Basilar Vasospasm Following Spontaneous and Traumatic Subarachnoid Haemorrhage: Clinical Implications

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Summary

Background. Cerebral vasospasm has been commonly described following subarachnoid haemorrhage (SAH) though its impact on neurological outcome, especially in head trauma, has not been yet elucidated. The purpose of this study was to monitor and correlate neurological condition and flow velocities (FVs) in the arteries of the brain after SAH and more particularly to investigate the influence of basilar artery (BA) vasospasm on neurological outcome.

Methods. Daily transcranial Doppler (TCD) evaluations were conducted in 116 consecutive patients with subarachnoid haemorrhage. SAH was of traumatic origin (tSAH) in 59 patients and spontaneous (sSAH) in 57 patients. Vasospasm in the MCA and ACA was defined by a mean FV exceeding 120 cm/s and three times the mean FV of the ipsilateral ICA. Basilar artery (BA) vasospasm was defined as moderate whenever the FV was higher than 60 cm/s and severe above 85 cm/s.

Findings. Sixty-two patients (53.4%) had elevated FVs in the BA, among these 34 (29.3%) had FVs above 85 cm/s. Basilar vasospasm was significantly more common in tSAH (59.7%) than in sSAH (40.3%, P = 0.041). In patients with moderate and severe BA vasospasm, FVs in the BA increased on the third day after admission and remained elevated for a week before returning to normal value by the end of the second week. This elevation in BA FVs in patients with BA vasospasm was followed by a significant and progressive worsening in the neurological condition at the end of the first week. Permanent neurological deficit was associated with elevated BA FVs consistent with moderate BA vasospasm whereas patients who remained in persistent vegetative state, had FVs consistent with severe BA vasospasm (P = 0.00019).

Interpretation. The present results further support that BA vasospasm may act as an independent factor of ischaemic brain damage following SAH, especially in head trauma.

Keywords: Basilar artery; vasospasm; subarachnoid haemorrhage; transcranial Doppler.

Introduction

Since its introduction by Aaslid et al. [1], transcranial Doppler (TCD) sonography has emerged as a reliable and sensitive method for the diagnosis and evaluation of cerebral arterial vasospasm following subarachnoid haemorrhage (SAH). Since then, numerous studies have emphasized the magnitude of this phenomenon in aneurysmal rupture as well as in head injury [7–12, 15, 17, 19]. In most instances, however, these studies have failed to provide convincing evidence correlating blood flow velocities with neurological outcome, especially in head injury [6, 10, 13]. Furthermore, most of these studies have been mainly concerned with the evaluation of the anterior cerebral vessels so that until very recently, there were no reliable sonographic criteria for the diagnosis of basilar vasospasm. Relying on the TCD criteria for the diagnosis of basilar artery vasospasm provided by Sloan et al. [14], we found in a retrospective analysis of patients suffering from SAH that basilar vasospasm was more frequent than previously thought. Moreover, flow velocities in the basilar artery proved to correlate to outcome, suggesting that basilar vasospasm may result in secondary brainstem damage, especially following traumatic subarachnoid haemorrhage. These results were compatible with the observations made by Lee et al. [7]. Using TCD and blood flow measurements, these authors showed that that basilar vasospasm was a significant predictor of poor outcome in head injured patients, independently from their age or neurological condition on admission [7].

In order to further elucidate the role played by basilar vasospasm, we performed a prospective TCD study on patients suffering from SAH and analyzed the relationship between daily changes in basilar flow velocities and neurological condition and outcome.
Patients and Methods

Patients

Between November 1996 and January 1999, 116 consecutive patients with subarachnoid haemorrhage were prospectively evaluated in the department of Neurosurgery in Rambam Medical Center. There were 50 women and 66 men, ranging in age from 14 to 84 years with a mean of 45.2 years (standard deviation: 19.2 years). Subarachnoid haemorrhage was of traumatic origin (tSAH) in 59 patients and spontaneous (sSAH) in 57 patients. Within the spectrum of tSAH, both subarachnoid and intraventricular haemorrhages were included. Patients with brain contusions were also recruited whenever associated cortical SAH was evident. Patients with tSAH were included. Patients with brain contusions were also recruited.

