Intracranial atherosclerotic stroke (ICAS) is a common stroke subtype, particularly in Asians. ICAS can be caused by various stroke mechanisms, including branch occlusive disease (BOD), which involves subcortical infarcts caused by parent arterial disease occluding the perforator’s orifice, and non-BOD, which involves infarcts beyond the subcortical area caused by artery-to-artery embolism. Although the clinical features may be similar between the BOD and non-BOD types, the underlying stroke mechanisms in patients with intracranial atherosclerosis may differ between the BOD- and non-BOD–type ICAS, requiring different treatment approaches.

With high-resolution magnetic resonance imaging (HR-MRI) techniques, vessel wall imaging findings have been reported for the various causes of intracranial stenosis. HR-MRI can be used to determine the mechanisms of ischemic stroke in patients with ICAS, plaque rupture, and plaque overgrowth of the perforator artery ostia. Intracranial plaque and remodeling patterns (positive or negative) may occur in association with ICAS mechanisms (BOD versus non-BOD). An intravascular ultrasound study showed that in patients with coronary syndromes, positive remodeling was associated with unstable plaques. The information on the location, extent, and stability of the plaques, as well as on the vascular remodeling, may guide treatment strategies in patients with ICAS.

In the present study, we hypothesized that the BOD type is not a milder form of ICAS and is different from the non-BOD–type ICAS in terms of the direction of arterial remodeling and plaque morphology. Specifically, unstable and large plaque burden with positive remodeling may be associated with the non-BOD–type ICAS like coronary plaques, whereas

Background and Purpose—Intracranial atherosclerotic stroke (ICAS) has various stroke mechanisms, including branch occlusive disease (BOD), subcortical infarcts caused by parent arterial disease occluding the perforator’s orifice, and non-BOD, infarcts beyond the subcortical area caused by artery-to-artery embolism. To test whether these 2 types of ICAS had different vascular pathophysologies, we compared the high-resolution magnetic resonance imaging characteristics between BOD and non-BOD ICAS.

Methods—Eighty patients with acute infarcts caused by ICAS of proximal middle cerebral artery or basilar artery without carotid/cardiac embolic sources or nonatherosclerotic causes were enrolled (36 BOD and 44 non-BOD patients). The steno-occlusive intracranial artery at the maximal stenosis was analyzed for vascular remodeling and wall enhancement.

Results—BOD had distinct radiological features in terms of vascular morphology and enhancement. BOD showed a milder stenosis than non-BOD (P<0.001). Positive remodeling was more frequently observed in non-BOD than in BOD (P=0.005). Wall area index was also lower in BOD. Plaque enhancement was observed in all but one non-BOD patient and in one fourth of BOD patients (P=0.003). Although both types showed an eccentric enhancement, this enhancement was more frequently distributed in the BOD group on the side where the perforators arose. As the number of asymptomatic intracranial stenosis increased, the degree of stenosis (rho=0.513, P=0.003) increased in the BOD group, whereas enhanced plaque area (rho=0.343, P=0.030) increased in the non-BOD group.

Conclusions—Our data indicate that BOD is a common and unique form of ICAS, distinct from non-BOD. These 2 types of ICAS have different vascular pathophysologies in terms of vascular remodeling and plaque characteristics.

Key Words: atherosclerosis ■ high-resolution wall MRI ■ intracranial stenosis ■ ischemic stroke ■ magnetic resonance imaging

Differential Vascular Pathophysiologic Types of Intracranial Atherosclerotic Stroke

A High-Resolution Wall Magnetic Resonance Imaging Study

Sookyung Ryoo, MD; Mi Ji Lee, MD; Jihoon Cha, MD; Pyoung Jeon, MD, PhD; Oh Young Bang, MD, PhD

Background and Purpose—Intracranial atherosclerotic stroke (ICAS) has various stroke mechanisms, including branch occlusive disease (BOD), subcortical infarcts caused by parent arterial disease occluding the perforator’s orifice, and non-BOD, infarcts beyond the subcortical area caused by artery-to-artery embolism. To test whether these 2 types of ICAS had different vascular pathophysologies, we compared the high-resolution magnetic resonance imaging characteristics between BOD and non-BOD ICAS.

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the progression of negative remodeling plaque may occlude the adjacent perforator ostia, as in the BOD-type ICAS. Thus, we evaluated the HR-MRI characteristics between BOD- and non-BOD–type ICAS, as well as the different degrees of intracranial atherosclerosis burden.

