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MR Imaging of the Internal Carotid Artery in Ischemic Cerebrovascular Disorders

—Clinical and Angiographic Correlation—

Jae Hong Lee, M.D., Byung-Woo Yoon, M.D., Jae Kyu Roh, M.D., Kee Hyun Chang*, M.D., Sang Bok Lee, M.D., Ho Jin Myung, M.D.

*Department of Neurology, Department of Radiology**
College of Medicine, Seoul National University, Seoul, Korea

This study was intended to correlate the appearance of the cavernous segment of the carotid artery on MR images with the presence of significant stenosis or occlusion of the cervical carotid artery as seen on angiograms in 37 patients with cerebrovascular disorders who had brain MRI and arteriography. Three patients demonstrated an isointense signal within the carotid artery's cavernous segment, correlating with complete carotid occlusion as seen angiographically. Ten patients had variable signal intensity and/or luminal narrowing in the carotid siphon; seven of these findings correlated with angiographic evidence of carotid occlusion, while carotid branch occlusion was seen angiographically in the other three. The demonstration of normal signal void within a normal-appearing cavernous segment of the internal carotid artery in the remaining 24 patients correlated with an absence of significant stenosis within the cervical segment in 21 patients. In the remaining three, significant disease of the internal carotid artery was found.
Isointensity or luminal irregularity within the intracranial carotid artery can indicate complete occlusion or slow flow. The presence of normal flow void in the intracranial segment does not exclude significant abnormality of the cervical segment of the carotid artery.

Key Words: *Cerebrovascular disorders, brain MRI, flow signal void, carotid artery stenosis or occlusion*

INTRODUCTION

Magnetic resonance(MR) imaging has great potential not only for anatomic depiction of blood vessel morphology but also for its flow state due to flow phenomena inherent to the MR methodology (Bradley et al., 1985; Atlas et al., 1988). Some recent reports suggested that the appearance of the intracranial carotid artery on routine MR brain scans can reliably assess the status of the subcranial carotid artery (Alverez et al., 1986; Heinz et al., 1989).

Address for Correspondence: *Jae Hong Lee, Department of Neurology, Asan Medical Center, 388-1, Pungnap-Dong, Songpa-Gu, Seoul 138-040, Korea (Tel: 02-480-3098)*
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This retrospective study on MR and angiographic correlations was undertaken to evaluate the capability of predicting carotid stenosis or occlusion by recognition of altered MR appearance of the cavernous carotid artery.

PATIENTS AND METHODS

We reviewed the medical records of patients with cerebrovascular disorders who had had both cerebral angiograms and brain MR scans between June 1988 to August 1990. Thirty-seven patients with angiographic abnormalities of the internal carotid artery (ICA) were selected. Thirty-one patients were men and six were women with ages ranging from 17 to 73 years (mean-53.0 years). The angiographic studies were all intraarterial and included views of the cervical carotid portion. The MR imaging was performed with a 2.0T superconducting unit (Spectro-20000, Goldstar, Seoul, Korea) with multislice spin echo (SE) sequences. The proton density images (TR/TE 2500/30) obtained in axial plane were selected for reviewing the cavernous portion of the ICA. Slice-thickness was 5mm and the interslice gap was 2mm. The acquisition matrix was either 180×256 or 256×256. The MR images were assessed with respect to normal signal void in the intracranial carotid segment to the level of the clinoid. Specifically, the immediate subclinoid segment in the axial plane was scrutinized in order to avoid the partial volume effect inherent to the cavernous or petrous segment.

When the flow signal voids of the cavernous ICA were invisible and replaced by abnormal signal intensities, they were described as high signal, iso-signal, and low signal respectively, compared with signal intensity of the brain parenchyme. The cavernous ICA was designated to be narrowed, when the diameter was smaller than or equal to that of the basilar artery on the axial images.

