Venous Hypertension as the Cause of Intracranial Hypertension in Patients With Transverse Sinus Dural Arteriovenous Fistula

Rebekah M. Ahmed, MBBS(Hons), FRACP, Bryan Khoury, MBCh, FRANZCR, Mark Wilkinson, MBChB, FRANZCR, Geoffrey D. Parker, MBBS, FRANZCR, G. Michael Halmagyi, MD, FRACP

Abstract: We describe 2 patients with transverse sinus dural arteriovenous fistulas (DAVFs) who presented with headache and papilledema due to intracranial hypertension. It has been proposed, but never proven, that venous hypertension causes the intracranial hypertension in DAVF. The data from our patients support this hypothesis. An additional factor leading to intracranial hypertension could be stenosis of the fellow transverse sinus.


CASE REPORTS

Case 1
A 51-year-old man presented with a 4-month history of left pulsatile tinnitus. He had an easily palpable left occipital artery, a bruit over the left mastoid, and moderately severe, bilateral papilledema. Visual acuity was 20/20 in each eye, and visual fields demonstrated enlarged blind spots without peripheral field constriction. MRA and magnetic resonance venogram (MRV) with auto-triggered elliptic centric ordered (ATECO) sequences (8) (Fig. 1A) showed occlusion of the left transverse and sigmoid sinuses, with an associated DAVF; the occipital artery was dilated and there was subtle cortical venous reflux (Cognard grade IIB) (9). Arteriography (Fig. 1B) confirmed a DAVF of the left transverse sinus with arterial supply from a large transmastoid occipital branch of the external carotid artery, the left middle meningeal artery, the left internal carotid artery via the meningo-hypophyseal trunk, and from both vertebral arteries via suboccipital branches. The left superior sigmoid sinus was severely attenuated with multiple venous channels, suggesting previous thrombosis. Venous sinus pressure measurements were recorded before and after embolization (Table 1).

The patient underwent 2 arterial embolization procedures with Onyx. After the second, a left carotid arteriogram demonstrated no reflux into cortical veins (Cognard grade IIA).

Five months after embolization, the patient still had papilledema but no longer had tinnitus. Lumbar puncture showed an opening pressure of 350 mm H₂O with normal CSF contents. Arteriography showed no change in the DAVF. Venography demonstrated that the left transverse sinus was occluded, and there was a swollen arachnoid granulation in the right transverse sinus impairing venous flow.
Six months after arterial embolization, the DAVF was embolized via the right transverse sinus, with 11 hydrocoils placed in the stump of the left transverse sinus (Fig. 1C). Superior sagittal sinus and torcular pressure decreased to 29 mm Hg, and the pressure gradient across the arachnoid granulation of the right transverse sinus decreased to 5 mm Hg. The patient’s papilledema resolved.

The most recent venogram documented that venous sinus pressure remained stable with a superior sagittal sinus pressure of 27 mm Hg and a pressure gradient across the right transverse sinus of 7 mm Hg (Table 1). The patient remains well without headache or papilledema.

### Case 2

An 85-year-old woman with a long history of left pulsatile tinnitus was found to have papilledema. Her visual acuity was 20/30 in each eye, and visual fields showed enlarged blind spots but no peripheral constriction. MRV disclosed a DAVF draining into the left transverse sinus and distal occlusion of the left sigmoid sinus, with no filling of the left internal jugular vein.

Arteriography (Fig. 2A) demonstrated a DAVF involving the left transverse sinus with no cortical reflux, and drainage across the torcular into the right transverse sinus and right internal jugular vein (Cognard grade IIA). Arterial supply was predominantly via the left occipital and middle meningeal branches, with lesser supply via suboccipital and posterior

