Clinical Significance of Fluid-Attenuated Inversion Recovery Vascular Hyperintensities in Borderzone Infarcts

Si Eun Kim, MD; Byung In Lee, MD; Sung Eun Kim, MD; Kyong Jin Shin, MD; JinSe Park, MD; Kang Min Park, MD; Hyung Chan Kim, MD; Joonwon Lee, MD; Hye Jin Baek, MD; Sung-cheol Jin, MD; Sam Yeol Ha, MD

Background and Purpose—Fluid-attenuated inversion recovery vascular hyperintensities (FVHs) are seen in some cases with cerebral hemodynamic impairment and collateral flow. Because the worst outcomes of patients with borderzone infarcts were mainly correlated with impaired hemodynamics, the presence of FVH might provide another clue for predicting the prognosis of patients with borderzone infarcts.

Methods—We reviewed 1377 consecutive patients with ischemic stroke. Cortical borderzone (CBZ) and internal borderzone infarcts were selected based on diffusion-weighted imaging. FVHs were defined as tubular- or serpentine-shaped hyperintensities in the subarachnoid space. We investigated the clinical significance of FVHs in borderzone-infarcted patients.

Results—Among 87 patients with borderzone infarcts, the presence of FVH was observed in 30 (34.5%). We identified 62 patients with CBZ infarcts and 25 patients with internal borderzone infarcts. In the cases with CBZ infarcts, the initial National Institutes of Health Stroke Scale scores and the portions of nonfavorable outcome at 3 months in the FVH(+) group were significantly higher than in the FVH(−) group (P<0.05 and P<0.001, respectively). Unlike the cases with CBZ infarcts, there were no significant differences of these clinical features between the FVH(+) group and the FVH(−) group in the patients with internal borderzone infarcts.

Conclusions—The findings of FVH are associated with relatively severe clinical presentation and nonfavorable prognosis in patients with CBZ infarcts, but not in patients with internal borderzone infarcts. The presence of FVH may help to identify CBZ-infarcted patients who require close observation and hemodynamic control. (Stroke. 2016;47:1548-1554. DOI: 10.1161/STROKEAHA.115.012285.)

Key Words: cortical ▾ diffusion magnetic resonance imaging ▾ hemodynamics ▾ infarction ▾ internal ▾ prognosis

Borderzone infarcts, or watershed infarcts, reportedly account for ≈10% of all brain infarcts.1 Their general prognosis is good.2,3 However, certain portions of borderzone infarcts have poor outcomes, depending on the type of borderzone and deterioration during the hospital stay. Patients with internal borderzone (IBZ) infarcts may show worse hospital stays, and such patients might remain disabled at 3 months after stroke onset. In contrast, cortical borderzone (CBZ) infarcts have a relatively benign clinical course.4 Few studies have predicted the prognosis of patients with borderzone infarcts. Differences in the prognosis of different types of borderzone might be related to differences in the pathogenesis of hemodynamic compromise and emboli.4,6

Vascular hyperintensities have been noted in fluid-attenuated inversion recovery (FLAIR) sequences and represent impaired hemodynamics and collateral flows in the setting of acute stroke and intracranial steno-occlusive disease.7-10 However, FLAIR vascular hyperintensities (FVHs) are seen in some, but not in all, cases with hemodynamic dysfunction and collateral flows.7,9 Considering these characteristics of FVHs, the presence of FVH might provide another clue for predicting the prognosis of patients with borderzone infarcts. Here, we investigated the patterns and clinical significance of FVHs in borderzone-infarcted patients.

Materials and Methods

Patients registered in the Haeundae Paik Stroke Registry between March 2010 and June 2014 who had an acute focal neurological deficit were included in the present study. The inclusion criteria were defined as the following: (1) patients who visited the hospital within
7 days of symptom onset; (2) patients with responsible lesions within the middle cerebral artery (MCA) distribution territory on diffusion-weighted imaging (DWI), who were initially evaluated using magnetic resonance imaging, including FLAIR, gradient echo, and DWI; (3) patients who underwent computed tomographic angiography, MR angiography, or conventional angiography for the evaluation of vessel status; and (4) patients with a complete evaluation, which included a personal history given by the patient or a family member, vascular risk factors, routine blood tests, and cardiological work-up that included an ECG, an echocardiogram, and 24-hour Holter monitoring.

