ORIGINAL ARTICLE

# Predictor's analysis of anterior circulation cerebral infarction after the endovascular treatment of anterior communicating artery aneurysms

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**Background:** Despite increasing acceptance of endovascular coiling for treating anterior communicating artery (ACoA) aneurysms, anterior circulation cerebral infarction (ACI) after embolization remains a limitation. With higher incidence, higher morbidity and higher mortality, it is one of the main factors influencing the ACoA aneurysms prognosis. Determining the risk factors leading to ACI after embolization will have clinical significance. Through retrospective case analysis, this study investigated the risk factors related to ACI after embolization in order to provide information to serve the clinical practice. **Materials and Methods:** A retrospective review was performed of patients who had undergone coiling of ACoA aneurysms from 2008 to 2012. All patients had ruptured prior to the completion of embolization. Cases with acute stroke symptoms without alternative diagnoses after embolization were diagnosed as ACI. A total of 32 risk factors such as age, sex, hypertension, diabetes mellitus, modified Fisher grade, Hunt-Hess grade, ventricular hemorrhage, etc. were analyzed using univariate and logistic regression analysis. **Results:** Univariate analysis showed that negative fluid volume balance ($P$ = 0.041 <0.05) and modified Fisher grade ($P$ = 0.049 <0.05) reached statistical significance, suggesting that they might be risk factors for ACI after embolization. Multiple logistic regression analysis showed that modified Fisher grade was significantly associated with ACI after embolization, suggesting that it was an independent risk factor (odds ratios (OR): 4.968, 95% confidence intervals (CI): 1.013-24.360, $P$ = 0.048). **Conclusion:** Modified Fisher grade is an independent risk factor for ACI after embolization.

**Key words:** Anterior circulation cerebral infarction, anterior communicating artery aneurysm, embolization, modified Fisher grade, risk factors

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## INTRODUCTION

The most frequently located site of ruptured intracranial aneurysms is the anterior cerebral artery (ACA),[1] and the anterior communicating artery (AcoA) is the most frequently found site for ruptured ACA distribution aneurysms.[2] The treatment of intracranial aneurysms has been revolutionized by the introduction of detachable coils for endovascular therapy,[3-6] and it has been demonstrated that it is a safe and effective method for treating aneurysms.[7] It is not unusual, however, to encounter either transient or permanent ischemic events after embolization procedure of ACoA aneurysms. Its prevalence of thromboembolic complications, including transient ischemic and stroke, has been reported to be 10.3%.[8,9] At present, reliable early warning methods are lacking, so anterior circulation cerebral infarction (ACI) is often diagnosed after clinical deterioration of neurological function, resulting in passive treatment. The risk factors relevant to ACI after embolization are poorly understood, making preventative treatment difficult. The current study is the first to elucidate the risk factors which lead to ACI after endovascular coiling of AcoA aneurysms.

During the past 2 decades, the emphasis on ischemic stroke pathogenesis has largely switched from hypoperfusion and hemodynamic-related explanations to thromboembolism as the predominant mechanism.[10] Nevertheless, the close relationship between severity of extracranial arterial stenosis and brain infarction,[11] the correlation between impaired functional blood flow reserve and subsequent brain infarction,[12] and the correlation of reduced collateral blood flow with a poor prognosis[13] indicate that ischemic stroke pathogenesis is a multifactorial process. This has led to a reappraisal of the risk factors of ACI after embolization.

The purpose of the present study was to investigate the inpatient database in ACoA aneurysms using

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endovascular treatment. In particular, we wished to elucidate the risk factors relevant to ACI after embolization using univariate and logistic regression analysis.