Methods

Patients and Workups

From January 2012 to February 2015, we prospectively recruited patients who were admitted to a tertiary university hospital if acute symptomatic ischemic infarctions because of intracranial atherosclerotic disease (ICAD) were present. Potential participants were defined as patients having focal or localized neurological symptoms within 7 days from onset, showing relevant lesions on diffusion-weighted imaging and any degree of stenosis on relevant intracranial vessels (M1 portion of the middle cerebral artery [MCA] or basilar artery [BA]). We only included patients who underwent HR-MRI. Patients with potential sources of cardioaortic embolism based on the modified Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification, extracranial atherosclerosis with significant (≥50%) stenosis in the relevant extracranial arteries, other stroke mechanisms (coagulopathy, Moyamoya disease, dissection, etc), or incomplete evaluations were excluded.

Clinical information, including age, sex, and vascular risk factors (hypertension, diabetes mellitus, dyslipidemia, coronary artery disease, smoking history, and history of previous stroke or transient ischemic attack), were acquired systematically. All patients underwent standardized diagnostic tests that included routine blood tests (total cholesterol, triglyceride, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, C-reactive protein, etc) and cardiac workups (electrocardiography, ≥24 hour cardiac telemetry, or echocardiography). Hematologic markers of prothrombotic tendency, including an antiphospholipid antibody, were evaluated in patients <50 years old. Local institutional review boards approved this study. All patients or patient guardians provided informed consent for participation.

We divided the patients into 2 groups according to their diffusion-weighted imaging lesion distribution as follows: (1) the BOD group, that is, patients with deep infarctions within the striatocapsular area, and (2) the non-BOD group, that is, patients with infarcts beyond the striatocapsular area (such as cortical infarctions, regardless of any subcortical deep infarcts).

The degree of asymptomatic ICAD was also measured in each patient. Atherosclerotic lesions of intracranial vessels on magnetic resonance angiography (MRA) were visually graded: 0, no stenosis; 1, stenosis ≤50%; 2, stenosis of 50% to 99%; and 3, occlusion. The assessment of the ICAD location included MCA, anterior and posterior cerebral arteries, BA, and intracranial portions of the internal carotid and vertebral arteries. The sum of the involved intracranial vessels was defined as the ICAD score.

MRI Protocols

All patients underwent MRI with a 3-tesla system (Achieva; Phillips Medical System, Best, the Netherlands) with a standard 8-channel head coil. Three-dimensional (3D), time-of-flight MRA of the intracranial arteries was initially performed with the following parameters: repetition time ($T_R$), 25 ms; echo time ($T_E$), 3.5 ms; slice thickness, 1.2 mm; slice spacing, 0.6 mm; flip angle, 20°; matrix, 880×332; and field of view, 25 cm. A neuroradiologist (J. Cha) selected the vessel and site of evaluation according to clinical presentation and 3D time-of-flight MRA findings and chose the combination of acquisition orientations (axial, or axial and sagittal). Black-blood HR-MRI using the spatial...
presaturation technique was performed with the following sequences: (1) axial and sagittal dual-echo proton density and T2-weighted images (TE, 2150 ms; TSE, 12.5/100 ms; echo train length, 10; slice thickness, 2 mm; no gap, 16 slices with 32 mm coverage; flip angle, 90°; matrix, 280×280; field of view, 14 cm; number of excitations, 2; acquisition time, 8 minutes 7 s); (2) sagittal T1 fluid-attenuated inversion recovery images were compared to determine the presence or absence and the pattern of enhancement. The presence of enhancement was defined as >20% increase in the normalized signal intensity of the plaque after contrast agent injection. Normalized signal intensity was calculated as (signal intensity of the plaque/signal intensity of the mid pons). Enhancement was considered eccentric if it was uniform or circumferential. Enhancement was regarded as eccentric if it was not 360° circumferential or if the thickest part was more than twice the thinnest part where circumferential enhancement was observed. Two neurologists (S.R. and M.J.L.) read the HR-MRI images. In cases of disagreement, a third reader was invited to resolve the issue. Enhanced area (EA) was also manually drawn. The enhancement degree was estimated using the following formula: EA/WA×100%. All quantitative data were remeasured 2 weeks later by a neurologist (S.R.) to estimate intraobserver variability. For most HR-MRI parameters, the inter- and intrarater reliability were good to excellent (online-only Data Supplement).