Two types of borderzone infarcts were considered based on the matched borderzone area on DWI. CBZ infarcts were defined as the hyperintense area, the border between the MCA and the anterior cerebral artery, or the MCA and posterior cerebral artery. IBZ infarcts were classified as having lesions if they were located between the deep and the superficial perforating arterial territories of the MCA. If there were other lesions in the contralateral hemisphere in addition to the borderzone infarct, we excluded those cases. Two authors (S.Y.H and S.E.K) who were blind to the patient information reviewed and classified the DWI data retrospectively.

FVHs were defined using the following criteria: (1: shape) tubular- or serpentine-shaped hyperintensities; (2: location) in the subarachnoid space corresponding to distal M1 or M2 segments within the Sylvian fissure (proximal location) or distal to the Sylvian fissure corresponding to M3 and M4 segments of the MCA (distal location); and (3: number) ≥2 numbers. Cases with no FLAIR images or FLAIR artifacts were excluded. Two authors who classified the DWI data also evaluated the FLAIR sequences. After analyzing the type of borderzone, we evaluated the presence and location of the FVH. Disagreements were adjudicated by consensus.

We analyzed the basic characteristics and clinical outcome for all patients with borderzone infarcts, dividing groups by the presence or the absence of FVH. Afterward, the same analyses were applied to the CBZ and IBZ infarct group. We also analyzed the radiological data of DWI lesion volume, perfusion abnormality on perfusion-weighted imaging, detailed vascular status of the relevant arteries, and collateral status using the Christoforidis’ collateral score. For measuring DWI lesion volumes, we used patterns of borderzone lesions (scattered/wedged pattern in CBZ infarcts; rosary/confluent pattern in IBZ infarcts) and the number of large lesions >1 cm because we could not acquire the volume of many tiny lesions of the borderzone area using an automated or manual method (Figure 1, illustrative cases of borderzone infarcts). Depending on the nature of the variables, differences between the groups were investigated using a Mann–Whitney U test, Student t test, Pearson χ² test, or a Fisher exact test. Statistical significance was established at P<0.05. Multivariate logistic regression analysis was applied to identify independent predictors of nonfavorable outcome of the modified Rankin Scale (mRS ≥2) at 3 months after stroke onset. Independent variables were age, initial National Institutes of Health Stroke Scale (NIHSS) score, presence of FVH [FVH(+)], treatment of intra-arterial thrombolysis or stent, progression after admission, and vessel status of severe stenosis (>70%) or occlusion. The results are presented as an odds ratios as an assumption of the relative risk with a 95% confidence interval. Institutional review board approval was obtained for this retrospective analysis.

Results

We identified 91 patients with borderzone infarcts of 1377 consecutive ischemic stroke patients (6.6%). We excluded 4 patients with unreadable FLAIR images because of motion artifacts. Among the remaining 87 patients, the presence of FVH was observed in 30 patients (34.5%). Details of the clinical and demographic characteristics of patients with borderzone infarcts according to presence of FVH are presented in Table 1. Demographic data, such as sex, age, and risk factors of hypertension, diabetes mellitus, smoking, hyperlipidemia, atrial fibrillation, previous stroke, or transient ischemic attack history, as well as blood pressure and glucose on arrival between the 2 groups, were not significantly different. The clinical presentation at admission and the seventh day afterstroke. We performed Pearson χ² test, Fisher exact test, or multivariate logistic regression analysis for all the variables. Differences between the groups were investigated using a Mann–Whitney U test, Student t test, Pearson χ² test, or a Fisher exact test. Statistical significance was established at P<0.05. Multivariate logistic regression analysis was applied to identify independent predictors of nonfavorable outcome of the modified Rankin Scale (mRS ≥2) at 3 months after stroke onset. Independent variables were age, initial National Institutes of Health Stroke Scale (NIHSS) score, presence of FVH [FVH(+)], treatment of intra-arterial thrombolysis or stent, progression after admission, and vessel status of severe stenosis (>70%) or occlusion. The results are presented as an odds ratios as an assumption of the relative risk with a 95% confidence interval. Institutional review board approval was obtained for this retrospective analysis.