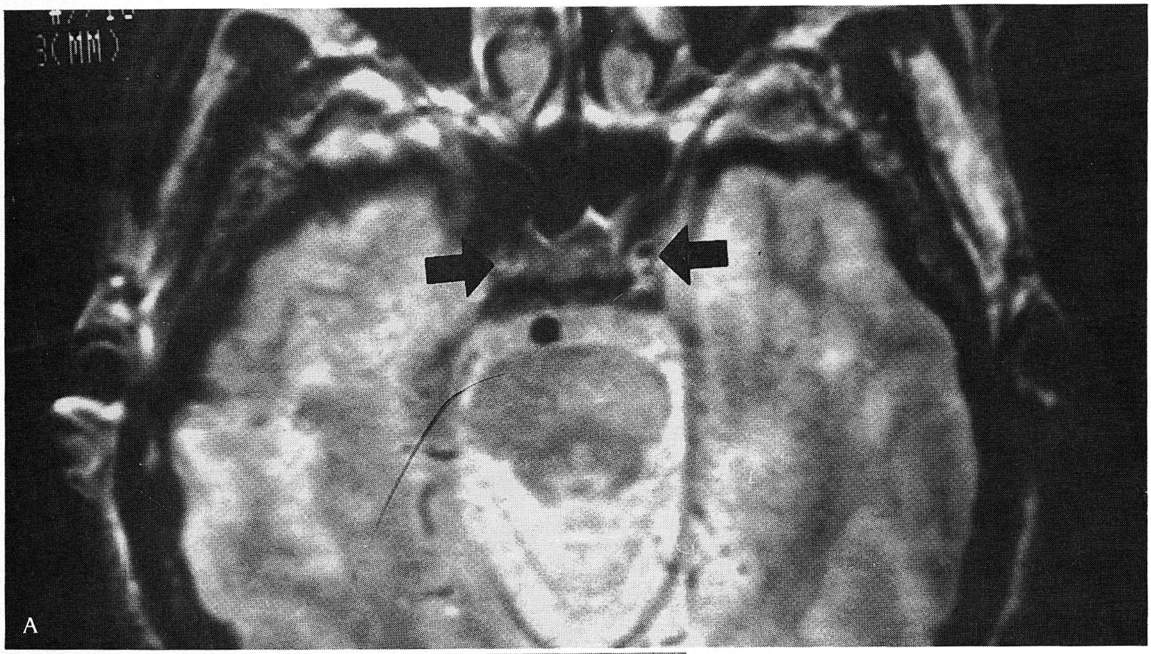
RESULTS

Twelve of the 37 patients showed an altered MR appearance of the cavernous segment of ICA. Clinical data of those who showed an abnormal flow signal void on MR are detailed in table 1. Eleven patients had

Table 1. Demographic and neuroimaging stuy data of which carotid flow voids on MR are abnormal

Patient No.	Sex/age	Neurologic disorder	Angiography	Carotid flow void on MR
1	M/63	R) MCA infarction	R) ICA proximal occlusion	R) isosignal
2	M/54	L) Borderzone infarction	L) ICA proximal occlusion	L) isosignal
3	F/45	Moyamoya disease	L) ICA proximal occlusion R) ICA distal occlusion	L) isosignal R) luminal narrowing
4	M/47	R) basal ganglia infarction	R) M1 occlusion	R) high signal
5	F/44	Corpus callosum infarction	L) ICA proximal occlusion	L) low signal
6	M/56	L) MCA infarction	B) ICA proximal occlusion	L) low signal B) luminal narrowig
7	M/60	L) MCA infarction	L) ICA proximal occlusion	L) low signal L) luminal narrowing
8	M/66	L) MCA infarction	L) ICA proximal occlusion	L) low signal L) luminal narrowing
9	M/43	L) MCA infarction	L) MCA occlusion	L) luminal narrowing
10	M/64	L) MCA infarction	B) ICA proximal occlusion	L) luminal narrowing
11	M/51	L) MCA infarction	L) ICA distal occlusion	L) luminal narrowing
12	M/45	L) basal ganglia infarction	L) M1 occlusion	L) luminal narrowing

R=right, L=left, B=both
ICA=internal carotid artery, MCA=middle cerebral artery,
M1=proximal horizontal portion of middle cerebral artery