**Statistics**

We examined the differences between the groups via a χ² test or Fisher exact test for categorical variables and analyzed the differences using a t test or Mann–Whitney U test for continuous variables. All statistical analyses were conducted using commercially available software (SPSS for Windows, version 20; SPSS Inc. Chicago, IL). P<0.05 was considered to be statistically significant.

**Results**

**General Characteristics**

Among 92 patients who met the inclusion criteria, patients with HR-MRI findings of Moyamoya disease (n=4), arterial dissection (n=2), and reversible cerebral vasospasm syndrome (n=1); those with proximal sources of embolism documented during the follow-up, such as paroxysmal atrial fibrillation or cervical carotid artery (n=3); and 2 patients with complete recanalization on follow-up MRA, suggesting cryptogenic embolism, were excluded. Eighty patients were finally included in this study (male, n=49 [61.3%]; age, 64.5±13.7 years; MCA, 61 [76.3%]; and BA, 19 [23.7%]). Thirty six (45.0%) patients were allocated into the BOD group, and 44 (55.0%) patients were allocated into the non-BOD group. Risk factors and laboratory findings were similar between the 2 groups (Table 1). We found no differences in other risk factors or laboratory results.
HR-MRI Characteristics of BOD

BOD had distinct radiological features in terms of vascular morphology and enhancement (Table 2 and Figure 1). Patients in the BOD group had milder stenosis than patients in the non-BOD group (stenosis degree, 40.7%±27.4% versus 74.5%±19.9%, respectively; P<0.001). Remodeling index differed between the groups (1.01 versus 1.25, respectively; P=0.031). Most patients with BOD (78.1%) did not show positive remodeling (defined as remodeling index ≥1.2), whereas more than half of patients in the non-BOD group (52.8%) showed positive remodeling (P=0.004). Similarly, wall area index was also lower in the BOD group compared with the non-BOD group (2.82 versus 6.44, respectively; P<0.001). Although all symptomatic vessels in the non-BOD group (except for one) were enhanced, a substantial proportion of symptomatic vessels in the BOD group (25.0%) were not enhanced (P=0.003). EA within the wall was smaller in the BOD group than in non-BOD group (enhancement degree [median]: 39.7% versus 60.9%, respectively; P=0.005). Both BOD and non-BOD groups showed similar enhancement patterns; most of them were eccentric (BOD, 92.6%; and non-BOD, 90.7%). However, the enhancement location was different. Enhancement was more frequently distributed at the superior half of the MCA or the posterior half of the BA in the BOD group, where the perforators arose, compared with the non-BOD group (Figure 2).

ICAD burden (the presence of asymptomatic stenosis of any degree) was not different between the 2 groups (P=0.617) (Table 2 and Figure 3A). Regarding the ICAD burden, the HR-MRI characteristics were different between the 2 groups (Figure 3B). As ICAD scores increased, the degree of stenosis also increased in the BOD group (rho=0.513; P=0.003), although the EA did not increase (rho=0.275; P=0.121). In contrast, the EA got larger as ICAD scores increased in the non-BOD group (rho=0.343; P=0.030); the WA increased according to the EA (rho=0.523; P=0.001). The degree of stenosis did not correlate with ICAD scores in the non-BOD group.

Discussion

The main finding of this study is that BOD is a unique form of ICAS that is different from non-BOD ICAS. Patients with
BOD showed a characteristic remodeling pattern (less outward), size and location of plaque enhancement (a smaller plaque enhancement adjacent to the perforator ostia), and pattern of ICAS progression (progression of stenosis without enlargement of unstable plaque with the increase in the asymptomatic ICAD burden).

In the present study, both remodeling and plaque enhancement patterns were evaluated in both 2 phenotypes of ICAS. There are increasing evidence of the presence of plaque and pattern of enhancing plaque in patients with ICAS.\textsuperscript{16,17} Results of others and our studies showed that plaques tend to locate opposite to the orifices of perforating arteries.\textsuperscript{18} However, few studies have reported about the remodeling pattern in patients with ICAS. One recent study showed that positive remodeling pattern was associated with larger plaques in patients with basilar atherosclerosis.\textsuperscript{13} Other study showed that microembolic signals on transcranial Doppler were observed more frequently in patients with positive remodeling of MCA.\textsuperscript{19} Moreover, BOD often is lumped in with non-BOD as ICAS,\textsuperscript{20,21} or only BOD-type ICAS (eg, paramedian pontine infarcts)\textsuperscript{22} was studied in most studies. To the best of our knowledge, no previous study has specifically compared the vascular pathophysiology in terms of vascular remodeling and plaque characteristics between 2 types of ICAS. Our results are in line with the studies of coronary artery disease that positive remodeling and large enhancing plaques are associated with unstable plaque, resulting in rupture of plaque (non-BOD type) in patients with ICAS.\textsuperscript{12}