### TABLE 1. Venous Sinus Pressures Recorded Before and After Embolization of DAVF of Transverse Sinus

<table>
<thead>
<tr>
<th></th>
<th>Case 1</th>
<th>Case 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-embolization</td>
<td>Post-embolization</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 Month</td>
</tr>
<tr>
<td>ASSS</td>
<td>53</td>
<td>29</td>
</tr>
<tr>
<td>MSSS</td>
<td>54</td>
<td>29</td>
</tr>
<tr>
<td>PSSS</td>
<td>52</td>
<td>29</td>
</tr>
<tr>
<td>Torcular</td>
<td>52</td>
<td>29</td>
</tr>
<tr>
<td>PTS</td>
<td>52</td>
<td>28</td>
</tr>
<tr>
<td>Mid TS</td>
<td>47*</td>
<td>27*</td>
</tr>
<tr>
<td>ATS</td>
<td>42</td>
<td>22*</td>
</tr>
<tr>
<td>Sup. sig. sinus</td>
<td>22*</td>
<td>21</td>
</tr>
<tr>
<td>Inf. sig. sinus</td>
<td>19</td>
<td>21</td>
</tr>
<tr>
<td>Jugular bulb</td>
<td>19</td>
<td>21</td>
</tr>
<tr>
<td>Internal jugular</td>
<td>18</td>
<td>22</td>
</tr>
<tr>
<td>Right atrium</td>
<td>17</td>
<td>17</td>
</tr>
</tbody>
</table>

*All pressure measurements are expressed in millimeters of mercury.

*pGradient across stenosis.

ASSS, anterior superior sagittal sinus; ATS, anterior transverse sinus; MSSS, mid-superior sagittal sinus; PSSS, posterior superior sagittal sinus; PTS, posterior transverse sinus; sup. sig. sinus, superior sigmoid sinus; inf. sig. sinus, inferior sigmoid sinus.

meningeal branches of the vertebral arteries and meningo-hypophyseal tentorial branches. There was no drainage into the left sigmoid sinus or left jugular vein. Venography demonstrated a narrowing of the right transverse sinus together with rapid inflow of unopacified blood via the fistula. The narrowing was felt to be due to a combination of swelling of an arachnoid granulation and elevated intracranial pressure. Superior sagittal sinus pressure measured 41 mm Hg (Fig. 2B, 2C; Table 1), with an abrupt drop to 15 mm Hg across the narrowed segment of the right transverse sinus (Fig. 2C).

The DAVF was embolized with Onyx via a transarterial approach. Superior sagittal sinus pressure decreased to 21 mm Hg, and the pressure gradient across the stenosis in the right transverse sinus resolved (Table 1). The patient developed a small intracerebral occipital hemorrhage but made a full recovery. Papilledema and pulsatile tinnitus resolved.

Four months later, venography showed normal venous sinus pressures (Table 1) and narrowing of the right transverse sinus had improved (Fig. 2D).

**DISCUSSION**

It has been proposed that intracranial hypertension in the setting of a transverse sinus DAVF is the result of a decreased...
CSF absorption secondary to venous hypertension (3,4,10). Our 2 cases support this concept. In our first patient, after embolization, venous pressure remained mildly elevated due to the ongoing arterial supply to the DAVF and narrowing of the right transverse sinus. Yet, treatment was sufficient to resolve the patient’s tinnitus and papilledema. In our second patient, embolization not only terminated flow in the DAVF but also lessened stenosis in the contralateral transverse sinus. Again, the patient’s symptoms resolved as did the papilledema and the venous hypertension.

It has been shown that the lack of even one functioning transverse sinus may lead to venous hypertension and intracranial hypertension (7,11). We propose that this also might be true in transverse sinus DAVFs causing intracranial hypertension, given the findings in our 2 patients.

Adding to the increase in venous pressures was the flow of arterialized blood into the transverse sinuses, leading to further venous hypertension and intracranial hypertension through decreased CSF absorption at the arachnoid villi. By embolizing the DAVFs, the venous hypertension improved, as did the intracranial hypertension and papilledema.

We propose that if a patient has a DAVF of one transverse sinus and the other transverse sinus is functioning, significant venous hypertension and subsequent intracranial hypertension will not develop. Cognard et al (3) in their review of DAVFs leading to intracranial hypertension reported that only 4 of 13 patients had normal venous sinuses. However, the authors did not look for stenosis of the contralateral sinus nor obtain pressure gradient measurements. It was reported that all patients had abnormal cerebral venous flow.

DAVFs with reflux into cortical veins (Cognard type IIB) pose great risk for hemorrhage and focal neurological damage (5). We believe that a DAVF with anterograde flow (Cognard type I) and reflux into the affected sinus (Cognard type IIA) are also not “benign” and may cause venous hypertension with papilledema and potential vision loss. Patients having arteriography for investigation of DAVFs should be considered for venography and manometry. In patients where venous hypertension is identified, especially in the setting of only one functioning transverse sinus, a full ophthalmic evaluation should be performed, including visual acuity, visual fields, and funduscopy. Embolization should be considered if there is any indication of risk of visual loss.

ACKNOWLEDGMENT

Dr Steve Reddel referred the second patient.

REFERENCES