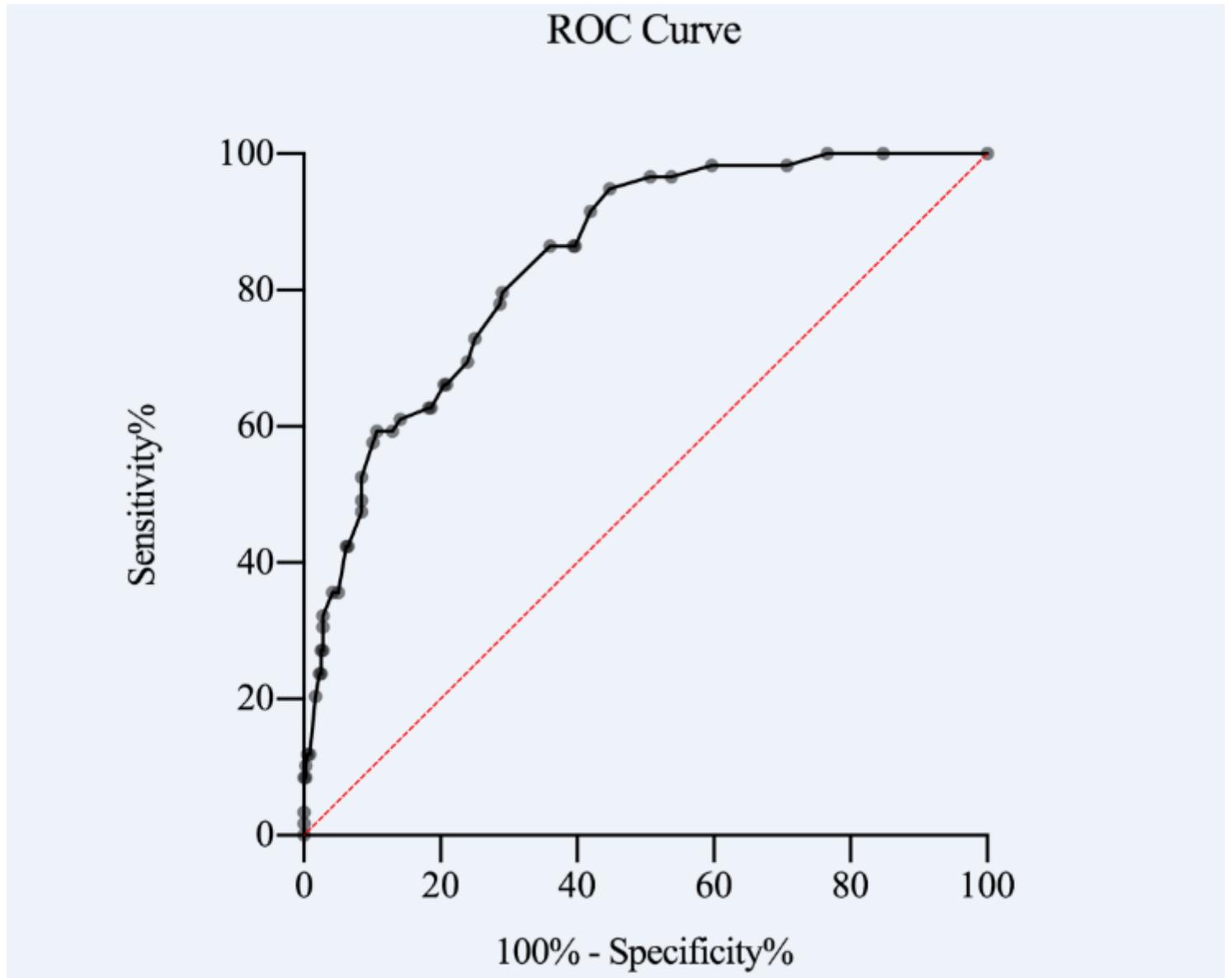


Figure 2

ROC curve for final regression model predicting shunt dependence (AUC =0.842). AUC, Area under the curve; ROC, Receiver operating characteristic.