Figure 1. Illustrative cases according to the type of borderzone (BZ) and pattern of lesions on diffusion-weighted imaging (DWI). Cortical BZ (CBZ) case A in A shows wedged-shape lesion on DWI, perfusion delay of only BZ area on time to peak (TTP) map, moderate stenosis of right cavernous internal carotid artery (ICA; arrow) without fluid-attenuated inversion recovery (FLAIR) vascular hyperintensities (FVH). In contrast, CBZ case B shows scattered lesion on DWI, perfusion delay in middle cerebral artery (MCA) territory with reduced cerebral blood flow (CBF) map, occlusion of right ICA, and distal location of FVH (within circle). Internal BZ (IBZ) case A in B reveals rosary-pattern lesions on DWI, perfusion delay in MCA territory with preserved CBF, severe stenosis of left distal ICA without FVH. IBZ case B shows confluent-pattern lesion on DWI with occlusion of right MCA M1, and proximal location of FVH within Sylvian fissure. Perfusion computed tomographic color maps show perfusion abnormalities in right hemisphere, characterized by mostly reduced CBF (color-coded blue) and mostly increased TTP (color-coded red) in lesion on DWI. CTA indicates computed tomography angiography; and MRA, magnetic resonance angiography.
admission were different between the 2 groups. The distinction of the initial clinical severity between the 2 groups was still observed in prognosis 3 months later. Compared with the FVH(−) group at 3 months after admission, the FVH(+) group that had presented with more severe symptoms initially showed a higher MRS ($P=0.001$) and more patients with a nonfavorable outcome ($mRS \geq 2; P<0.001$) and poor outcome ($mRS \geq 3; P=0.005$).

However, the characteristics of the patients with borderzone infarcts according to FVH varied when differentiating this borderzone group based on the type of borderzone (Table 2). Clinical presentation at admission and prognosis at 3 months according to existence of FVH were distinct between the patients with CBZ infarcts ($n=62; 71.3\%$) and IBZ infarcts ($n=25; 28.7\%$). In the cases with CBZ infarcts, the initial and seventh day after admission NIHSS scores of the FVH(+) group were significantly higher ($P<0.05$) than those of the FVH(−) group. This FVH(+) group had more patients with a nonfavorable outcome ($mRS \geq 2; P<0.001$) and a poor outcome ($mRS \geq 3; P=0.01$) at 3 months after admission than the FVH(−) group. The FVH(+) group had more patients with severe stenosis ($>70\%$) or occlusion of the relevant artery and perfusion delay on anterior cerebral artery or MCA territory than the FVH(−) group ($P<0.001$). However, the semiquantitative DWI lesion volumes were not different between the 2 groups.

Unlike the cases with CBZ infarcts, there were no significant differences in these clinical and radiological features between the FVH(+) group and the FVH(−) group in the patients with IBZ infarcts. In other words, the FVH(+) group with IBZ infarcts did not show a significantly worse clinical presentation on the day admitted or 3 months later compared with the
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FVH(−) group with the same type of infarct. Details of the clinical and radiological characteristics of the patients with borderzone infarcts according to the type of borderzone and presence of FVH are presented in Tables 2 and 3 and Figure 2.

Univariate analysis of the clinical and radiological factors associated with nonfavorable outcome (mRS at 3 months ≥2) according to the type of borderzone is reported in Table I in the online-only Data Supplement. After adjusting for confounding factors associated with prognosis at 3 months, multivariate logistic regression analysis showed that the initial NIHSS (odds ratio, 2.59; 95% confidence interval, 1.45–4.63; \(P=0.001\)) and the FVH(+) (odds ratio, 9.98; 95% confidence interval, 1.55–64.03; \(P=0.015\)) were independently associated with a nonfavorable outcome (mRS at 3 months ≥2) in the patients with CBZ infarcts. On the contrary, only the factor of progression after admission (odds ratio, 17.6; 95% confidence interval, 1.7–181.29; \(P=0.016\)) was independently associated with a nonfavorable outcome in patients with IBZ infarcts; the other factors, such as FVH(+), initial NIHSS, and severe stenosis or occlusion, did not show significance.

