Two-Stage Cerebral Hemodynamic Changes in Staged Carotid Angioplasty and Stenting

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Background/Objective: We sought to assess the different patterns of cerebral hemodynamic changes in staged carotid angioplasty and stenting (CAS). *Methods:* We prospectively recruited a cohort of patients with regular angioplasty or staged angioplasty from October 2013 to August 2015. The hemodynamic changes, including peak systolic, end-diastolic, and mean flow velocities in the ipsilateral middle cerebral artery (MCA) and the contralateral anterior cerebral artery (ACA), were recorded by transcranial Doppler ultrasound. *Results:* Between October 2013 to August 2015, 25 patients (age range from 48 to 78 years, 96% male) with CAS were recruited, of whom 13 patients received staged angioplasty and 12 patients received regular angioplasty. Patients in the staged angioplasty group showed a higher degree of stenosis in the internal carotid artery. After the procedure, the peak systolic, diastolic, and mean flow velocities in the ipsilateral MCA in the staged angioplasty group were significantly higher than those in the regular angioplasty group (P = .028, .036, and .027, respectively). In the staged angioplasty group, first, the peak systolic, end-diastolic, and mean flow velocities in the contralateral ACA decreased significantly soon after balloon dilation in stage I (P = .042, .033, and .034, respectively). Second, the peak systolic, diastolic, and mean flow velocities in the ipsilateral MCA increased after stent placement in stage II (P = .006, .042, and .003, respectively). *Conclusions:* Two-stage hemodynamic changes were observed in staged angioplasty. The velocities in the ipsilateral MCA started to increase significantly after the collateral flow diminished. **Key Words:** Ultrasonography—Doppler—transcranial—carotid stenosis—angioplasty—stents.

Introduction

Atherosclerotic stenosis of the internal carotid artery (ICA) causes 10%-15% of all strokes.[1] For symptomatic carotid stenosis, carotid angioplasty and stent placement have been introduced as a safe alternative to carotid endarterectomy with similar long-term functional outcome and risk of fatal or disabling stroke.[1] Besides regular carotid angioplasty and stenting (CAS), a new technique, named staged CAS, has been explored. Patients with staged CAS would undergo balloon angioplasty with an undersized balloon several days before stenting.[2,3]

Previous studies have proven that regular CAS can help achieve intracranial hemodynamic stabilization.[4,5] However, little is known about staged CAS. The main purpose of our study was to assess, with transcranial Doppler (TCD) ultrasound, the different patterns of hemodynamic change in regular CAS group and staged CAS group.

Materials and Methods

Patients

The study population was a prospective cohort of consecutive patients over the age of 18 years old who

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presented with an ICA stenosis of more than 50% and received CAS between October 2013 and August 2015. The eligibility criterion was a demonstrated stenosis of the ICA of more than 50% on digital subtraction angiography (DSA). Exclusion criteria were as follows: inadequate acoustic temporal windows and retrograde flow in the contralateral anterior cerebral artery (ACA) from the ipsilateral ACA via the anterior communicating artery detected on TCD due to serious stenosis or complete occlusion in the contralateral ICA. Informed consent was obtained from all patients before the treatment.

Baseline risk factors for atherosclerosis, which included age, sex, hypertension, dyslipidemia, diabetes mellitus, history of ischemic heart disease, and smoking, were investigated for each patient. The degree of stenosis was calculated from DSA using the methods of the North American Symptomatic Carotid Endarterectomy Trial and was stratified into the following categories: 0%-50% stenosis, 50%-69% stenosis, 70% or more than 70% stenosis to near occlusion, near occlusion, and total occlusion.[6]

Carotid Artery Stent Placement Protocol

Patients who had a high degree of carotid stenosis (>90%) or were considered at high risk of postprocedural hyperperfusion syndrome (HS) based on their clinical presentations were scheduled for staged CAS and others received regular CAS.[3] All procedures were performed by the femoral approach by using the Seldinger technique. After systemic heparinization, an 8-French interventional sheath inserted via the femoral artery was introduced into the target common carotid artery and a guidewire was inserted into the distal part of the ICA. Distal embolus protection devices (Emboshield, Abbott, USA) were used.

In the regular CAS group, the stenosis was predilated with a semicompliant balloon (LitePAC, Abbott, USA) and a self-expanding nitinol stent (Precise, Cordis, USA; Acculink, Abbott, USA) was deployed. In the staged CAS group, an undersized and semicompliant balloon was inserted into the stenotic lesion and inflated with nominal pressure for 60 seconds (the diameter at full dilation was usually 2-4 mm) in stage I. When the minimum luminal diameter exceeded 2.0 mm on DSA, the procedure was considered successful and finished. In stage II, the stenosis was predilated with a semicompliant balloon (LitePAC), and a self-expanding nitinol stent (Precise, Acculink) was deployed. In our study, the median interval between stages I and II was 7 days (range from 4 to 47 days).

After the procedure, all the patients were under continuous blood pressure monitoring for 24 hours and signs of neurological deficits (such as aphasia, dysarthria, and hemiplegia) were recorded. Information about postprocedural computed tomography or magnetic resonance imaging was also collected.

TCD Examination and TCD Indices

TCD was performed using a digital power-motion Doppler unit with 2-MHz pulsed-wave diagnostic transducers. Middle cerebral artery (MCA) and ACA were insonated through the temporal window at depths of 50-65 mm and 65-75 mm, respectively. The TCD examination was done before the procedure, soon after balloon dilation and after stent placement. The peak systolic velocity (PSV), end-diastolic velocity (EDV), mean flow velocity (MV), and pulsatility index (PI) in the ipsilateral MCA and contralateral ACA were measured. One doctor did all the TCD examinations to maintain a constant angle of insonation.

PSV and EDV represent the highest and lowest flow velocities during the cardiac cycle. MV is a central parameter in TCD and is equal to $[PSV + (EDV \times 2)]/3$.[7] In our study, a decreased value indicates hypotension or decreased cerebral blood flow (CBF).[8] The PI provides information on downstream cerebral vascular resistance and is equal to $(PSV - EDV)/MV$.[9] Proximal stenosis or occlusion may lower the PI due to downstream arteriolar vasodilation, whereas distal occlusion or constriction may increase the PI.[10]

Statistical Analysis

Statistical analyses were performed using SPSS, Version 21 (SPSS Inc., Chicago, IL). A P value less than .05 was considered to indicate statistical significance. Quantitative variables are given as mean ± standard deviation, and qualitative variables are expressed as frequency and percentage. Continuous variables were compared by Student's t-test and categorical variables were compared by chi-square or Fisher's exact test. To evaluate hemodynamic effects, a paired t-test was used to compare the TCD parameters before and after CAS.

Results

From October 2013 to August 2015, 42 patients underwent CAS, of which 33 patients were assessed by TCD before and after the procedure. Eight patients were excluded from the study due to retrograde flow of the contralateral ACA (n = 4, 3 patients with contralateral ICA occlusion and 1 patient with contralateral ICA near occlusion) and incomplete TCD examinations (n = 4). Therefore, the final sample for this analysis included 25 participants of whom 13 patients received staged CAS and 12 patients received regular CAS. DSA found that 19 (76%) of 25 patients were presented with the anterior communicating artery. The median interval between stages I and II was 7 days. The 2 groups did not differ in age, sex, or risk factors of cerebrovascular diseases, as shown in Table 1. Two cases of CAS are represented in Figures 1 and 2.