Objective: We examined the usefulness of Doppler ultrasonography for the diagnosis of severe stenosis of the proximal vertebral artery (VA).

Case Presentations: We performed Doppler ultrasonography of the VA in patients diagnosed with cerebral ischemia of the posterior circulation. Incorporating the diagnostic criteria for severe stenosis at the origin of the internal carotid artery (maximum peak systolic flow velocity: ≥200 cm/sec, or acceleration time: ≥110 msec), patients were screened for proximal VA stenosis and cerebral angiography was conducted if they fulfilled the above criteria. In all six patients in whom proximal VA stenosis was suspected on ultrasonography, angiography confirmed severe stenosis, and endovascular treatment was performed. In five patients who underwent postoperative ultrasonography, an improvement of the stenosis was confirmed.

Conclusion: Doppler ultrasonography is useful for the screening and postoperative assessment of proximal VA stenosis.

Keywords: vertebral artery, stenting, balloon dilatation, Doppler ultrasonography, carotid artery ultrasonography

Introduction

Diagnosis of severe stenosis at the origin of the internal carotid artery using Doppler ultrasonography has been established. Previous studies demonstrated the efficacy of carotid endarterectomy or stenting in patients with severe carotid artery stenosis. On the other hand, concerning stenosis of the proximal vertebral artery (VA), which may cause cerebral ischemia involving the posterior circulation, a simple screening method has not yet been established, and the efficacy of endovascular treatment procedures at this site, such as stenting and balloon angioplasty, has not been verified. We performed Doppler ultrasonography for the screening of proximal VA stenosis with the diagnostic criteria used for the origin of the internal carotid artery, and VA stenoses suspected on ultrasonography were confirmed by cerebral angiography. In addition, we utilized Doppler ultrasonography for postoperative follow-up after endovascular treatment of proximal VA stenosis. Here, we report the usefulness of Doppler ultrasonography for the diagnosis of proximal VA stenosis in our series.

Case Presentations

In all patients with cerebral ischemia of the posterior circulation, Doppler waveforms at the V1 to V2 portions (from the periphery of the origin of the VA to the transverse process of the third cervical vertebra) were recorded using 7.5 MHz linear and 6.0 MHz convex probes (Toshiba Medical Systems, Tokyo, Japan). Those at the V0 portion (at the origin of the VA) were also recorded if possible. Using the same criteria for severe stenosis at the origin of the internal carotid artery, we made a diagnosis of suspected severe stenosis with a maximum peak systolic flow velocity (maxPSV) of ≥200 cm/sec on VA waveforms. When the acceleration time (AcT) was prolonged...
to ≥110 msec, we suspected the presence of severe stenosis in the proximal region (Fig. 1). A definitive diagnosis of severe stenosis was made using cerebral angiography, and balloon angioplasty or stenting at the stenotic lesion was performed when endovascular treatment was considered necessary based on the clinical course. Postoperative follow-ups were conducted using Doppler ultrasonography of the VA.

Using the above methods, severe stenosis was detected in six patients (67–95 years, three males, three females) between January 2014 and March 2016 (Table 1). The stenotic sites detected on ultrasonography were the V0 (origin) in four patients, V1 (at the entry site of the VA into the transverse foramen of the sixth cervical vertebra) in one patient, and V2 (area between the transverse foramen of the third and sixth cervical vertebrae) in one patient. In two of the four patients with V0 stenosis, the increase in the flow velocity could be confirmed while directly observing the V0 portion using ultrasonography. In the other two patients, their stenoses were suspected based on AcT prolongation at the distal V2 portion. In patients with V1 and V2 stenosis, we directly confirmed an increase in the maxPSV at the stenotic site using ultrasonography. Subsequent cerebral angiography confirmed the presence of severe stenosis in each case, all of which were consistent with the sites suspected on ultrasonography. Furthermore, occlusion or hypoplasia of the contralateral VA was observed in all six patients. There was no patient in whom subsequent angiography revealed severe VA stenosis despite the absence of abnormalities on Doppler ultrasonography of the VA.

We performed stenting in cases with V0 stenosis, and balloon angioplasty for cases with V1 and V2 stenosis.