Discussion

In this study, the prognostic value of FVHs was different depending on the type of borderzone infarct. This discrepancy could be because of the difference in the pathogenesis of these 2 distinct borderzone types. Although the pathophysiology of ischemic stroke in the borderzone area has been debated, the current established idea suggests an interaction between hemodynamic disturbance and microembolization.\(^{10,11}\) The interaction between 2 mechanisms dissimilarly influences each other according to the type of the borderzone infarct. However, even within the same CBZ-infarcted type, the interaction between the 2 pathogenic mechanisms might manifest differently. The characteristics of FVH representing impaired hemodynamics suggests that the group of FVH(+) patients with CBZ infarcts could be much more influenced by hemodynamic instability than FVH(−) patients with the same type of infarct. This difference might be correlated with a nonfavorable outcome in the FVH(+) group of CBZ-infarcted patients in this study. This is similar to the finding that IBZ-infarcted patients showed a worse outcome than patients with CBZ infarcts, because of

Table 2. Clinical Characteristics for Patients With BZ Infarcts According to the Type of BZ and Presence of FVH

<table>
<thead>
<tr>
<th>CBZ Infarcts (n=62)</th>
<th></th>
<th>IBZ Infarcts (n=25)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>FVH (−) n=45</td>
<td>FVH (+) n=17</td>
<td>PValue</td>
<td>FVH (−) n=12</td>
</tr>
<tr>
<td>Initial NIHSS score (median, range)</td>
<td>2 (0–9)</td>
<td>4 (0–11)</td>
<td>0.04*</td>
</tr>
<tr>
<td>Seventh day NIHSS score (median, range)</td>
<td>1 (0–7)</td>
<td>3 (0–10)</td>
<td>0.03*</td>
</tr>
<tr>
<td>In hospital at seventh day, n (%)</td>
<td>30 (66.7)</td>
<td>16 (94.1)</td>
<td>0.047†</td>
</tr>
<tr>
<td>Hospitalization period, d (median, range)</td>
<td>7 (2–50)</td>
<td>16 (3–53)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Previous TIA, n (%)</td>
<td>18 (40.0)</td>
<td>6 (35.3)</td>
<td>NS</td>
</tr>
<tr>
<td>Progression after admission, n (%)</td>
<td>2 (4.4)</td>
<td>5 (29.4)</td>
<td>0.013‡</td>
</tr>
</tbody>
</table>

Treatment

- tPA injection, n (%) | 2 (4.4) | 1 (5.9) | NS | 0 | 1 (7.7) | NS |
- Intervention treatment, n (%) | 10 (22.2) | 9 (52.9) | 0.03‡ | 3 (25) | 4 (30.8) | NS |
- IA thrombolysis, n§/n | 2/2 | 2/2 | 0 | 2/3 |
- Emergent or elective stent insertion, n | 8 | 1 | 3 | 0 |
- EC-IC bypass operation, n | 0 | 6 | 0 | 1 |
- Severe stenosis (>70%) or occlusion, n (%) | 14 (31.1) | 16 (94.1) | <0.001† | 9 (75) | 13 (100) | NS |

Prognosis at 3 mo

- modified Rankin Scale, (median, range) | 1.0 (0–3) | 2.0 (0–4) | 0.003* | 2.5 (0–4) | 3.0 (0–5) | NS |
- Poor outcome (≥3), n (%) | 4 (8.9) | 6 (35.3) | 0.01‡ | 6 (50) | 8 (61.5) | NS |
- Nonfavorable outcome (≥2), n (%) | 10 (22.2) | 12 (70.6) | <0.001† | 8 (66.7) | 8 (61.5) | NS |
- Symptoms to MRI time, h, (median, range) | 17 (1–89.3) | 16.5 (1–61) | NS | 28.5 (6–134) | 25.5 (2–107) | NS |

FVH(−), CBZ, EC-IC, FVH, IA, IBZ, MRI, NIHSS, NS, TIA, tPA, TICI.

\(^*\)Mann–Whitney U test.

\(^†\)Pearson \(\chi^2\) test.

\(^‡\)Fisher exact test.

\(§\)Recanalized cases of TICI ≥2b.

CBZ indicates cortical borderzone; EC-IC, extracranial–intracranial; FVH, fluid-attenuated inversion recovery vascular hyperintensities; IA, intra-arterial; IBZ, internal borderzone; MRI, magnetic resonance imaging; NIHSS, National Institutes of Health Stroke Scale; NS, nonsignificant; TIA, transient ischemic attack; TICI, Thrombolysis in Cerebral Infarction; and tPA, tissue-type plasminogen activator.

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the dissimilar influence of hemodynamic instability between the 2 types of borderzone infarcts." Although the main pathogenesis of CBZ infarcts seems to be microembolization, an additional hemodynamic-compromised insult besides emboli could affect the FVH(+) group in CBZ-infarcted patients.

