Cerebellar Infarction
Clinical and Neuroimaging Analysis in 293 Patients
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for the Tohoku Cerebellar Infarction Study Group

Background and Purpose: We performed this multicenter study to explore the full spectrum of the clinical characteristics and neuroimaging findings of cerebellar infarction, including patients with mild to severe illnesses.

Methods: We studied 293 consecutive patients with cerebellar infarction diagnosed by computed tomography and/or magnetic resonance imaging who were admitted to 36 hospitals during 5 years.

Results: Cerebellar infarcts constituted 2.3% of the total patients with acute brain infarction. The backgrounds and risk factors were similar to those in patients with infarctions of the cerebral hemispheres. At least 24% were embolic, and the diagnosis of embolism could not be ruled out in 27%. Infarcts involving the superior cerebellar artery (SCA) region (52%) and the posterior inferior cerebellar artery (PICA) region (49%) were far more frequent than those involving the anterior inferior cerebellar artery (AICA) region (20%). Patients with SCA infarcts exhibited obtunded consciousness and ataxia more frequently than those with PICA infarcts (P<.05). Infarcts in the PICA regions were associated with abnormalities of the PICA (64%) or the vertebral arteries (57%), whereas infarcts in the SCA and AICA regions were associated with abnormalities in the SCA or AICA, respectively, in approximately 30% of patients, in the basilar artery in approximately 16%, and in the vertebral artery in more than 60% of patients. Outcomes were poorer with SCA infarcts than with AICA and PICA infarcts.

Conclusions: These data indicate similar frequencies of SCA and PICA infarcts and illustrate the difference in clinical presentation and outcomes between SCA and PICA infarcts. They also indicate that not only in situ thrombosis but also cardiogenic or artery-to-artery embolism and the insufficiency of collateral circulation play important roles in the pathogenesis of cerebellar infarction. (Stroke. 1993;24:1697-1701.)

KEY WORDS • angiography • cerebellar infarction • risk factors • tomography, x-ray computed

Numerous reports have been published concerning clinical and autopsy findings of cerebellar infarction. However, most of these studies have focused on either patients with fatal outcomes examined at autopsy or patients with severe massive cerebellar infarction who needed surgical intervention. Therefore, our knowledge concerning the full spectrum of cerebellar infarcts encompassing patients with mild to severe illnesses is still insufficient. It is because of the difficulty in diagnosing cerebellar infarction without the aid of computed tomography (CT) or magnetic resonance imaging (MRI) and because cerebellar infarction is relatively infrequent (1.5% in autopsy series, 0.6% in CT series, 0.7% in CT series) that detailed analyses on a sufficient number of patients are difficult in individual institutions. Previous CT-based studies on a considerable number of patients have reported a greater fatality rate of cerebellar infarction than other forms of cerebral infarction in 30 patients and differences in the clinical characteristics between infarctions in the distribution of the superior cerebellar artery (SCA) and the posterior inferior cerebellar artery (PICA) in 66 patients.

In this multicenter study, we included consecutive series of CT/MRI-confirmed patients (n=293) with mild to very severe illnesses whose infarctions were mainly in the cerebellum and analyzed (1) the incidence, demographic characteristics, and risk factors of cerebellar infarction; and (2) the location of lesions and their relations with clinical characteristics, vascular changes demonstrated in angiography, and outcome.

Subjects and Methods
Thirty-six institutions throughout the Tohoku district of Japan participated in the study. We reviewed clinical records, CT, and MRI scans of patients with acute brain infarctions who were admitted to the hospitals from January 1, 1987 to December 31, 1991 (5 years). We included patients having infarctions mainly involving the cerebellum. We excluded patients whose lesions were mainly in the brain stem, extending to the cerebellum, such as midbrain and pontine infarctions and lateral medullary syndrome. There were 12 545 patients with acute brain infarction, among whom 293 (2.3%) had main lesions in the cerebellum. CT and/or MRI was performed in all patients immediately after admission and was repeated several days later to confirm the diagnosis or if there was any deterioration. Angiography

