Focal Neurological Deficit at Onset of Aneurysmal Subarachnoid Hemorrhage: Frequency and Causes

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Background and Aim: Focal neurological deficit (FND) is a recognized presenting symptom of aneurysmal subarachnoid hemorrhage (SAH). However, little is known on how often aneurysmal SAH patients present with FND and what the responsible mechanisms are. The aim of this study was to examine the frequency and causes of FND at onset in aneurysmal SAH. *Methods:* We reviewed the records of consecutive aneurysmal SAH patients over 5 years and identified those who presented with FND. We developed several potential mechanisms for FND based on consensus between 2 separate evaluating neurologists. We then compared the characteristics of aneurysmal SAH patients who presented with and without FND. Logistic regression models were used to assess for association of FND with poor outcome. *Results:* Of a total of 213 patients, 10.3% presented with FND. The junction of the internal carotid and posterior communicating arteries was the most common aneurysm location in patients with FND (36.4%). Causes of FND at presentation were intraparenchymal hematoma in 45.5%, early cerebral infarction in 22.7%, parenchymal compression by subarachnoid thrombus in 18.2%, and seizure with Todd's paralysis in 13.6%. Patients with FND were older ($P = .001$) and had higher rates of in-hospital death and severe disability at discharge ($P < .0001$), compared to those without focal deficit. FND was independently associated with poor outcome (odds ratio: 4.62, confidence interval: 1.41-15.14; $P = .01$). *Conclusion:* One in every 10 aneurysmal SAH patients presents with FND. FND at presentation has diverse mechanisms, is not associated with a specific aneurysm location, and is independently associated with poor outcome. **Key Words:** Subarachnoid hemorrhage—symptoms—focal deficit—outcome.

Introduction

Aneurysmal subarachnoid hemorrhage (SAH) is a neurological emergency with high morbidity and mortality. The principal symptom of aneurysmal SAH is abrupt onset of severe headache. Transient loss of consciousness can occur in approximately one half of cases.[1] Although recognized as a potential presenting symptom, focal neurological deficit (FND) at onset of aneurysmal SAH is relatively uncommon and has traditionally been attributed to rupture of middle cerebral artery aneurysms, with resultant intraparenchymal hematoma (IPH).[2] It has been suggested that aneurysmal SAH patients who present with FND are at a higher risk for developing permanent neurological deficits.[3] However, the impact of FND

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Received April 15, 2016; revision received June 8, 2016; accepted July 4, 2016.

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1052-3057/$ - see front matter

http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2016.07.009

at presentation on functional outcome has not been formally investigated. Moreover, the proportion of patients who present with FND at onset of aneurysmal SAH is unknown. In this study, we aimed to determine the frequency and causes of acute FND in aneurysmal SAH patients and compare their outcomes with those who presented without FND.

Methods

Patient Selection and Data Collection

We used an institutional stroke registry at a public district hospital in San Antonio, Texas, to extract data on consecutive patients aged 18 years or older admitted with the diagnosis of SAH from January 2010 to October 2015. Institutional board approval was obtained prior to data extraction. SAH was defined as sudden headache with or without coma or focal deficit, and presence of subarachnoid blood, confirmed by computed tomography (CT) scan. We excluded SAH cases due to trauma, arteriovenous malformations, vasculitis or vasculopathy, mycotic aneurysms, infectious or neoplastic lesions, and angiogram-negative SAH. Admission records of all patients were individually reviewed for FND at presentation, which was defined as hemiparesis, hemisensory loss, hemianopia, or aphasia before surgical management of the aneurysm and not due to delayed cerebral ischemia. As such, FND first documented more than 12 hours after onset of aneurysmal SAH was not included in that pool. Data collected were age, gender, Fisher grade, World Federation of Neurosurgical Societies (WFNS) classification, aneurysm size and location, seizure on presentation, hydrocephalus, modified Rankin Scale score at discharge, whether the deficit was present on discharge, and occurrence of delayed cerebral ischemia. The criteria for delayed cerebral ischemia were new focal neurologic signs and/or deterioration of the level of consciousness more than or equal to 48 hours of presentation with no evidence of worsening hemorrhage, plus radiological evidence of cerebral infarction with or without angiographic evidence of vasospasm. All aneurysmal SAH patients were managed in accordance with the guidelines of the American Stroke Association.[4]

We categorized patients based on the mechanisms for FND agreed upon according to independent evaluation by 2 neurologists. Categories included the following: (1) IPH; (2) cisternal/fissure hematoma with parenchymal compression (mass effect); (3) early cerebral infarction; (4) Todd's paralysis; and (5) undetermined. Seizures at onset were documented if an event was witnessed or described by bystanders, emergency medical personnel, emergency department staff, or the examining neurologist. Early cerebral infarction was diagnosed when radiographic evidence of ischemia corresponding to the FND was present on CT or magnetic resonance imaging on admission or within the first 24 hours of hospitalization.

Statistical Analysis

We compared the baseline characteristics of patients who presented with and without FND. Nominal variables were presented as proportions and compared using χ^2 or Fisher's exact test, wherever appropriate; ordinal variables as medians with interquartile range and compared using the Mann–Whitney *U*-test; and continuous variables as means ± standard deviation and compared using the Student's *t*-test. Rates were compared using the *z*-score test for 2 population proportions. To examine for associations, simple and multiple logistic regression analyses were performed with prespecified variables impacting survival and outcome. Poor outcome, defined as modified Rankin Scale score greater than or equal to 4, was used as the dependent variable. To avoid collinearity with FND, WFNS grade was not included in the multivariate model. Significance for all analyses was set at $P \leq .05$.

Results

Frequency and Causes of FND

Among a total of 213 patients with aneurysmal SAH, 22 (10.3%) presented with FND. The mean age for this group was 58.6 ± 13.2 years, and 45.5% were men. Deficits included hemiparesis in 91% and aphasia in 9%. Causes of FND were IPH in 45.5%, early cerebral infarction in 22.7%, cisternal/fissure hematoma with parenchymal compression in 18.2%, and seizure with Todd's paralysis in 13.6%. Aneurysms of the posterior communicating segment of the internal carotid artery comprised the majority of the ruptures (36.4%) in patients with FND, followed by the middle cerebral artery (27.2%). Specific characteristics of these patients along with respective causes for FND are summarized in Table 1.

Comparison of Characteristics

Table 2 compares the characteristics of patients with and without FND. Patients with FND at onset were on average older ($P = .001$) and presented with higher median Fisher ($P = .004$) and WFNS ($P < .0001$) grades. Furthermore, patients with FND had higher median modified Rankin Scale scores at discharge, compared with those without deficit ($P < .0001$).

Outcomes

The majority of patients (81.8%) had persistent deficit on discharge or at the time of death. The most common scenario wherein FND was reversible was seizure with Todd's paralysis.

Seven patients with FND (31.8%) died in the hospital and another 8 were discharged with severe disability (modified Rankin Scales scores, 4 and 5). The rate of poor outcome at discharge was significantly higher in patients presenting with FND (68.2% versus 18.8%; $P < .0001$).