In our study, this assumption was supported by the 3 points of our results. The first point concerns the difference in vascular status between the 2 groups of CBZ-infarcted patients. The FVH(+) cases with CBZ infarcts had a higher portion of patients with severe stenosis (>70%) or occlusion than the FVH(−) patients with the same type of infarct (31.1% versus 94.1%; \( P < 0.001 \); Table 2; Figure 2). This gap of vascular status might influence the hemodynamic status dissimilarly to both groups. The second point is that there is no difference in DWI lesion volume in our results. Although we could not acquire the absolute volume of the lesions, the pattern or number of relatively large lesions did not show a distinction between the FVH(−) and FVH(+) groups of patients with CBZ infarcts. This suggests that lesions in DWI might have little effect on the different prognoses shown between the 2 groups. The last point regards the difference in perfusion findings. A substantial number of patients in our study did not receive a perfusion computed tomographic or magnetic resonance imaging. Although we consider this a limitation, the difference of perfusion status between the 2 groups seems to be obvious. When we analyzed valid perfusion images, all patients in the FVH(+) group with CBZ infarcts showed a perfusion delay in the MCA or anterior cerebral artery territory, including the borderzone area, unlike the FVH(−) group (Table 3). The difference in the hemodynamic condition shown in these points might be related to the relatively high initial NIHSS (2.0 versus 4.0; \( P = 0.04 \)) and nonfavorable outcome (22.2% versus 70.6%; \( P < 0.001 \)) in the FVH(+) group of patients with CBZ infarcts, compared with the FVH(−) group of patients with CBZ infarcts.

However, the prognostic role of FVH that was observed in patients with CBZ infarcts was not observed in patients with IBZ infarcts. Only the factor of progression after admission independently influenced the prognosis in a logistic regression. Our study has 2 limitations for analysis of this prognosis in IBZ-infarcted patients. One is the small sample size of these type of infarcts; the other is the absence of perfusion data in many cases. However, none of the clinical situations,

Figure 2. Detailed vascular status of the relevant arteries according to the type of borderzone (BZ) and presence of fluid-attenuated inversion recovery vascular hyperintensities (FVH). ACA indicates anterior cerebral artery; CBZ, cortical BZ; IBZ, internal BZ; ICA, internal carotid artery; and MCA, middle cerebral artery.
such as the initial and seventh day NIHSS score, hospitalization period, progression after admission, and poor or nonfavorable outcome at 3 months, were different between the FVH(−) and FVH(+) groups of IBZ-infarcted patients (Table 2). This suggests that the insignificance of FVH on the prognosis of IBZ-infarcted patients might last even in more cases with IBZ infarcts. A previous report suggested that a compromised hemodynamic state seemed to correlate with a worsening of the clinical course caused by infarct growth in IBZ-infarcted patients.13 Because most cases of IBZ-infarcted patients had a severe stenotic or occluded vessel status and territorial delay on time to peak perfusion map, we need to observe perfusion status in more detail, namely the cerebral blood flow or cerebral blood volume perfusion map and the pattern or location of the worst misery perfusion, rather than FVH. A larger sample size and thoughtful analysis of perfusion data could aid in identifying factors influencing the progression and prognosis of IBZ-infarcted patients.

The presence of FVH has been associated with a different prognostic value according to the location of the FVH in previous reports.12 In particular, Lee et al14 showed that the presence of distal FVH was correlated with a better outcome. However, this subdivision in to proximal and distal FVH did not show significance when it came to the prognosis of borderzone-infarcted patients in our study (Table II in the online-only Data Supplement). In case of the FVH(+) group with IBZ infarcts, all cases showed proximal location of FVH. The location of FVH seems to be insignificant with regard to the prognosis of borderzone-infarcted patients, especially in CBZ infarcts.

Our study has several limitations. First, the prognosis may be correlated with differences caused by treatment. The cases treated by interventions such as intra-arterial thrombolysis, stent insertion, or bypass operation were significantly higher in the FVH(+) group of CBZ-infarcted patients; however, the ratio of the treatment among only the cases with severe stenotic or occluded vessels between 2 groups was not different. In addition, the recanalized status in cases treated by intra-arterial thrombolysis was good in both groups. Although there are differences in medical treatment, including induced hypertension, we think that the factor of treatment is not associated with the different prognoses between the FVH(−) and FVH(+) groups of CBZ-infarcted patients. Second, our study is a retrospective observational study. We could not rule out the possibility of bias arising from being limited to a chart review approach. Finally, as mentioned before, the sample size of the IBZ infarcts was small when compared with that of the CBZ infarcts. A study with more cases of IBZ infarcts might reveal the significance of FVH in IBZ-infarcted patients more clearly.
Conclusions
This study suggests that the presence of FVH is associated with a relatively severe clinical presentation and a nonfavorable prognosis in patients with CBZ infarcts, but not in patients with IBZ infarcts. The presence of FVH may help to identify CBZ-infarcted patients who require close observation and hemodynamic control.