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was performed in 157 (54%) patients. Because the decision to perform angiography depended mainly on the policies of individual institutions, our angiography data did not seem to suffer from serious bias. We reviewed angiographic films and recorded the presence or absence of plaques, stenosis (with grades of <25%, 25% to 50%, 50% to 75%, and >75%), and occlusion for individual cerebellar arteries and the vertebral and basilar arteries. Cases of occlusion and greater than 50% stenosis were submitted to statistical analysis. The diagnosis of cardioembolic embolism was made based on at least two of the following: abrupt onset; evidence of heart diseases such as valvular diseases, myocardial infarction, and a trial fibrillation; CT and/or MRI findings of hemorrhagic infarctions; and absence of severe atherosclerotic changes on angiography. The consciousness levels were graded into clear, confused, obtunded, and comatose. The vascular territories of infarctions were determined in terms of the SCA, anterior inferior cerebellar artery (AICA), and PICA based on the studies of Lazorthes et al.,11 Salamon,12 and Savoiardo et al.13

After admission, 149 (51%) patients received glycerol, 37 (13%) mannitol, 40 (14%) steroid, and 26 (9%) fibrinolytic medication. Twenty-four patients underwent emergency surgery of decompression, removal of tissues, ventricular drainage/shunting, and combinations of these. Patients were admitted for 51±90 days (mean±SD). By 3 months after onset, the outcomes of patients were evaluated as independent, dependent, bedridden, vegetative, and expired. Intergroup differences in frequencies were analyzed by the χ² test.

Results

The mean±SD age of the patients with cerebellar infarction was 65±12 years. Two hundred sixteen (74%) of the patients were men, and 77 were women. One hundred forty-three (49%) were thrombotic, 70 (24%) embolic, and the diagnosis of thrombosis/embolism could not be differentiated clearly in 80 (27%). The backgrounds and risk factors of the patients are presented in Table 1.

Symptoms at onset and neurological signs on examination are listed in Table 2. Dizziness/vertigo was the most frequent symptom, followed by nausea/vomiting, unsteady gait, headache, dysarthria, and tinnitus (Table 2). We did not find significant differences in the incidence of individual symptoms among patients having infarctions in the SCA, AICA, and PICA territories (data not shown). Frequent neurological signs included limb ataxia, truncal ataxia, dysarthria, and nystagmus. Approximately two thirds of patients were alert, and one third were confused, obtunded, or comatose. A significantly greater proportion of patients with embolism (36/70, 51%) had disturbed consciousness compared with patients with thrombosis (35/143, 24%) (P<.005). Obtundation or coma (15/115, 13%) and limb ataxia (73/115, 63%) were significantly more frequent in those with SCA infarcts than in those with PICA infarcts (1/106, 1%, P<.001 and 52/106, 49%, P<.05, respectively).

Cerebellar infarctions were unilateral in 257 (88%) patients and bilateral in 36 (12%). Bilateral cerebellar infarcts were significantly more frequent in patients with embolism (15/70, 21%) than in patients with thrombosis (14/143, 10%) (P<.05). Hemorrhagic infarctions developed in 32 (11%) patients and occurred far more frequently in patients with embolism (20/70, 29%) than in patients with thrombosis (5/143, 3%) (P<.001). The infarct areas included the SCA region in 151 (52%), the AICA region in 59 (20%), and the PICA region in 145 (49%) (infarcts overlapping adjacent vascular territories were assigned to individual arteries). Forty-five (15%) patients had brain-stem infarcts, 72 (25%) patients brain-stem compression, 14 (5%) herniation, and 29 (10%) hydrocephalus.

### Table 1. Background and Risk Factors in 293 Patients With Cerebellar Infarction

<table>
<thead>
<tr>
<th>Factor</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>133</td>
<td>45</td>
</tr>
<tr>
<td>Smoking</td>
<td>110</td>
<td>38</td>
</tr>
<tr>
<td>History of stroke</td>
<td>70</td>
<td>24</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>68</td>
<td>23</td>
</tr>
<tr>
<td>High hematocrit (≥45%)</td>
<td>65</td>
<td>22</td>
</tr>
<tr>
<td>Low HDL (≤45 mg/dL)</td>
<td>61</td>
<td>21</td>
</tr>
<tr>
<td>Diabetes</td>
<td>48</td>
<td>16</td>
</tr>
<tr>
<td>Hypercholesterolemia (≥230 mg/dL)</td>
<td>33</td>
<td>11</td>
</tr>
<tr>
<td>Hyperuricemia (≥7 mg/dL)</td>
<td>16</td>
<td>6</td>
</tr>
<tr>
<td>History of myocardial infarction</td>
<td>7</td>
<td>2</td>
</tr>
</tbody>
</table>

HDL indicates high-density lipoprotein.