## MATERIALS AND METHODS

### Patient selection

Case files from patients that had undergone coil embolization of cerebral anterior communication artery aneurysms from October 2008 to April 2012 at Tangshan Gongren Hospital were retrospectively reviewed. Patients with anterior communication artery occlusion during embolization were excluded from the study. The patency of 54 patients' ACA and ACoA during embolization was evaluated in the study. These patients were all ruptured. The stroke rate was 8.7% in ruptured patients for one time before embolization finished and 37.5% in ruptured patients for twice before embolization finished. The mean age of the study group was 53.6 years with a 1:1 female-to-male ratio. Hospital records including clinical charts, operative reports, and radiological studies were used to obtain patient characteristics including: Age, coil type, rupture status, sex, aneurysm location, aneurysm size, embolization results, complication, and necessity of retreatment. The timing of the infarction following treatment occurred at 3 postoperative hours to 3 days. This study was conducted in accordance with the Declaration of Helsinki. This study was conducted with approval from the Ethics Committee and Intuitional Review Board of Hebei Medical University. Written informed consent was obtained from all participants.

### Endovascular treatment

The technique for endovascular coiling alone and stent-assisted coiling (Boston Scientific/Target, California, America) has been previously described in the literature.[14-16] All procedures were performed using a biplane angiographic system (Philippe, Amsterdam, Holland) with three-dimensional rotational and digital subtraction capabilities and with the patients under general anesthesia and a systemic anticoagulant. Anticoagulation was aimed at keeping the activated clotting time at two to three times above the normal value (approximately 100 s) during catheterization, stent, and coil placement. Our protocol includes: 1) Pretreatment with aspirin (300 mg) and clopidogrel bisulfate (300 mg) 30 min before coiling for stented patients and no pretreatment before coiling for non-stented patients, 2) systemic intravenous heparinization after placement of the femoral sheath for a goal activated clotting time of 250-300 s, 3) systemic intravenous heparinization 12 h after the procedure, 4) continuation of clopidogrel bisulfate (75 mg daily) for 6 months for stented patients and discontinuation of clopidogrel bisulfate for non-stented patients, and 5) aspirin (100 mg daily) for all patients indefinitely.

### Risk factors for anterior circulation infarction after embolization

The risk factors considered in the study were sex, age, smoking, alcohol consumption, hypertension, diabetes mellitus, hyperfibrinogenemia, hyperlipidemia, ischemic changes on electrocardiogram (ECG), fever, fluid volume balance, modified Fisher grade,[17] Hunt-Hess grade,[18] intracerebral hematoma, ventricular hemorrhage, cerebral swelling, hydrocephalus, unilateral dysplasia of cerebral anterior circulation, direction of aneurysm body, aneurysm blood supply from left ACA, aneurysm rupture times, time from aneurysm recent rupture before admission to embolization, stent use, packing degree,[19] leukocyte count, platelet count, hematocrit, comorbidity score,[20] aneurysm neck width, aneurysm parent artery width, aneurysm size, and embolization time. We defined the potential risk factors as follows:

1. Smoking (currently smoking more than one cigarette per day)
2. Alcohol consumption (drinking more than twice per week)
3. Hypertension (systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg, and/or presence of antihypertensive drug treatment)
4. Diabetes mellitus was defined as a high-fasting plasma glucose level (≥126 mg/dl), or a current treatment with an oral hypoglycemic agent or insulin
5. Hyperfibrinogenemia was defined as a high plasma fibrinogen level (≥400 mg/dl)
6. Hyperlipidemia was defined as a fasting serum total cholesterol level of ≥240 mg/dl, or a current treatment with an antihyperlipidemic agent
7. Ischemic changes on electrocardiogram (ECG; ST-T change, flat T, and negative T)
8. Fever (normal body temperature range 36.0-37.4°C, low grade fever range 37.5-38.0°C, and high grade fever ≥38.1°C)
9. Fluid volume balance was defined as intake and output balance from admission to the end of operation
10. Packing degree was classified as defined by Raymond *et al*.[19] Class 1 — complete occlusion, class 2 — residual neck, and class 3 — residual aneurysm sac.

### Computed tomography (CT) scans

For all 54 patients, cerebral angiography showed patency of the cerebral artery during the process of treatment. Neurological deficit was present in seven patients during recovery from general anesthesia after embolization. Clinical symptoms included motor dysfunction, abulia, aphasia, urinary incontinence, etc. Non-contrast brain CT (Siemens, Munich, Germany) was performed immediately after the procedure. ACI on CT was indicated for all seven patients. For the 47 patients demonstrating no neurological deficit, repeat CTs were requested at days 1 and 7 or sooner

at the physician's discretion and no cerebral infarction occurred.

