Spontaneous Closure of Transverse Sinus Dural Arteriovenous Fistula
—Case Report—

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Abstract
A 60-year-old man presented with transverse sinus dural arteriovenous fistula (AVF) manifesting as sudden onset of headache and nausea, which underwent spontaneous closure 5 years after the onset. Computed tomography on admission revealed small intraventricular hemorrhage in the right lateral ventricle. No intracranial vascular lesion was detected and magnetic resonance angiography was used at yearly follow up. Two years after the first admission, he suffered diplopia and cerebral angiography revealed transverse sinus dural AVF. Right pulsatile tinnitus occurred 4 years after the first admission. The symptoms suddenly disappeared 5 years after the first admission, and follow-up angiography showed disappearance of the dural AVF. The exact mechanism of the spontaneous occlusion of dural AVF remains unknown. This case of spontaneous transverse sinus dural AVF closure occurred without disruption of sinus patency, suggesting that thrombosis of the draining veins into sinuses was not involved.

Key words: dural arteriovenous fistula, transverse sinus dural arteriovenous fistula, spontaneous closure, spontaneous occlusion

Introduction
Dural arteriovenous fistulas (AVFs) account for 10–15% of intracranial arteriovenous malformations, and the pathogenesis and treatments are well established, but the natural history, especially the mechanisms of spontaneous closure, remains unclear. Thrombosis of the draining vein or sinus could trigger the secondary closure of dural AVF, but the precise mechanism is unknown. Only 17 cases of spontaneous closure have been reported.

We report a case of angiographically proven spontaneous regression of transverse sinus dural AVF.

Case Report
A 60-year-old man was referred to our department in November 2001 with sudden onset of headache and nausea. He was alert and had no paresis. Computed tomography on admission revealed intraventricular hemorrhage in the right lateral ventricle and the third ventricle, suggesting a bleeding point in the right caudate nucleus (Fig. 1A). Magnetic resonance (MR) angiography disclosed no vascular lesion accounting for the ventricular hemorrhage (Fig. 1B). He was conservatively treated and discharged 17 days after admission without neurological deficit. Two years after the first admission, he complained of slight diplopia caused by right trochlear nerve paresis. Follow-up MR angiography showed dilation of the right occipital artery and early partial filling of the right transverse and sigmoid sinuses (Fig. 2). We recommended detailed examination of the vascular lesion by cerebral angiography.

Cerebral angiography revealed right transverse sinus dural AVF filling through the right occipital artery, right middle meningeal artery, and meningeal branches from the right ascending pharyngeal artery (Fig. 3). No cortical reflux was found and the normal...
Fig. 1 A: Computed tomography scans on first admission revealing right intraventricular hemorrhage, possibly from the right caudate nucleus. B: Magnetic resonance angiograms showing no remarkable vascular anomaly.

Fig. 2 Magnetic resonance angiograms (A) and digital subtraction angiogram (B) 2 years after the first admission showing dilation of the right occipital artery and early partial filling of the right transverse and sigmoid sinuses (arrows).

Fig. 3 Selective external carotid angiograms, anteroposterior (A) and lateral projections (B), revealing a right transverse sinus dural arteriovenous fistula filling through the right occipital artery, right middle meningeal artery, and meningeal branches from the right ascending pharyngeal artery, no cortical reflux, and preserved normal venous circulation.

Fig. 4 Selective angiograms, lateral projections, from the right internal carotid artery (A) and the right external carotid artery (B), 4 years after the first admission revealing additional shunting channels from tentorial branches from the right internal carotid artery (A, arrow), decreased shunt flow supplied by the occipital artery and ascending pharyngeal artery (B, arrow) compared with the findings 2 years previously, and no cortical reflux and no blockade of the normal venous circulation.

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Venous circulation was preserved. He was conservatively treated, resulting in gradual improvement of the diplopia, and no neurological symptoms appeared for 2 years.