### Table 1  Summary of the cases

<table>
<thead>
<tr>
<th>Case</th>
<th>Patient</th>
<th>Stenosis</th>
<th>Tx</th>
<th>%stenosis (pre → post)</th>
<th>Measurement sites of echo</th>
<th>Echo results (pre)</th>
<th>Echo results (post)</th>
<th>Timing of post-echo</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>84F</td>
<td>Lt V0</td>
<td>Stent</td>
<td>79 → 12%</td>
<td>V2</td>
<td>MV = 5 cm/s</td>
<td>AcT = 242 ms</td>
<td>18 Mo</td>
</tr>
<tr>
<td>2</td>
<td>67M</td>
<td>Lt V0</td>
<td>Stent</td>
<td>74 → 20%</td>
<td>V2</td>
<td>MV = 28 cm/s</td>
<td>AcT = 133 ms</td>
<td>21 Mo</td>
</tr>
<tr>
<td>3</td>
<td>83F</td>
<td>Lt V0</td>
<td>PTA</td>
<td>79 → 46%</td>
<td>V2 (stenosis)</td>
<td>MaxPSV = 276 cm/s</td>
<td>MV = 32 cm/s</td>
<td>8 Mo</td>
</tr>
<tr>
<td>4</td>
<td>74F</td>
<td>Rt V0</td>
<td>Stent</td>
<td>80 → 28%</td>
<td>V2</td>
<td>MV = 17 cm/s</td>
<td>AcT = 50 ms</td>
<td>POD3</td>
</tr>
<tr>
<td>5</td>
<td>95M</td>
<td>Rt V1</td>
<td>PTA</td>
<td>69 → 50%</td>
<td>V1 (stenosis)</td>
<td>MaxPSV = 450 cm/s</td>
<td>MV = 21 cm/s</td>
<td>Not performed</td>
</tr>
<tr>
<td>6</td>
<td>81M</td>
<td>Lt V0</td>
<td>Stent</td>
<td>71 → 37%</td>
<td>V2</td>
<td>MV = 8 cm/s</td>
<td>AcT = 193 ms</td>
<td>1 Mo</td>
</tr>
</tbody>
</table>

AcT: acceleration time; MaxPSV: maximum peak systolic velocity; Mo: months; MV: mean velocity; POD: postoperative day; PTA: percutaneous transluminal angioplasty; Tx: treatment
Doppler Ultrasonography for Proximal VA Stenosis

at the left V2 portion, as well as the disappearance of
blood flow signals at the right V1 to V2, were noted, sug-
gest looking at severe stenosis at the left V0 portion and occlusion
at the right V0 portion. After admission, consciousness dis-
turbance and tetraparesis occurred repeatedly despite med-
cical treatment; severe stenosis at the left V0 was thought to
have caused posterior circulation ischemia. We considered
it difficult to control the symptoms by medical treatment alone. On 3 days after her admission, we confirmed 79%
stenosis at the left V0 portion with cerebral angiog-
phy and performed stenting at this site on the same day.
A Precise (Johnson & Johnson, Cordis, Miami, FL, USA)
7 × 40 mm stent was deployed at the stenotic site. Postop-
erative Doppler ultrasonography of the VA on the day of
surgery confirmed a marked improvement in blood flow

Representative cases
Case 1 (Fig. 2): An 84-year-old female presented with
weakness of the right half of her body. Magnetic resonance
imaging showed multiple acute brain infarctions involving
the brainstem and cerebellum. On Doppler ultrasonogra-
phy of the VA, the left V0 could not be observed, but the
prolongation of the systolic AcT (MV = 5 cm/sec, AcT =
242 msec) with a reduction in the mean velocity (MV)

neither recurrent symptom nor ≥50% restenosis. In five
of the six patients, postoperative Doppler ultrasonography
of the VA could be obtained. The reduction of maxPSV at
the stenotic site or AcT prolongation at the distal stenotic
area was observed in all the cases, suggesting the improve-
ment of their stenoses.

Fig. 2 Case 1. Stenting for stenosis at the origin of the left vertebral artery. Under distal vertebral artery pro-
tection with Filterwire (Boston Scientific, Marlborough, MA, USA), a Precise (Johnson & Johnson,
Cordis, Miami, FL, USA) 7 × 40 stent was deployed at the stenotic site. ([A] before surgery; [B] intra-
operative finding; [C] after surgery) Doppler ultrasonography of the vertebral artery showed the marked
prolongation of the AcT with a reduction in the MV before surgery (MV = 5 cm/sec, AcT = 242 msec,
[D]), and improvement of waveforms was noted immediately after surgery (MV = 17 cm/sec, AcT =
75 msec, [E]). AcT: acceleration time; MV: mean velocity
at the left V2 portion (MV = 17 cm/sec, AcT = 75 msec). Subsequent follow-up showed a further improvement (MV = 14 cm/sec, AcT = 49 msec) 18 months after the surgery. The improvement was maintained 27 months after surgery (MV = 15 cm/sec, AcT = 44 msec). There was no finding suggestive of restenosis at the site of stenting. There were no recurrent brain infarctions afterwards.

Case 3 (Fig. 3): An 83-year-old female was referred to our hospital to perform detailed examinations after brainstem infarction. Doppler ultrasonography of the VA showed a localized increase in maxPSV (276 cm/sec) at an area adjacent to the transverse foramen of the fourth cervical vertebra at the left V2 portion, suggesting severe V2 stenosis. Hypoplasia of the right VA was also observed. Medical treatment with dual-antiplatelet therapy was started. However, right thalamic infarction, in which the source of embolism could have been the stenotic V2, recurred. Considering that the possibility of further recurrent brain infarctions, endovascular treatment was selected. Cerebral angiography revealed 79% stenosis at the same site as indicated on ultrasonography and balloon angioplasty was performed. Using a Gateway (Stryker, Kalamazoo, MI, USA) monorail 2.5 × 12 balloon, successful angioplasty was completed. There has been no recurrent brain infarction during the subsequent 18-month follow-up. Doppler ultrasonography of the VA 8 months after the surgery showed a reduction of maxPSV (153 cm/sec) at the stenotic site.