### Statistical analysis

With seven patients in the ACI group and 47 patients in the no ACI group, the study was designed to detect the risk factors of ACI after embolization and a two-sided a level of 0.05.

Descriptive statistics, stratified by cerebral anterior circulation state, were calculated for baseline demographic and clinical characteristics. Student's *t*-test was used to compare continuous variables, the $\chi^2$ test to compare categorical variables, and the rank sum test to compare multicategorical variables according to cerebral infarction or no cerebral infarction of anterior circulation after embolization. Multiple logistic regression analysis was performed to identify independent risk factors for ACI in the two groups. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated after adjusting for possible confounders (aneurysm size, modified Fisher grade, and embolization time). All *P*-values are two-sided with statistical significance evaluated at the 0.05 a level. Ninety-five percent CIs were calculated to assess the precision of the obtained adjusted odds ratio estimates. All analyses were performed using Statistical Package for Social Sciences (SPSS) 17.0 (SPSS, Chicago, IL).

## RESULTS

Table 1 summarizes the clinical and laboratory data of the study patients. After exclusion criteria were met, 54 ACoA aneurysms from 54 patients were analyzed. Of these, seven (13.0%) were categorized into Group I (ACI after embolization) and 47 (87.0%) were categorized into Group II (no ACI after embolization). The neurological outcomes of the ischemic stroke patients included brain swelling, motor dysfunction, abulia, aphasia, etc. Fifty-four ACoA aneurysms (100%) were ruptured in patients presenting with subarachnoid hemorrhage.

Only two of the analyzed variables were significantly different between groups. Univariate analyses of clinical and laboratory data showed that negative fluid volume balance and modified Fisher grade were more prevalent in the ACI group than in the no ACI group [Table 1]. Other clinical and laboratory data, including age, sex, hypertension, diabetes mellitus, Hunt-Hess grade, and ventricular hemorrhage showed no differences between groups.

**Table 1: Univariate analysis of the clinical and laboratory data of the anterior circulation cerebral infarction and no anterior circulation cerebral infarction groups**

| | Infarction (*n* = 7)% | No infarction (*n* = 47)% | *P*-value |
|---|---|---|---|
| Sex (women, %) | 4 (57.1) | 24 (51.1) | 1.000 |
| Smoking (%) | 28.6 | 25.5 | 1.000 |
| Alcohol consumption (%) | 14.3 | 23.4 | 0.957 |
| Hypertension (%) | 57.1 | 38.3 | 1.000 |
| Diabetes mellitus (%) | 57.1 | 25.5 | 0.177 |
| Hyperfibrinogenemia (%) | 71.4 | 29.8 | 0.083 |
| Hyperlipidemia (%) | 28.6 | 19.1 | 0.941 |
| Ischemic changes on ECG (%) | 14.3 | 34.0 | 0.539 |
| Fever (%) | | | 0.316 |
| 36.0-37.4°C | 71.4 | 87.2 | |
| 37.5-38.0°C | 28.6 | 8.5 | |
| >38.1°C | 0 | 4.3 | |
| Negative fluid volume balance (%) | 85.7 | 40.4 | 0.041 |
| Modified Fisher grade (%) | | | 0.049 |
| 0-I | 14.3 | 36.2 | |
| II | 0 | 17.0 | |
| III | 14.3 | 17.0 | |
| IV | 71.4 | 29.8 | |
| Hunt-Hess grade (%) | | | 0.520 |
| I | 71.4 | 80.9 | |
| II | 0 | 4.2 | |
| III | 28.6 | 14.9 | |
| IV | 0 | 0 | |
| V | 0 | 0 | |
| Intracerebral hematoma (%) | 14.3 | 6.4 | 0.436 |
| Ventricular hemorrhage (%) | 71.4 | 46.8 | 0.420 |
| Cerebral swelling (%) | 14.3 | 2.1 | 0.245 |