Four years after the first admission, in 2005, he was referred to our department with right pulsatile tinnitus. Second cerebral angiography disclosed additional shunting channels from tentorial branches from the right internal carotid artery (Fig. 4). Shunt flow supplied by the occipital artery and ascending
Fig. 5 Selective angiograms, lateral projections from the right internal carotid artery (A) and the right external carotid artery (B), and anteroposterior projection of the venous phase from the right internal carotid artery (C), 5 years after the onset showing disappearance of the transverse sinus dural arteriovenous fistula (A, B), no thrombotic or occlusive findings, and patency of the venous circulation in the sinuses (C).

Table 1 Spontaneous closure of transverse sinus dural arteriovenous fistulas

<table>
<thead>
<tr>
<th>Author (Year)</th>
<th>Age (yrs)/Sex</th>
<th>Symptoms at diagnosis</th>
<th>Feeder</th>
<th>Cognard classification type</th>
<th>Symptoms at occlusion</th>
<th>Sinus</th>
<th>Duration* (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Magidson and Weinberg (1976)</td>
<td>39/F</td>
<td>tinnitus, bruit</td>
<td>MMA, OA</td>
<td>I</td>
<td>vertigo, cephalalgia</td>
<td>thrombosed</td>
<td>30</td>
</tr>
<tr>
<td>Hansen and Sogaard (1976)</td>
<td>23/F</td>
<td>tinnitus, bruit</td>
<td>VA, OA</td>
<td>I</td>
<td>none</td>
<td>patent</td>
<td>11</td>
</tr>
<tr>
<td>Endo et al. (1979)</td>
<td>26/F</td>
<td>tinnitus, vertigo</td>
<td>MMA, OA</td>
<td>Ia + b</td>
<td>none</td>
<td>patent</td>
<td>24</td>
</tr>
<tr>
<td>Kataoka and Taneda (1984)</td>
<td>56/M</td>
<td>coma, IVH</td>
<td>OA</td>
<td>Ia</td>
<td>coma</td>
<td>thrombosed</td>
<td>14</td>
</tr>
<tr>
<td>Luciani (2001)</td>
<td>42/M</td>
<td>tinnitus, bruit</td>
<td>OA, MMA</td>
<td>I</td>
<td>none</td>
<td>patent</td>
<td>185</td>
</tr>
<tr>
<td></td>
<td>57/F</td>
<td>tinnitus, bruit</td>
<td>PMA, MMA</td>
<td>I</td>
<td>none</td>
<td>patent</td>
<td>240</td>
</tr>
<tr>
<td>Moriya et al. (2007)</td>
<td>60/M</td>
<td>tinnitus</td>
<td>OA, MMA</td>
<td>I</td>
<td>none</td>
<td>patent</td>
<td>48</td>
</tr>
</tbody>
</table>

*Duration from the initial diagnosis to confirmation of spontaneous closure. IVH: intraventricular hemorrhage, MMA: middle meningeal artery, OA: occipital artery, PMA: posterior meningeal artery, VA: vertebral artery.

pharyngeal artery was decreased compared with the findings 2 years before. There was no cortical reflux and no blockade of the normal venous circulation. The right tinnitus continued without aggravation for 1 year.

He presented with transient chest pain in September 2006, and cardiac catheter angiography was performed in the cardiovascular department. His tinnitus persisted after the examination, but suddenly disappeared in October 2006. Third cerebral angiography 5 years after the onset showed disappearance of the dural AVF and no new blockade or stenotic changes of the sinuses (Fig. 5).

Discussion

Only 7 cases of spontaneous resolution of transverse sinus dural AVFs have been reported in addition to the present case.4,7-12) All cases are summarized in Table 1. Five cases were Cognard classification type I and the venous circulation in the sinus was patent. The mean period from diagnosis to occlusion was 78.9 months. Our patient did not suffer any head trauma, but underwent cardiac catheterization for angina 1 month before the disappearance of pulsatile tinnitus. Transverse sinus dural AVF spontaneously closed 7 days after diagnostic cerebral angiography in one case.12) An allergic response to the contrast medium might cause thrombotic change in the dural AVF, but the relationship is unclear.

