Comparison in two serial TCD findings of symptomatic middle cerebral artery (MCA) between MCA
disease and tandem arterial pathology
Kyung Bok Lee, Moo Young Ahn

Department of Neurology, College of Medicine, Soonchunhyang University, Seoul

= Abstract =

Background and Objective: Striatocapsular infarction is seen in both middle cerebral artery (MCA) territories on magnetic resonance angiography (MRA). This study aimed to compare transcranial Doppler (TCD) findings between MCA disease and tandem arterial pathology (TAP) to distinguish between these two types of stroke.

Method: Patients were selected who had striatocapsular infarction due to MCA disease or TAP. All patients underwent contrast-enhanced magnetic resonance angiography (CE-MRA) and TCD on the same day and 7 days later. Stenosis or occlusion of the MCA was defined as an average flow velocity of ≤80 cm/s, and partial or complete occlusion was determined using TIMI grade and TCD waveform analysis. Results: In the TAP group, 9 patients (39.1%) showed decreased or normalized flow velocity compared to only 3 patients (12.0%, P = 0.046) in the MCA disease group.

Conclusion: In patients with MCA disease, MRA and TCD findings with a decrease or normalization of flow velocity were less frequent compared to patients with TAP. These findings support the use of repeated TCD examinations to distinguish MCA disease from tandem arterial pathology.

Key words: TCD; Acute infarction, MCA

Address for Correspondence:
Kyung Bok Lee, M.D.
Department of Neurology, College of Medicine, SOONCHUNHYANG University, SOONCHUNHYANG University Hospital
22 Daesagwan-gil (657 Hannam-dong), Yongsan-gu, Seoul, 140-743, Korea
Telephone: +82-2-709-9026
FAX: +82-2-709-9226
E-mail: kblee@hosp.sch.ac.kr

INTRODUCTION

Striatocapsular infarctions are caused by simultaneous occlusion of more than one orifice among the
immediately adjacent small, long lenticulostriate arteries. In contrast to the original concept that striatocapsular infarctions occur nearly exclusively because of embolic occlusion of the proximal middle cerebral artery (MCA), a significant proportion of this type of infarction also occur as a result of in situ thrombosis of the MCA. According to the previous reports, the most frequent causes of striatocapsular infarcts are artery-to-artery embolism from internal carotid artery (ICA; 38%) and cardiogenic embolism (37%), as well as atherosclerotic disease in the MCA at the origin of lenticulostriates (32%). Furthermore, the MCA disease is a relatively common cause of stroke in patients with Asian ancestry, and is expected to be more important cause of striatocapsular infarctions.

The identification of stroke subtype is valuable for both practicing clinicians and the optimal design of clinical stroke trials because the etiology of ischemic stroke affects management, outcome, and prognosis. In addition, the treatment of intracranial atherosclerotic disease may be different from extracranial ICA disease as well as cardiogenic embolism. However, the TOAST (Trial of Org 10172 in Acute Stroke Treatment) system which is the most widely used stroke classification, is insufficient for differentiating between in situ thrombotic striatocapsular infarction and ICA disease or cardioembolism, although it exhibits higher agreement between physicians. Because of concomitant atherosclerotic changes in the proximal vessels or cardiogenic embolic sources and remnants of emboli in MCA (embolic stenosis), many striatocapsular infarcts are frequently included in the undetermined etiology (two or more) according to TOAST classification.

Recently, contrast enhanced (CE) MRA is advantageous in displaying detailed vessel anatomy including that of aortic arch, and in reducing the appearance of artifacts. However, either middle cerebral artery (MCA) disease or embolism from proximal atherosclerosis can manifest MCA occlusion on angiography. In this study, we were to determine were to elucidate the differences in serial Transcranial Doppler (TCD) findings between intra cranial MCA disease and tandem arterial pathology.