*(Continued)*

**Table 1: Univariate analysis of the clinical and laboratory data of the anterior circulation cerebral infarction and no anterior circulation cerebral infarction groups *(Continued)***

| | Infarction (*n* = 7)% | No infarction (*n* = 47)% | *P*-value |
|---|---|---|---|
| Hydrocephalus (%) | 0 | 19.1 | 0.469 |
| Unilateral dysplasia of cerebral interior circulation (%) | 42.9 | 23.4 | 0.526 |
| Direction of aneurysm body (%) | | | 0.379 |
| Anterior surface | 57.1 | 70.2 | |
| Posterior surface | 14.3 | 14.9 | |
| Superior surface | 14.3 | 12.8 | |
| Inferior surface | 0 | 2.1 | |
| Complicated direction | 14.3 | 0 | |
| Aneurysm blood supply from left ACA (%) | 85.7 | 74.5 | 0.861 |
| Aneurysm rupture times (%) | | | 0.095 |
| One time before embolization finished | 57.1 | 89.4 | |
| Twice before embolization finished | 42.9 | 10.6 | |
| Time from aneurysm recent rupture before admission to embolization (%) | | 70.2 | 0.923 |
| 1-3 days | 71.4 | 25.5 | |
| 3 days-2 weeks | 14.3 | 4.3 | |
| >2 weeks | 14.3 | | |
| Stent use (%) | 28.6 | 8.5 | 0.169 |
| Packing degree (%) | | | 0.515 |
| Complete occlusion | 85.7 | 91.5 | |
| Residual neck | 14.3 | 8.5 | |
| Residual sac | 0 | 0 | |
| Age (years) | 53.3±8.2 | 53.9±9.6 | 0.866 |
| Leukocyte count ($10^{-9}$/l) | 12.8±3.9 | 11.8±3.6 | 0.500 |
| Platelet count ($10^{-9}$/l) | 231.1±73.2 | 226.4±50.8 | 0.828 |
| Hematocrit | 0.41±0.04 | 0.42±0.05 | 0.827 |
| Comorbidity score | 1.43±0.79 | 1.49±0.88 | 0.864 |
| Aneurysm neck width (mm) | 3.31±1.65 | 2.04±0.74 | 0.089 |
| Aneurysm parent artery width (mm) | 1.57±0.32 | 1.72±0.46 | 0.405 |
| Aneurysm size (mm) | 6.85±3.38 | 4.15±1.41 | 0.080 |
| Embolization time (h) | 2.70±1.35 | 1.51±0.56 | 0.059 |

ECG = Electrocardiogram; ACA = Anterior cerebral artery; Statistically significant, $P < 0.05$ (two-tailed)

Multiple logistic regression analyses adjusted for possible confounding factors. The adjusted OR for modified Fisher grade (OR: 4.968, 95% CI: 1.013-24.360, $P = 0.048$) was significantly higher in the ACI group [Table 2].

**Table 2: Results of multiple logistic regression analysis applied to the anterior circulation cerebral infarction and no anterior circulation cerebral infarction groups**

| | OR | 95% CI | | *P*-value |
|---|---|---|---|---|
| | | Lower | Upper | |
| Modified Fisher grade | 4.968 | 1.013 | 24.360 | 0.048 |
| Aneurysm size | 2.033 | 0.842 | 4.906 | 0.115 |
| Embolization time | 5.539 | 0.795 | 38.583 | 0.084 |

OR = Odds ratio; CI = Confidence interval; Patients in the no anterior circulation cerebral infarction group served as control; Statistically significant, $P < 0.05$ (two-tailed)

## DISCUSSION

Although the risk factors of ischemic stroke in the anterior circulation have been extensively researched, there have been fewer studies focusing on the risk factors of ACI after embolization;[21,22] however, these studies do reveal risk factors comparable to those reported in the present study. Previously, it was demonstrated that modified Fisher grade was the most influential risk factor for anterior circulation stroke.[23,24] Given these findings, we evaluated the risk factors of ACI after embolization and report the resulting effects on cerebral stroke here.