Cases of spontaneous closure of dural AVFs have various clinical characteristics. First, the incidence of Cognard classification type I was high. Type I includes dural AVFs that drain into a sinus, with normal antegrade flow.3) The percentage of type I in dural AVFs which underwent spontaneous closure...
reached 67%,12) Our case had antegrade venous draining flow into the transverse sinus without cortical reflux, classified as type I. Second, the clinical symptoms changed before spontaneous closure. Transient clinical symptoms, such as vertigo, auditory disturbance, nausea, and conscious disturbance, were observed before the disappearance of initial symptoms in some reports. No significant symptoms such as aura were observed before the disappearance of pulsatile tinnitus in our case, but the cause of the right trochlear nerve paresis was not detected at the second admission. The consecutive angiographic changes from the uncertain onset of trochlear nerve paresis to the appearance of pulsatile tinnitus might reflect the entire process from the beginning of the dural AVF to its development and disappearance. Unfortunately cerebral angiography was not performed at the onset, so we cannot more precisely analyze the natural history of the dural AVF. However, we could observe the changes in arterial supply and abnormal venous flow of the dural AVF together with the changes in clinical symptoms. Reduction of shunt flow from the external carotid artery was observed at the second angiography, and this change in the flow dynamic might be important in the process of spontaneous obliteration of dural AVF.

Two mechanisms for the spontaneous closure of dural AVFs have been proposed, sinus thrombosis and intracranial hemorrhage. The influence of sinus thrombosis on spontaneous dural AVF closure is supported by angiographic findings in three patients.2,11,15) Turbulent arterial blood flow induced by atherosclerosis might promote thrombosis on the venous side of the dural AVF or sinus.3,11) Temporaroy occlusion of the arterial supply by the maneuver technique is often used as a treatment for dural AVF, and repeated temporary occlusion of the occipital artery resulted in improvement in 5 of 9 cases of transverse dural AVF.6) These results suggest that temporary stagnation of blood flow might promote the spontaneous occlusion of dural AVFs. An interesting theory for the development of dural AVFs is based on the findings that changes of the intradural multiple arterial channels affect the sinus thrombosis and promote the development of dural AVFs.13) Thrombosis in the sinus promoted the development of intradural vascular networks, resulting in arteriosinus communication and dural AVFs.5,13) Changes in intradural arterial flow might affect not only the development of dural AVFs, but also their spontaneous closure. Intradural arteriosinus channels might continually change and the development of AVFs caused by the increase of arterial shunts or spontaneous occlusion of dural AVFs could partly explain the natural history. In our case, we could not detect sinus thrombosis either radiologically or symptomatically. Intraventricular hemorrhage might affect or be affected by any change in venous circulation. More careful follow up and detailed examination by cerebral angiography could reveal the presence of thrombosis and the marked change from the occurrence to disappearance of dural AVF.

The second mechanism for spontaneous dural AVF closure is related to intracranial hemorrhage. Hemorrhage could trigger these closures, whether linked to the hematoma-mediated mass effect or to secondary vasospasm of the feeding vessels.11) In our case, no hematoma-mediated mass effect or secondary vasospasm of the feeding vessels was detected and no hemorrhage was observed during the development and obliteration of the lesion. The presence of intracranial hematoma might not have had a compressive effect or caused an ischemic insult, and hematoma was unlikely to have affected the spontaneous closure of dural AVF.

The causes of the initial intraventricular hemorrhage and trochlear nerve paresis remain unclear in this case, but changes in arterial supply or venous circulation actively occurred throughout the process, and spontaneous closure was part of the natural history of this dural AVF, but dural AVF might recur so careful follow up is required.

References
8) Kataoka K, Taneda M: Angiographic disappearance


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