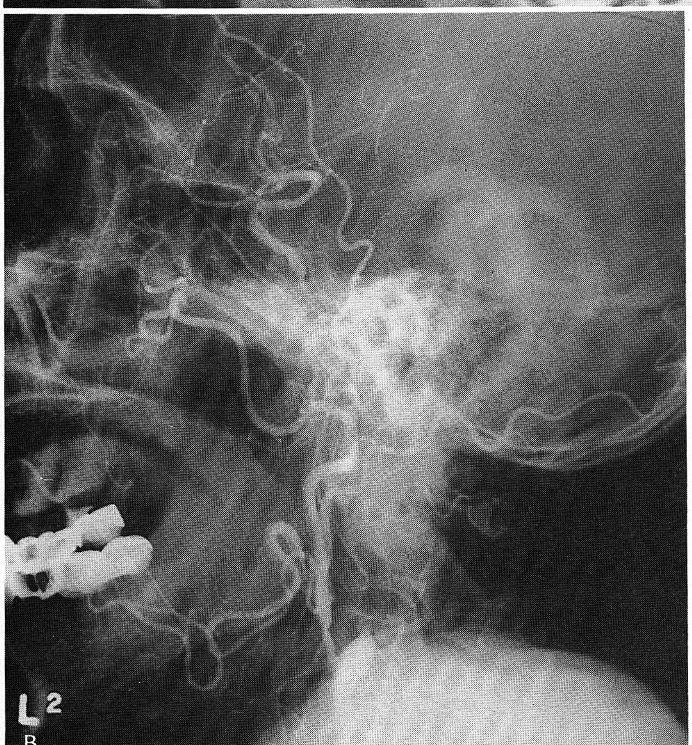


Fig. 1. 64-year-old man with acute onset of right hemiparesis.
A. MR scan, 2500/30, at level of the cavernous ICA shows normal flow signal void (arrows) in both carotid arteries.
B. Angiogram shows complete cervical ICA occlusion with patent cavernous siphon due to collateral blood supply from the ophthalmic artery.

cerebral infarctions and one had moyamoya disease. Eight patients showed abnormal signal intensities and eight revealed narrowing of the lumen (Four had both.).

The abnormalities observed on cerebral angiography are summarized in table 2. Nine patients had a mild degree of vessel wall irregularity. Six had ICA narrowing and 13 ICA occlusion. Nine patients had ICA branch occlusion (MCA 8, ACA 1).

Twenty-nine of the 37 patients showed normal signal void within the cavernous carotid lumen on MR. All but one had cervical ICA patency on angiogram. The one patient who showed cervical ICA occlusion had collateral blood supply from the ophthalmic artery and the cavernous portion of the ICA was visualized on angiogram (Fig. 1).

Signal intensities of cavernous ICA on MR were compared with ICA patency on angiogram in table 3. Compared with the signal intensity of the brain parenchyme, high signal, iso-signal and low signal intensity were designated. Eight patients showed abnormal signal intensies; 3 iso-signal, 1 high signal, and 4 low signal intensities. Three patients with iso-signal intensity all showed cervical ICA occlusion on angiogram (Fig. 2). One patient with high signal intensity had a patent cervical ICA. All the patients with low signal intensity showed cervical ICA occlusion but cavernous ICA patency was observed in two of them by collateral circulation through the ophthalmic artery.

Lumen size of the cavernous ICA on MR was compared with the state of the cervical ICA on angiogram in table 4. The five cases in which the lumen contour could not be exactly delineated by abnormal signal intensity were excluded. Twenty-four patients showed normal lumen size of the cavernous ICA. Among them, 21 had a normal cervical ICA on angiogram and 3 showed stenosis of cervical ICA. Eight patients showed a narrowing of the cavernous ICA lumen size on MR; 5 patients demonstrated cervical ICA occlusion on angiogram, while no significant disease of the cervical ICA was found in the other three. There was a statistical significance ($p<0.001$) between narrowing of lumen size of the cavernous ICA on MR and stenosis or occlusion of the cervical ICA on angiogram.

Table 2. Findings of cerebral arteriography

1) Mild vessel wall irregularity		(n=9)
2) ICA narrowing		(n=6)
cervical	3	
cavernous	1	
supraclinoid	2	
3) ICA occlusion		(n=13)
cervical	8	
cavernous	0	
supraclinoid	5	
4) ICA branch occlusion		(n=9)
MCA	8	
ACA	1	
Total		37

ICA=internal carotid artery
MCA=middle cerebral artery
ACA=anterior cerebral artery

DISCUSSION

The detection of significant carotid stenosis or occlusion on routine brain MR imaging is an important finding. There are many times in clinical practice when extracranial carotid stenotic or occlusive disease is not suspected and brain MR imaging incidentally suggests its presence. In patients with suspected cerebrovascu-

Table 3. Signal intensities (S.I.) of cavernous ICA on MR vs. Cervical & cavernous ICA patency on angiogram

S.I. of cavernous ICA	Cervical ICA		Cavernous ICA	
	patent	occluded	patent	occluded
1) Normal flow void (n=29)	28	1	29	0
2) Isosignal intensity (n=3)	0	3	0	3
3) High signal intensity (n=1)	1	0	1	0
4) Low signal intensity (n=4)	0	4	2	2

Table 4. Cavernous ICA luminal size on MR vs. Cervical ICA on angiogram

Lumen size of cavernous ICA	Cervical carotid artery		
	Normal	Stenosis	Occlusion
Normal (n=24)	21	3	0
Narrowing (n=8)	3*	0	5

*: cavernous ICA narrowing (1), supraclinoid ICA occlusion (1), MCA occlusion (1) (Chi-square=18.0, $\lambda=0.625$, $p<0.001$)