Disclosures
None.

References
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Cover Title: FLAIR hyperintensities in borderzone infarcts

Key Words: FLAIR, borderzone, infarct

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Table I. Univariate analysis of the clinical and radiological factors associated with non-favorable outcome (MRS at 3 months ≥2) according to the type of BZ.

Table II. Clinical characteristics for patients with CBZ infarcts according to the location of FVH

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E-mail : H00251@paik.ac.kr
Table I. Univariate analysis of the clinical and radiological factors associated with non-favorable outcome (MRS at 3 months ≥2) according to the type of BZ.

<table>
<thead>
<tr>
<th></th>
<th>CBZ infarcts (N = 62)</th>
<th>IBZ infarcts (N=25)</th>
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<tr>
<td></td>
<td>Favorable outcome (MRS&lt;2) (N=40)</td>
<td>Non-favorable outcome (MRS≥2) (N=22)</td>
<td>p</td>
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<tr>
<td>Age, y (median, range)</td>
<td>68 (48-85)</td>
<td>74 (52-88)</td>
<td>0.09</td>
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<td>Initial NIHSS score (median, range)</td>
<td>1.0 (0-7)</td>
<td>4.5 (1-11)</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>Progression after admission, n(%)</td>
<td>2 (5.0%)</td>
<td>5 (22.7%)</td>
<td>0.09</td>
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<td>Treatment of intervention, n(%) (IA, stent insertion or bypass)</td>
<td>10 (25.0%)</td>
<td>8 (36.4%)</td>
<td>0.389</td>
</tr>
<tr>
<td>FVH(+), n(%)</td>
<td>5 (12.5%)</td>
<td>12 (54.5%)</td>
<td>&lt;0.01†</td>
</tr>
<tr>
<td>Severe stenosis (&gt;70%) or occlusion n(%)</td>
<td>15 (37.5%)</td>
<td>14 (63.6%)</td>
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</table>

*Mann–Whitney U test; †Fisher’s exact test
Table II. Clinical characteristics for patients with CBZ infarcts according to the location of FVH

<table>
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<th>FVH (+) group of CBZ infarcts (N=17)</th>
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<td></td>
<td>Proximal FVH (+) N=10</td>
<td>Distal FVH (+) N=7</td>
<td>p</td>
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<td>Initial NIHSS score (median, range)</td>
<td>3 (0-11)</td>
<td>5 (1-10)</td>
<td>NS</td>
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<td>7th day NIHSS score (median, range)</td>
<td>4 (0-10)</td>
<td>1 (0-6)</td>
<td>NS</td>
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<td>In hospital at 7th day, n(%)</td>
<td>10 (100%)</td>
<td>6 (85.7%)</td>
<td>NS</td>
</tr>
<tr>
<td>Hospitalization period, day (median, range)</td>
<td>16 (9-53)</td>
<td>12 (3-43)</td>
<td>NS</td>
</tr>
<tr>
<td>Progression after admission, n(%)</td>
<td>4 (40.0%)</td>
<td>1 (14.3%)</td>
<td>NS</td>
</tr>
<tr>
<td>Intervention treatment such as IA thrombolysis, stent insertion or EC-IC bypass operation, n(%)</td>
<td>8 (80.0%)</td>
<td>1 (14.3%)</td>
<td>0.02*</td>
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<td>Prognosis at 3 months</td>
<td></td>
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<td>Poor outcome (≥3), n(%)</td>
<td>4 (40.0%)</td>
<td>2 (28.6%)</td>
<td>NS</td>
</tr>
<tr>
<td>Non-favorable outcome (≥2), n(%)</td>
<td>7 (70.0%)</td>
<td>5 (71.4%)</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Fisher’s exact test; NS : Non-Significant
Clinical Significance of Fluid-Attenuated Inversion Recovery Vascular Hyperintensities in Borderzone Infarcts

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