Our HR-MRI ICAS findings are in line with the ICAS luminal changes. We have recently reported on the luminal changes of BOD and non-BOD types of ICAS.\textsuperscript{6} Our previous analysis of stenosis morphology revealed that stenotic lesions in BOD are rather diffuse, although they show mild degrees of stenosis, suggesting different pathophysiologic mechanisms of stenosis between BOD and non-BOD. In addition, the atherosclerotic burden was similar between BOD and non-BOD, suggesting that BOD is not a mild form of ICAS.\textsuperscript{6}

Patients with BOD often exhibit a milder degree of stenosis with small, deep infarcts, rarely showing demonstrable perfusion abnormalities or microembolic signals on transcranial Doppler. Stenting may be harmful for these patients because perforator strokes can occur after stenting.\textsuperscript{23–25} Therefore, patients are often misdiagnosed as having small arterial occlusions, and so physicians may treat these patients like those with small arterial occlusions. Our present data regarding the spatial relationship between plaque burden and the enhancing area with the origin of perforators in patients with BOD suggest that the treatment strategies for BOD may be similar to those for non-BOD. Early intervention with a high-dose statin may decrease plaques in patients with ICAS. Decreasing plaques with statins may be as important in patients with BOD as in patients with non-BOD because even small changes in plaque size located adjacent to the perforator ostia may deteriorate microvascular circulation; the hospital course and recurrence rate of BOD is reportedly not benign.\textsuperscript{5,26} Our serial follow-up HR-MRI study in patients with ICAS is currently ongoing to test whether plaque-stabilizing therapies reduce the plaque burden and size in patients with BOD, as well as in those with non-BOD (clinicaltrials.gov Identifier NCT02458755). Strategies for reducing positive

![Figure 2.](http://stroke.ahajournals.org/)

**Figure 2.** The location of plaque enhancement in branch occlusive disease (BOD; A) and non-BOD (B). MCA indicates middle cerebral artery.
remodeling, as well as reducing plaque activity and size, may be particularly important in patients with non-BOD.

Limitations and Conclusions
This study has several limitations. First, although all patients underwent comprehensive workups, including vascular, laboratory, and cardiological studies, the reader should interpret this study’s results with caution, given its cross-sectional nature and the limited sample size. Further studies with a long-term follow-up of a large cohort are needed. Second, a signal gap or even a narrowing in MRA may represent only a partially recanalized clot. To overcome this problem, we performed electrocardiography and echocardiography for all patients according to the current guidelines for cardiac monitoring, and we excluded those with proximal embolic sources in the present study. In addition, serial electrocardiography was performed during follow-up. However, higher yield of atrial fibrillation detection with in-hospital and outpatient telemonitoring, and more recently implantable loop recorder, has been reported, and there is still possibility of the presence of paroxysmal atrial fibrillation in our patients. Third, beside atherosclerotic plaque, other pathology may cause intracranial stenosis. One HR-MRI study of young adult patients with unilateral MCA disease and minimal atherosclerotic risk factors showed that various pathologies, such as dissection, Moyamoya disease, and vasculitis, are associated with intracranial stenosis. However, HR-MRI can be a complementary tool for intracranial vasculopathy differentiation, and we additionally excluded these pathologies using HR-MRI. Fourth, the wall enhancement observed in our patients may have been because of luminal (and not wall) enhancement coming from slow-moving blood adjacent to the vessel wall (pseudoenhancing slow flow artifact). Additional studies using true double-inversion recovery sequences are needed. However, in patients with ICAS, enhancement was often observed on the nonstenosed segment, either on the asymptomatic side or on the nonstenosed side, on time-of-flight MRA. In contrast, enhancement was rarely observed in the asymptomatic stenosed segment in patients with ICAD. Finally, in the present study, all analysis was performed at a single level, the location of the most severe stenosis. 3D volumetric analysis has been applied in a previous study assessing carotid plaque.

In conclusion, our HR vessel wall imaging data indicate that BOD- and non-BOD-type ICAS have different vascular pathophysiologies in terms of vascular remodeling and plaque characteristics. Further studies are needed, with a large cohort in different racial/ethnic groups, to generalize our results.

Sources of Funding
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Disclosures
None.