### Table 2. Symptoms, Signs, and Consciousness Levels on Admission in 293 Patients With Cerebellar Infarction

<table>
<thead>
<tr>
<th>Symptom</th>
<th>No.</th>
<th>%</th>
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</thead>
<tbody>
<tr>
<td>Dizziness/Vertigo</td>
<td>206</td>
<td>70</td>
</tr>
<tr>
<td>Nausea/vomiting</td>
<td>165</td>
<td>56</td>
</tr>
<tr>
<td>Gait disturbance</td>
<td>116</td>
<td>40</td>
</tr>
<tr>
<td>Headache</td>
<td>94</td>
<td>32</td>
</tr>
<tr>
<td>Dysarthria</td>
<td>59</td>
<td>20</td>
</tr>
<tr>
<td>Tinnitus</td>
<td>14</td>
<td>5</td>
</tr>
<tr>
<td>Signs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Limb ataxia</td>
<td>172</td>
<td>59</td>
</tr>
<tr>
<td>Truncal ataxia</td>
<td>133</td>
<td>45</td>
</tr>
<tr>
<td>Dysarthria</td>
<td>123</td>
<td>42</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>111</td>
<td>38</td>
</tr>
<tr>
<td>Hemiparesis</td>
<td>59</td>
<td>20</td>
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<tr>
<td>Facial palsy</td>
<td>23</td>
<td>8</td>
</tr>
<tr>
<td>Anisocoria</td>
<td>17</td>
<td>6</td>
</tr>
<tr>
<td>Conjugate deviation</td>
<td>18</td>
<td>6</td>
</tr>
<tr>
<td>Horner's syndrome</td>
<td>15</td>
<td>5</td>
</tr>
<tr>
<td>Upward gaze palsy</td>
<td>12</td>
<td>4</td>
</tr>
<tr>
<td>Loss of light reflex</td>
<td>11</td>
<td>4</td>
</tr>
<tr>
<td>Consciousness levels on admission</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clear</td>
<td>195</td>
<td>67</td>
</tr>
<tr>
<td>Confused</td>
<td>73</td>
<td>25</td>
</tr>
<tr>
<td>Obtundled</td>
<td>20</td>
<td>7</td>
</tr>
<tr>
<td>Comatose</td>
<td>5</td>
<td>2</td>
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</tbody>
</table>

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TABLE 3. Relations of Vascular Territories of Infarctions and Angiographic Abnormalities

<table>
<thead>
<tr>
<th>Vascular Territories of Infarctions</th>
<th>SCA (n=59)</th>
<th>AICA (n=6)</th>
<th>PICA (n=69)</th>
<th>SCA+AICA (n=8)</th>
<th>SCA+PICA (n=2)</th>
<th>AICA+PICA (n=5)</th>
<th>SCA+AICA+PICA (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vertebral artery</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Ipsilateral</td>
<td>23</td>
<td>2</td>
<td>28</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>5</td>
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<tr>
<td>Contralateral</td>
<td>15</td>
<td>2</td>
<td>11</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td><strong>Basilar artery</strong></td>
<td>9</td>
<td>1</td>
<td>6</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td><strong>PICA</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ipsilateral</td>
<td>13</td>
<td>2</td>
<td>44*</td>
<td>1</td>
<td>1*</td>
<td>2*</td>
<td>5*</td>
</tr>
<tr>
<td>Contralateral</td>
<td>8</td>
<td>0</td>
<td>9</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>2</td>
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<tr>
<td><strong>AICA</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ipsilateral</td>
<td>11</td>
<td>2*</td>
<td>11</td>
<td>1*</td>
<td>1*</td>
<td>2*</td>
<td>2*</td>
</tr>
<tr>
<td>Contralateral</td>
<td>10</td>
<td>0</td>
<td>9</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td><strong>SCA</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ipsilateral</td>
<td>20*</td>
<td>0</td>
<td>4</td>
<td>1*</td>
<td>0</td>
<td>0</td>
<td>3*</td>
</tr>
<tr>
<td>Contralateral</td>
<td>11</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>