**MATERIAL AND METHODS**

We prospectively recruited patients with acute MCA infarction involving striatocapsular region who were admitted to the SOONCHUNHYANG University Hospital between March 2005 and April 2007. The inclusion criteria included an acute infarction which was larger than 15mm on axial DWI and located in the lenticulostriate artery territory with or without infarction in the superficial perforator territory, as defined according to the previously published templates. Patients with multiple small infarctions in the lenticulostriate territory which were unlikely to be lacunar infarction were also included. All patients underwent conventional MRI and axial/coronal DWI on a 1.5-T system with echo-planar imaging capability (Sonata; Simens Medical System) within 3 days of stroke onset. DWI was obtained in the three planes with a single-shot, echo-planar, spin-echo pulse sequence with a TR of 2500 ms, a TE of 76 ms, one excitation, and two B values (0 and 1000 s/mm²). We determined the presence of MCA M1 stenosis on CE-MRA using a previously described method. A diagnosis of significant (>50%) intracranial and extracranial stenosis was made by two experienced stroke neurologist, and was confirmed by a neuroradiologist. We executed tests for proximal embolic sources
(tranthoracic echocardiography, TTE; transesophageal echocardiography, TEE; Holter monitoring; aortic arch CE-MRA) in most patients, and high- and low-risk cardioaortic sources were distinguished using an arbitrary 2% annual or on-time primary stroke risk threshold. Sources associated with high primary risk for ischemic stroke include left atrial thrombus, left ventricular thrombus, atrial fibrillation, paroxysmal atrial fibrillation, sick sinus syndrome, sustained atrial flutter, recent myocardial infarction (within 1 month), rheumatoid mitral or aortic valve disease, bioprosthetic and mechanical heart valves, chronic myocardial infarction together with low ejection fraction less than 28%, symptomatic congestive heart failure with ejection fraction less than 30%, dilated cardiomyopathy, and nonbacterial thrombotic endocarditis. Stroke mechanisms were classified into MCA disease (MCAD), stroke of tandem arterial pathology (STAP), and stroke from proximal embolism (SPE). For example, because the absence of M1 stenosis is generally considered as recanalization of an embolus, patients without MCA stenosis were classified as SPE independent of evidence of proximal embolic sources, and patients who had M1 stenosis without proximal embolic source documented were classified as MCAD. However, patients who had both MCA stenosis and a proximal embolic source were defined as STAP. For these patients, transcranial Doppler (TCD) were performed 0 days and 7 days after CE-MRA. For the analysis of TCD diagnosis of M1 stenosis, we used a mean flow velocity (MFV) cutoff of (80 cm/s in the MCA as the criterion for stenosis. The MFVs of MCA were checked at depth of 60 and 66 mm on both sides. Complete or partial occlusions were diagnosed according to the TIMI flow grades and TCD wave forms. All data were analyzed by SPSS 15.0 software (Chicago, IL, USA). The (2 or Fisher’s exact test and Kruskal-Wallis test were used to compare the frequency of recanalization with respect to stroke subtypes. Results are presented as odds ration (OR) as estimates of relative risk with 95% confidence interval (CI). A probability value of P<0.05 was considered statistically significant.

RESULTS
The study population comprised STAP (n=23) and MCAD group (n=25) aged of 62.1(9.84 (mean(SD) years. CE-MRA including aortic arch were done in all patients, and TTE, TEE, and Holter monitoring for the proximal embolic source were performed in 100%, 78.9%, and 90.1%, respectively. In the two serial TCD performed with a 1-week interval in the STAP group, 9 patients (39.1%) showed a reduced or normalized MCA flow velocity. These findings were observed in only 3 patients in the MCAD group (12.0%, P=.046; table 1).
Table 1. Two serial TCD findings of symptomatic MCA between STAP and MCAD groups.

<table>
<thead>
<tr>
<th>TCD findings</th>
<th>STAP (n=23)</th>
<th>MCAD (n=25)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial</td>
<td>Follow-up</td>
</tr>
<tr>
<td>Complete or partial occlusion (TIMI 0-2)</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Stenotic flow (MFV(80)</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>Normal flow (MFV&lt;80)</td>
<td>5</td>
<td>14</td>
</tr>
<tr>
<td>Poor temporal window</td>
<td>4</td>
<td>4</td>
</tr>
</tbody>
</table>

We compared intracranial MRA images between the STAP and MCAD group. There were 16 proximal M1 stenoocclusion and 9 distal M1 stenoocclusion in the MCAD group. However, the STAP group had 13 proximal M1 stenoocclusion and 10 distal M1 stenoocclusion (P=.769). There was a tendency toward more frequently occurring abrupt M1 cutoff with otherwise normal appearing arteries in the STAP group (13/23 patients, 56.5%) than in the MCAD group (7/25 patients, 28.0%, P=.078). There were 15 patients with other
intracranial stenotic lesions in the MCAD group, and 7 in the STAP group ($P=0.049$).

**DISCUSSION**

We have demonstrated that TCD can be a useful for elucidating stroke mechanisms in striatocapsular infarctions. There is a significantly reduced or normalization of flow velocity on TCD in STAP group. The location of MCA M1 stenosis was not significantly different between the STAP and MCAD group. The patients of STAP group could be reclassified as a SPE or MCAD case after confirmation of recanalization on angiography or TCD. Actually, many cases of the STAP group might have been included in SPE group, because STAP group had an tendency toward more frequently occurring abrupt cutoffs on MRA and a higher rate of normalization of flow velocity on TCD. Complete recanalization of a prior occlusion by angiographic methods or ultrasound usually supports an embolic cause. 

A limitation of this study is that although the MCA flow velocities had reduced in the STAP group, it may have been attributed to mere hemodynamic changes (decreased luxury perfusion). Future studies should employ angiography to monitor serial changes of the MCA stenoses.

In conclusion, significantly more patients with STAP showed reduced or normalized MCA flow velocity on follow-up TCD. This finding suggests that serial TCD as well as angiography can be used to discriminate intracranial MCA disease from stroke of proximal artery to artery embolization. It could therefore be helpful for determining the mechanisms in patients with striatocapsular infarctions that are currently described as undetermined etiology according to TOAST classification.

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