Case 5 (Fig. 4): A 95-year-old male presented at another hospital with visual-field disorder. Under a diagnosis of multiple acute brainstem and cerebellar infarctions, medical
treatment was initiated. However, the infarcts enlarged, and he was referred to our hospital. Doppler ultrasonography of the VA showed a localized increase in maxPSV at the right V1 (maxPSV = 450 cm/sec) and prolongation of the AcT with a reduction in the MV at the V2 (MV = 21 cm/sec, AcT = 117 msec), suggesting severe stenosis at the V1. Considering hypoplasia of the left VA, this severe V1 stenosis was suspected to be the source of recurrent infarction. We speculated that the hemodynamic load on posterior circulation might be strong and endovascular intervention was indicated. Cerebral angiography showed 69% stenosis at the same site as indicated on ultrasonography, and balloon angioplasty at this site was performed. Using a Gateway (Stryker) monorail 2 × 12 mm balloon, successful angioplasty was performed and the stenosis rate reduced to 50%. Subsequently, there were no recurrent brain infarctions, and the patient was transferred to another hospital for rehabilitation. Follow-up Doppler ultrasonography of the VA could not be performed.

**Discussion**

Previous studies demonstrated that a maxPSV over 200 cm/sec at the stenotic site on Doppler ultrasonography corresponded to a stenosis of 70% by the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria in patients with internal carotid artery stenosis, and that the prolongation of the AcT to ≥110 msec at the distal carotid artery suggested a stenosis of over 60% by the NASCET criteria at its origin. Another study reported that similar findings were useful for the diagnosis of stenosis at VA origin. In this study, we performed Doppler ultrasonography of the VA in patients with posterior circulation ischemia as screening to determine whether cerebral angiography should be indicated. We incorporated the same criteria as used for the diagnosis of severe stenosis at the origin of the internal carotid artery, and there was no inconsistency between the ultrasonography and angiography findings. Ultrasonography can be simply performed.
at patients’ bedside and it is appropriate for repeated examination since it does not require contrast media. Therefore, it was not only useful as a screening before detailed vascular examination, but also applicable for the follow-up after endovascular interventions.

With respect to the assessment of VA stenosis using Doppler ultrasonography, a study reported a method to diagnose severe VA stenosis based on the stenotic-/distal-stenotic-site maxPSV ratio. However, the V0 portions are not visible in all patients; therefore, this criterion cannot be used in some patients. Furthermore, the following precautions must be considered for the diagnosis of severe stenosis based on a maxPSV of ≥200 cm/sec: 1) if the steal phenomenon of the contralateral subclavian artery is present, moderate or slight stenosis may be overdiagnosed as severe stenosis due to a maxPSV of >200 cm/sec through an increase in the flow velocity of the tested-side VA (false positive); 2) if tandem stenosis is present at the distal area of the tested-side VA, severe stenosis may be underdiagnosed as moderate or slight stenosis due to a maxPSV of <200 cm/sec through a reduction in the flow velocity of the proximal VA (false negative); and 3) if long-segment stenosis is partially severe, the maxPSV may not increase (false negative). All subjects of our study showed contralateral hypoplasia or occlusion, and the hemodynamics were relatively simple: posterior circulation blood flow depended on affected-side VA blood flow. In such cases, there may have been direct associations among the progression of stenosis, the increase in maxPSV and the symptoms related to blood flow reduction. Therefore, Doppler ultrasonography can be useful for the detection of lesions requiring treatment. However, the usefulness of Doppler ultrasonography in cases with sufficient blood flow of contralateral VA should be examined in a larger number of patients in the future.

For the assessment of the VA using Doppler ultrasonography, ultrasonographic skill and experience are required for the following reasons: the course of this artery involves an area deeper than the internal carotid artery; some areas surrounded by the transverse process are poorly visualized; and the vascular diameter is extremely small if VA was hypoplastic. However, if the technicians are skilled in the detection of severe stenosis at the origin of the internal carotid artery, it is practically possible to detect stenosis of the proximal VA without problems. In the future, it should be investigated in comparison with angiography findings in a larger number of patients, as to whether the diagnostic criteria for internal carotid artery stenosis are also applicable for VA stenosis.

The usefulness of endovascular treatment for the proximal VA area has not been clarified. However, as in our series, some patients with severe stenosis of the proximal VA resist medical treatment and experience repeated ischemia of the posterior circulation system. If candidates for endovascular treatment can be detected in the early stage using Doppler ultrasonography, its usefulness may further become apparent.

## Conclusion

Doppler ultrasonography of the proximal VA can be performed noninvasively and repeatedly. It is useful not only for the screening of severe stenosis at the V0-V2 portion, but also in follow-up after endovascular interventions such as stenting and angioplasty.

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## Disclosure Statement

There is no conflict of interest for the main author or coauthors.

## References