Values are number of patients with occlusion or >50% stenosis of individual arteries. SCA indicates superior cerebellar artery; AICA, anterior inferior cerebellar artery; and PICA, posterior inferior cerebellar artery.

*Main arteries irrigating individual infarct territories.

The relations between vascular territories of infarctions and abnormalities of the cerebellar, basilar, and vertebral arteries are presented in Table 3. In the SCA infarct patients, 34% (20/59) had primary abnormalities in the SCA, 15% (9/59) in the basilar artery, and 70% (38/54) in the right or left vertebral arteries. Similarly, in the AICA infarct patients, 33% (2/6) had abnormalities in the AICA, 17% (1/6) in the basilar artery, and 66% (4/6) in the vertebral arteries. In patients with PICA infarcts, 64% (44/69) had abnormalities in the PICA and 57% (39/69) in the vertebral arteries. Severe abnormalities were also found in cerebellar arteries that do not distribute directly to infarct regions (Table 3). The collateral circulation was insufficient in 55% (54/98) of patients without brain-stem compression, in 62% (8/13) of patients with moderate brain-stem compression, and in 92% (12/13) of patients with severe brain-stem compression necessitating surgical intervention.

Outcomes were recorded in 282 patients. One hundred ninety-four (69%) patients were classified as independent, 60 (21%) dependent, 11 (4%) bedridden, 2 (1%) in a vegetative state, and 15 (5%) died. The patients with embolism had a nonsignificant trend to have a lower rate of independent recovery (38/65, 58%) compared with patients with thrombosis (98/138, 71%) (P=.08). The rate of patients classified as independent decreased with the more severe state of consciousness disturbance on admission: 152/195 (78%) in alert patients, 32/75 (43%) in confused patients, and 3/25 (12%) in obtunded or comatose patients. Conversely, the rate of patients who became bedridden, vegetative, or died increased with more severe levels of consciousness disturbance: 2% in alert patients, 13% in confused patients, and 57% in obtunded or comatose patients. The prognosis of patients was also related to the regions of the cerebellar arteries involved (Table 4). Among patients having lesions involving single cerebellar artery territories, the rate of patients classified as independent was greater in patients with AICA and PICA infarcts than in patients with SCA infarcts (P<.005 for PICA infarcts). The rate was lower in patients with combined involvement of two cerebellar arteries and was lower still in patients with combined involvement of the three cerebellar arteries.

Of the 24 patients who underwent emergency surgery, 7 (29%) patients recovered and were classified as independent, 9 (28%) dependent, and 2 (8%) were bedridden, 1 (4%) vegetative, and 5 (21%) died (3 of the cerebellar infarcts and 2 of pneumonia).

**Discussion**

The observed incidence of cerebellar infarcts among total brain infarctions (2.3%) is comparable to the results of Macdonell et al (1.5%). However, because it has been reported that half of old cerebellar infarcts found at autopsy were asymptomatic and because symptoms and signs in cerebellar infaracts are very similar to benign peripheral labyrinthine disorders, many patients with cerebellar infarcts are likely to be overlooked if CT/MRI studies are not performed. Although some previous studies reported the relative rarity of SCA infarcts, the frequencies of SCA and PICA infarcts in our series are consistent with other reports focusing on cerebellar infarcts, in which SCA infarcts were as frequent as or more numerous than PICA infarcts. Similarly, our exclusion of cerebellar infarcts associated with brain-stem infarcts may explain the results that PICA infarctions were related more frequently with abnormalities in PICA than in the ipsilateral vertebral artery, the known usual site of occlusion in PICA infarcts. We did not find watershed infarcts, which is consistent with the results of Amaerence et al but inconsistent with those of others. This may be because the cerebellar infarcts in our patients were large enough to cause severe symptoms.