Management Protocol

All patients were admitted to the department after initial CT scan evaluation and resuscitation measures. Mechanical ventilation, sedation and intracranial pressure monitoring were performed as indicated. Increased intracranial pressure was treated by hyperventilation guided by jugular bulb oxymetry and 20% mannitol. Propofol and atracurium were selected for ventilation and intracranial pressure control because of their rapid onset and offset allowing neurological evaluation as needed. In all patients, fluid regimen aimed at the maintenance of mean arterial pressure at 95 to 100 mm Hg and haematocrit at 30 to 35%. Nimodipine was administered routinely only in the sSAH group (2 mg/h). In the tSAH group, only patients with TCD evidence of vasospasm for at least 48 hours and jugular bulb oxymetry less than 80% were treated by nimodipine.

TCD Recordings

TCD evaluations were performed using an Intraview system (Rimed Inc., Raanana, Israel) with a 2-MHz pulse-waved range-gated transducer, according to the technique described by Aaslid et al. [1]. Both the middle (MCAs) and anterior cerebral arteries (ACAs) were insonated through the temporal acoustic window, whereas the vertebral and basilar arteries were assessed through the foramen magnum. Basilar artery location was defined at an insonation depth above 80 mm, according to the technique described by Fujioka and Douville [4]. Internal carotid arteries (ICAs) were insonated extracranially below the mandible. For each vessel, both mean flow velocity (FV) and Gosling pulsatility index (PI) were recorded and selected for analysis.

TCD recordings were performed within the first 48 hours after admission and thereafter every day until the patients’ discharge or TCD stabilization at normal FV values. A total of 578 TCD studies were performed in 116 patients (mean 7.56 studies per patient).

Vasospasm in the MCA and ACA was defined by a mean FV exceeding 120 cm/s and three times the mean FV of the ipsilateral ICA, according to Aaslid et al. [2] and Lindegaard et al. [8]. Basilar artery (BA) vasospasm was defined as moderate whenever the FV was higher than 60 cm/s and severe above 85 cm/s. Similarly, in an attempt to differentiate between basilar hyperemia and vasospasm, the ratio between BA and ICA FV was also calculated and analyzed.

Clinical Features

Neurological status was evaluated on admission by means of Glasgow Coma Scale score [18] after resuscitation measures and offset of sedative drugs. During the hospitalization course, the Glasgow Coma Scale score (GCS) was recorded at least every 8 hours.

Statistical Analysis

Analysis of variance, Chi-square and Fisher tests were used for statistical analysis. Correlations between GCS and FVs within different clinical subsets of patients were made by using a normalized GCS score, which took into account substantial differences in GCS range between sSAH and tSAH patients. Correlations’ trends were assessed by multiple regression analysis. In all statistical tests, a P value less than 0.05 was considered to be statistically significant.

Results

Vasospasm: Overall Results

Complete TCD evaluations could be achieved in 108 patients (93.1%). In 8 patients some at least of the anterior vessels could not be insonated because of an inadequate acoustic temporal window. The basilar artery, however, could be investigated in all instances. Forty-eight patients (42.2%) had TCD signs of vasospasm in the MCA and/or the ACA. Among these patients, 37 (75.5%) had associated vasospasm of the BA. Vasospasm of the anterior circulation vessels was as common in tSAH as in sSAH patients.

Sixty-two patients (53.4%) had elevated FVs in the BA, among these 34 (54.8%) had FVs above 85 cm/s. Basilar vasospasm was significantly more common in head-injured patients (59.7%) than in sSAH patients (40.3%, P = 0.041). Correlation between the amount of subarachnoid haemorrhage on CT scan and FVs was found with the BA (P = 0.028) but not with the anterior circulation vessels. As a correlate, the incidence BA vasospasm was significantly higher in patients with severe SAH (P = 0.008).

Patients with BA vasospasm were significantly younger than the remaining patients (Table 1). On the contrary, there was no difference in the incidence of associated non-neurological injuries or systemic complications such as sepsis between patients with or without BA vasospasm (Table 1). Increased ICP, however,