In the present study, the prevalence of ACI after embolization (13.0%) was found to be high in patients with the endovascular treatment of ACoA aneurysms and modified Fisher grade was found to be the major determinant for anterior circulation infarction after embolization. This risk factor is consistent with the previously reported result of anterior circulation stroke.[24] Seven of 55 patients undergoing coil embolization of ACoA aneurysms developed ACI after embolization, in which the patency of anterior circulation during embolization was observed. Thrombus formation during coil embolization occurred in one patient. Few studies have reported the prevalence of ACI after coil embolization of ACoA aneurysms. Thromboembolic events during endovascular treatment

of intracranial aneurysms were reported to be between 2.9 and 6%.[25] Fang *et al*., showed morbidity or mortality caused by perioperative stroke occurred at a 3% rate in patients with ACoA aneurysms.[26] Why then, was the stroke rate (13%) so high in our study? The higher stroke rate is likely because the patients undergoing coil embolization of cerebral anterior communication artery aneurysms in this study had all ruptured once or twice prior to embolization.

The possible mechanisms for ACI after embolization were previously thought to be vasospasm, thrombosis, and emboli.[27,28] No pulmonary embolism, cardiogenic embolism, deep venous thrombosis, cerebral vascular arteriosclerosis, nor cerebral vascular stenosis were found in the current study. Our study confirmed that vasospasm and thrombosis were the most common etiology for ACI after embolization. In patients with subarachnoid hemorrhage due to ACoA aneurysm, the significant risk factors determining outcomes were age, Glasgow coma scale (GCS), Hunt-Hess grade, vasospasm, ventricular hemorrhage, and hydrocephalus.[29,30] However, the associations between ACI after embolization of ACoA aneurysm and the risk factors, such as, age, sex, negative fluid volume balance, modified Fisher grade, aneurysm size, embolization time, and so on are not being determined because of the limited number of studies undertaken. In the present study, multiple logistic regression analysis revealed that modified Fisher grade is independently associated with ACI after embolization; whereas, no association is found for other variables, which include negative fluid volume balance, aneurysm size, and embolization time. The previous studies reported that a closed relationship was observed between modified Fisher grade and delayed ischemic neurological deficit.[31-34] These findings suggest that modified Fisher grade is a strong determinant for ACI after embolization, and that a patient with a high modified Fisher grade is at higher risk of ACI after embolization. ACI after embolization is a potent predictor of a poor outcome in ACoA aneurysm patients.[30,35-37] Accordingly, modified Fisher grade prior to stroke is required for prevention of ACI after embolization. As much higher grade of modified Fisher grade, we can enhance our prevention and treatment for vasospasm and thrombosis.

The present study has several limitations that should be considered. First, this is a retrospective study, and inaccurate or insufficient assessment might have occurred. Second, this is a relatively small study, so the predictor of outcome could be significant by chance. Third, because the patients were recruited from a tertiary hospital, they may not represent the general stroke population after embolization. Fourth, aneurysm characteristics and assessment of embolization were assessed using aneurysm size, neck width, and Raymond classification; but not using Murayama classification, aneurysm volume in cubic millimeters, neck-to-volume ratio, or volumetric packing density.[14,38] Fifth, CT was used in this study, which is not very sensitive in the evaluation of stroke. Thus, some of the stroke patients may not be ruled out. Finally, all our patients were Chinese, and our data may not be generalizable to other ethnic groups. As such, we await the outcome of a prospective, randomized, controlled trial when treating cerebral aneurysms.

Overall, the present study provides clues regarding the prevalence and causative risk factor of ACI after embolization. Furthermore, the data may increase clinicians' awareness of risk factors related to ACI.

## CONCLUSION

Our results showed high prevalence of ACI after embolization, and a modified Fisher grade was identified as the most important risk factor therefore. Further studies on the mechanism, prevention, and treatment of ACI after embolization are needed to provide specific guidance on its long-term management.

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