References
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**Supplemental Material**

Supplemental Table I. Inter-rater reliability for measuring HRMR parameters

<table>
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<th>Parameter</th>
<th>ICC</th>
<th>95% CI</th>
<th>P</th>
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<tr>
<td>VA</td>
<td>0.974</td>
<td>(0.924 – 0.991)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LA</td>
<td>0.978</td>
<td>(0.934 – 0.993)</td>
<td>&lt;0.001</td>
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<tr>
<td>WA</td>
<td>0.932</td>
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<td>&lt;0.001</td>
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<tr>
<td>RI</td>
<td>0.912</td>
<td>(0.725 – 0.972)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Stenosis</td>
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<td>(0.948 – 0.995)</td>
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<tr>
<td>EA</td>
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<td>EA (%)</td>
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Supplemental Table II. Intra-rater reliability for measuring HRMR parameters

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<th>Parameter</th>
<th>ICC</th>
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<th>P</th>
</tr>
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<tbody>
<tr>
<td>VA</td>
<td>0.978</td>
<td>(0.936 - 0.993)</td>
<td>&lt;0.001</td>
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<tr>
<td>LA</td>
<td>0.985</td>
<td>(0.954 - 0.995)</td>
<td>&lt;0.001</td>
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<td>WA</td>
<td>0.956</td>
<td>(0.870 - 0.985)</td>
<td>&lt;0.001</td>
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<td>WA Index</td>
<td>0.862</td>
<td>(0.588 - 0.954)</td>
<td>&lt;0.001</td>
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<td>RI</td>
<td>0.909</td>
<td>(0.730 - 0.970)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Stenosis</td>
<td>0.933</td>
<td>(0.801 - 0.978)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>EA</td>
<td>0.916</td>
<td>(0.973 - 0.997)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>EA (%)</td>
<td>0.859</td>
<td>(0.581 - 0.953)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
Abstract

Differential Vascular Pathophysiologic Types of Intracranial Atherosclerotic Stroke
A High-Resolution Wall Magnetic Resonance Imaging Study

Sookyung Ryoo, MD; Mi Ji Lee, MD; Jihoon Cha, MD, et al.

Departments of Neurology; and Radiology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, South Korea.

Background and Objective: In intracranial atherosclerotic stroke (ICAS), the lesions vary depending on the vascular territory. In this study, we assessed the vascular territories with high-resolution wall magnetic resonance imaging (HR-WMRI) in patients with ICAS.

Methods: A total of 50 patients with ICAS were enrolled. Of them, 21 patients were enrolled in the HR-WMRI study. A radiologist identified the vascular territory of the ICAS lesion in each patient, and the cases were classified into four vascular territories: anterior cerebral, middle cerebral, anterior communicating, and posterior circulation territories.

Results: The HR-WMRI study identified the vascular territories of the ICAS lesions in 20 patients (95%). The frequency of vascular territories was as follows: anterior cerebral (10 patients, 48%), middle cerebral (9 patients, 43%), anterior communicating (1 patient, 5%), and posterior circulation (5 patients, 24%).

Conclusion: HR-WMRI is a useful tool for identifying the vascular territories of ICAS lesions.
두개강내벽경화에 의한 뇌졸중의 다양한 혈관 병태생리학적 원인
고 혈압도 혈관벽 자기공명영상 연구

Differential Vascular Pathophysiologic Types of Intracranial Atherosclerotic Stroke
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(Stroke. 2015;46:2815-2821.)

Key Words: atherosclerosis, high-resolution wall MRI, intracranial stenosis, ischemic stroke, magnetic resonance imaging

배경과 목적
두개강내벽경화에 의한 뇌졸중은 가지폐색질환(branch occlusive disease, BOD: 관통동맥의 입구 폐색을 일으키는 모둥백질환에 의한 폐질환)과 non-BOD(동맥간색전에 의해 유 발되는 폐질환의 외 경색)을 포함하여 다양한 뇌졸중 기전을 가진다. 이러한 두 종류의 뇌경색이 서로 다른 혈관 병태생리학적 특성을 가진다. 또한 두 종류의 뇌경색이 서로 다른 혈관 병태생리학적 특성을 가진다면, 이를 근거로 하여 우리는 BOD와 non-BOD 사이의 고혈압도 자기공명영상의 특성을 비교하였다.