Other major findings to be addressed are as follows: (1) cerebellar infarctions were produced by cardiogenic embolism in at least one fourth of patients; (2) cerebellar infarcts were associated not only with abnormalities of cerebellar arteries that distribute directly to the infarct
areas but also with stenosis/occlusion of other cerebellar arteries, the basilar artery, and the vertebral artery; and (3) prognosis was related to the presence and grades of consciousness disturbance, which reflect brain-stem involvement, and functional disability occurred most frequently in those with SCA infarcts compared with those with lesions in other single artery regions.

The importance of a cardiac source of emboli in the pathogenesis of infarctions in the vertebrobasilar system has been suggested previously.\(^1\) In cerebellar infarcts, the differential diagnosis of thrombosis and embolism was often difficult because the vestibulocerebellar symptoms appear suddenly regardless of the underlying etiologies, and the subsequent clinical progression is not exactly ascribable to further thrombus formation or to the development of brain edema. Thus, in our series, at least 24% of the cerebellar infarcts were embolic and in another 27% the diagnosis of embolism could not be ruled out. This is in accordance with the results of Amarenco et al.,\(^2\) who reported that cardioembolic embolism was proven in 24% and presumed in 19% of their patients. Other studies also have reported similar (22%, Feely\(^2\) \(\approx\) 25%, Sypert and Alvord\(^1\)) or even much higher (50%, Heros\(^2\)) rates of embolism. The incidence of embolism in their studies and ours is greater than in a previous study on vertebrobasilar system infarctions (9.2%, Castaigne et al\(^2\)). This seems to indicate that infarctions mainly involving the cerebellum caused by occlusion of long circumferential cerebellar arteries\(^3\) are more frequently produced by embolism than infarctions involving both the cerebellum as well as the brain stem due to occlusion of vertebral and/or basilar arteries. In our results, embolic infarctions became hemorrhagic 10 times more frequently than thrombotic infarctions. The smaller rate of hemorrhagic infarctions (11%) in our patients than that reported by Sypert and Alvord\(^1\) and Heros\(^2\) (approximately 25%) may be because their patients had large infarctions and came to autopsy or underwent surgical intervention.\(^3\)

Previous studies have reported that the SCA infarcts were associated with abnormalities of the SCA, basilar artery, and the vertebral artery with various frequencies.\(^2\) In our SCA and AICA infarct patients, as many as two thirds had occlusion or severe stenosis of the right or left vertebral arteries, suggesting that a substantial proportion of SCA and AICA infarcts appears to be related to abnormalities of the vertebral arteries. Although the roles of the abnormalities of the vertebral arteries are not precisely known, it is most probable that they may provide a source of artery-to-artery embolism, as has been suggested in previous studies.\(^2\) In addition, our findings indicate that abnormalities of cerebellar arteries that do not distribute directly to infarct regions play an important role in the production of cerebellar infarcts. This was demonstrated particularly for the SCA infarcts, of which 41% were associated with occlusion/stenosis in the PICA and AICA. We also found that in almost all patients presenting with brain-stem compression severe enough to receive surgical intervention, infarct areas received little blood supply from the cerebellar arteries distributing to other areas. These findings suggest a substantial role of insufficient collateral circulation in the production of massive cerebellar infarctions. Taken together, cardiogenic and artery-to-artery embolism and insufficiency of collateral circulation play important roles in the pathogenesis of infarctions mainly involving the cerebellum that occur in the areas of long circumferential arteries of the infratentorial posterior circulation, and their pathogenetic mechanisms are very similar to cortical infarctions in the supratentorial anterior circulation.