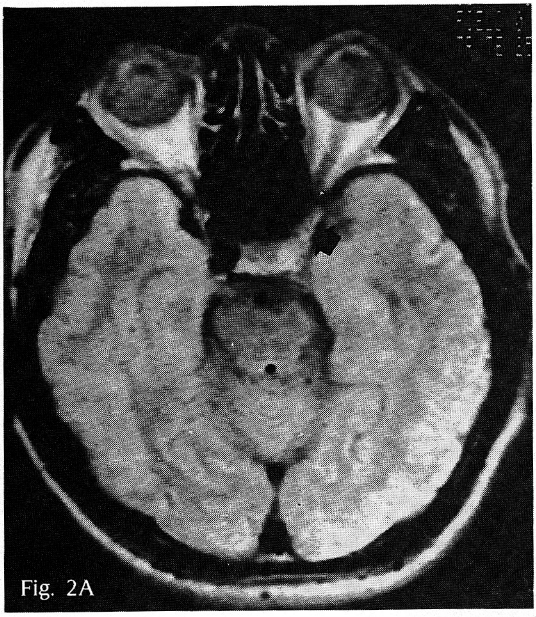


Fig. 2A

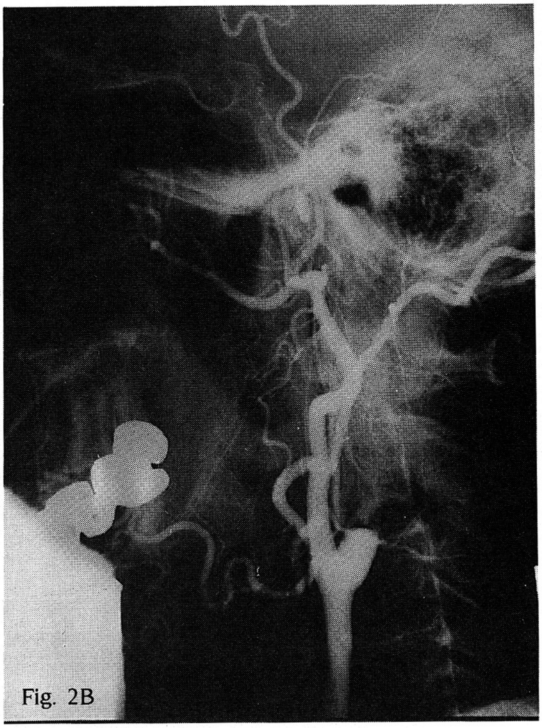


Fig. 2B

Fig. 2. 54-year-old man with sudden onset of right arm weakness and left borderzone infarction documented on MR scan.

A. MR image, 2500/30, shows isointense signal from left carotid ICA (arrow).

B. Angiogram shows complete occlusion of the cervical portion of the left ICA.

lar disease, MR imaging is often the first technique that shows an alteration of flow and leads to angiography, which documents it.

The carotid arterial lumen on MR in the healthy subject is usually hypointense secondary to high-velocity signal loss in rapidly flowing blood as seen on intermediate and T2-weighted images (Heinz et al., 1989). When significant stenosis or complete occlusion is present, the signal from protons remaining within the volume being imaged (thrombosis or arrested flow) gives rise to an increased signal within the carotid artery that is most easily seen within the horizontal canal in the petrous bone (Brant-Zawadzki, 1990).

This retrospective review seems to provide the evidence that the MR appearance of the intracranial carotid artery can be used to assess the status of extracranial carotid artery. All patients with an isointense signal within the cavernous ICA had complete occlusion of the cervical ICA. The reverse was not necessarily true. In three of our patients with complete occlusion of the cervical ICA, a normal flow void was found in one and a low signal was demonstrared in the other two patients. Other signal intensities (high or low signal) of the cavernous ICA could not be correlated with the status (stenosis or occlusion) of the cervical ICA. The finding of luminal narrowing of the cavernous ICA on MR also suggests the possibility of significant compromise of the cervical segment of the ICA. Five of 8 patients with luminal narrowing of the cavernous ICA on MR showed cervical ICA occlusion on angiogram.

There are, however, some caveats in predicting the status of the extracranial carotid artery using the MR appearance of the intracranial carotid artery. For example, collateral circulation through the ophthalmic artery may mask the abnormality of the flow signal void. Flow phenomena distal to the cavernous ICA such as MCA occlusion also may affect the signal void of the cavernous ICA. The significance of high signal or low signal intensity cannot be clarified in terms of the flow status of the extracranial carotid artery.

The presence of normal flow void in the intracranial carotid artery is somewhat reassuring, but it does not always exclude significant abnormality of the cervical segment of the ICA, as shown in one of our cases. Carotid angiography still must be performed, when the clinical indications exist, to confirm the status of the artery. Nevertheless, the flow void of the cavernous ICA on MR reliably suggests the status of the cervical segment of the internal carotid artery.

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