방법
경동맥 및 심장내력을 완전과 비복합경화성 완전을 가진 경우는 제외한 두개강내벽(관통 viscous 혈관 또는 가보동맥)적경화증에 의한 뇌졸중을 가진 80명의 급성뇌경색 환자들이 모집되었다. 이 중 BOD는 36명, non-BOD는 44명이었다. 가장 심한 혈착을 보인 위치에서 혈관재형성과 혈관벽조영중증 여부를 평가하였다.

결과
BOD는 혈관재형성과 조영증차 관련해서 non-BOD와 다른 영상학적 특성을 보였다. BOD는 non-BOD에 비해서 경미한 혈착 정도(P<0,001)를 보였고, 드문 양성 혈관재형성 반도를 보였으며, 더 낮은 혈관벽 영역지수를 보였다. 혈관경화관조영중량은 non-BOD에서는 한 명을 제외하고 모든 종류에서 관찰되었는데 비해, BOD에서는 25%에서만 관찰되었다(P=0,003). 두 종류 모두 혈관벽에 전반적인 조영중량을 보였지만, BOD군에서는 관통동맥이 기각하는 반에 조영중량이 더 흔하게 관찰되었다. 두종군 두개강내 혈착 혈관의 수가 증가함에 따라서 BOD군에서는 혈착의 정도가 더 증가(rho=0,513, P=0,003)하였고, non-BOD군에서는 조영중량의 증가(rho=0,343, P=0,030)하였다.

결론
본 연구에서 BOD는 매우 흔하고, non-BOD와는 다른 특징을 가진다. 두 종류의 뇌졸중 기전은 혈관재형성 및 조영증차 특징관련하여 서로 다른 혈관 병태생리학적 특성을 가진다.
Intracranial atherosclerotic stroke (ICAS; branch occlusive disease [BOD] vs non-BOD) according to asymptomatic intracranial atherosclerotic disease (ICAD) burden. A, Types of ICAS depending on the ICAD score. B, High-resolution magnetic resonance imaging (HR-MRI) characteristics of ICAS depending on the ICAD score.

Table 2. Comparison of HR-MRI Findings Between the BOD and Non-BOD Groups

<table>
<thead>
<tr>
<th>Vascular morphology</th>
<th>BOD (n=35, 45.5%)</th>
<th>Non-BOD (n=42, 45.5%)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stenosis degree, %</td>
<td>40.7±27.4</td>
<td>74.1±20.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Wall area index</td>
<td>2.82 (1.67–4.61)</td>
<td>6.39 (3.88–8.50)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Remodeling index (Ri)</td>
<td>1.09±0.44</td>
<td>1.25±0.43</td>
<td>0.039</td>
</tr>
<tr>
<td>Remodeling pattern, n (%)</td>
<td>0.016</td>
<td>0.016</td>
<td></td>
</tr>
<tr>
<td>Negative (Ri&lt;0.95)</td>
<td>10 (31.3)</td>
<td>10 (27.8)</td>
<td></td>
</tr>
<tr>
<td>No change (0.95≤Ri&lt;1.2)</td>
<td>15 (46.9)</td>
<td>7 (19.4)</td>
<td></td>
</tr>
<tr>
<td>Positive (Ri≥1.2)</td>
<td>7 (21.9)</td>
<td>19 (52.8)</td>
<td></td>
</tr>
<tr>
<td>Vascular activation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enhancement in symptomatic vessel, n (%)</td>
<td>26 (74.3)</td>
<td>41 (97.6)</td>
<td>0.004</td>
</tr>
<tr>
<td>Enhancement pattern, n (%)</td>
<td></td>
<td>≥0.999</td>
<td></td>
</tr>
<tr>
<td>Concentric</td>
<td>2 (7.7)</td>
<td>4 (9.8)</td>
<td></td>
</tr>
<tr>
<td>Eccentric</td>
<td>24 (69.4)</td>
<td>37 (92.2)</td>
<td></td>
</tr>
<tr>
<td>Enhancement amount</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enhanced area, mm²</td>
<td>1.34 (0–3.20)</td>
<td>5.15±3.05</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Enhancement degree, %</td>
<td>40.5 (0–64.7)</td>
<td>63.0±24.6</td>
<td>0.001</td>
</tr>
<tr>
<td>Asymptomatic ICAD score</td>
<td>6 (3–8)</td>
<td>6 (4–9)</td>
<td>0.617</td>
</tr>
</tbody>
</table>

Values are presented as mean±SD or median (interquartile range), unless otherwise specified. BOD indicates branch occlusive disease; HR-MRI, high-resolution magnetic resonance imaging; and ICAD, intracranial atherosclerotic disease.