The usual course of cerebellar infarctions is benign with variable degrees of functional recovery in the majority of patients but sometimes may be progressive, leading to brain-stem compression and hydrocephalus in a minority of patients.\(^13\) Our results indicate that neurological disabilities increased greatly with only a mild disturbance of consciousness indicative of brain-stem involvement and that more severe brain-stem involvement led to life-threatening aggravation in approximately 10% of patients. Although immediate operative intervention is necessary for such patients, we could not find any symptoms or signs that predicted subsequent severe cerebellar swelling. However, our results indicate that infarcts limited to the PICA territory exerted less severe effects than SCA infarcts, which is not consistent with some previous studies. The reported higher tendency for PICA infarcts to lead to severe mass effect and brain-stem compression appears to be mainly due to involvement of greater areas of the cerebellum extending to the territories of other
cerebellar arteries. In our results, obtundation and coma occurred more often with SCA infarcts than with PICA infarcts, which may reflect the greater probability for the midbrain reticular formation to be involved in SCA infarcts and may reflect the exclusion in our series of subjects with identified significant brain-stem involvement. We also found that ataxia of the limb and trunk was more frequent with SCA infarcts than with PICA and AICA infarcts. This may be because SCA irrigates important structures in the cerebellum such as the dentate nucleus, anterior vermis, and the superior cerebellar peduncle, which conveys most of the cerebellar efferents.

Appendix

Study Participants
Takashi Moriyama (Department of Neurosurgery, Aomori City Hospital); Shigeaki Kanayama (Department of Neurosurgery, Hachinohe City Hospital); Yutaka Hirata (Department of Neurology, Research Institute for Brain and Blood Vessels, Akita); Hajime Kagaya (Department of Neurology, Nakadohi Hospital); Kazuo Ebina (Department of Neurosurgery, Nakadohi Hospital); Nobuo Kamisato (Department of Neurosurgery, Akita Rousai Hospital); Masayoshi Kowada (Department of Neurosurgery, Akita University School of Medicine); Hideki Tsukubakisa (Department of Neurosurgery, Hokkushin Central Hospital); Iwao Sakai (Department of Neurosurgery, Iwate Medical University); Kimiaki Utsugisawa (Department of Neurology, Iwate Medical University); Kenichi Tamura (Department of Neurology, Iwate Prefectural Central Hospital); Hiroshi Higuchi (Department of Neurosurgery, Iwate Prefectural Central Hospital); Takahiko Kikuchi (Department of Neurology, Iwate Prefectural Daito Hospital); Hirotaka Sugiyama (Department of Neurosurgery, Iwate Prefectural Kamaishi Hospital); Takashi Yoshimoto (Department of Neurosurgery, Institute of Brain Diseases, Tohoku University School of Medicine); Keiji Koshu (Department of Neurosurgery, Kohnan Hospital); Kouji Narikawa (Department of Neurology, Miyagi National Hospital); Yukihito Miyazawa (Department of Neurology, National Sendai Hospital); Kenji Tsuburaya (Department of Neurology, Tokohu Kosei-Nenkin Hospital); Tadao Matsuhashi (Department of Neurosurgery, Southern Tohoku Research Institute for Medical Science); Masakazu Kitahara (Department of Neurology, Ishinomaki Red Cross Hospital); Masatoshi Oba (Department of Neurosurgery, Kesennuma County Hospital); Takehide Onuma (Department of Neurosurgery, Sendai City Hospital); Osamu Nakai (Department of Surgical Neurology, Yamagata University); So Sato (Department of Neurosurgery, Yamagata City Hospital); Tsutomu Miura (Yonaizawa General Hospital); Takao Watanabe (Department of Neurosurgery, Yonezawa City Hospital); Kazuo Watanabe (Institute for Neuroscience, Southern Tohoku Research); Namio Kodama (Department of Neurosurgery, Fukushima Medical School); Masaaki Usui (Department of Neurosurgery, Aizu Central Hospital); Kiyomi Yamane (Department of Neurology, Neurological Institute, Ohta-Atami Hospital); Hirobumi Metoki (Department of Internal Medicine, Reimeikyo Rehabilitation Hospital); Yasuhiro Kikuchi (Department of Neurosurgery, Masu Memorial Hospital); Hirobumi Seki (Department of Neurosurgery, Obara Medical Center); Yasuo Kurashima (Department of Neurosurgery, Fujita General Hospital); and Kenjiro Shindo (Department of Neurosurgery, Yuru General Hospital).

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References
Cerebellar infarction. Clinical and neuroimaging analysis in 293 patients. The Tohoku Cerebellar Infarction Study Group.
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