Borden type 3 superior sagittal sinus dural arteriovenous fistula, angiographic features and endovascular treatment.

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Purpose

Superior sagittal sinus dural arteriovenous fistulas (SSS-DAVFs) are relatively rare vascular lesions which represent approximately 4.6-14% of intracranial DAVFs \(^{(1-2,4)}\). Partial or complete occlusion of the SSS is a highly associated condition, and may be the precursor to the development of the SSS-DAVF \(^{(2)}\). Borden Type-3 DAVFs drain retrograde\(^{##}\) into cortical veins. Therefore, Borden Type-3 SSS-DAVFs are frequently associated with aggressive neurologic symptoms including cerebral hemorrhage, conscious disturbance, and dementia. Although SSS-DAVFs have been treated by several techniques, including open surgery, transvenous embolization, transarterial embolization and combinations thereof, only a few case series have been reported. Here, we describe angiographic features and results of the endovascular treatment of Borden type-3 SSS-DAVFs.

Methods and Materials

We retrospectively reviewed 6 consecutive patients with Borden type-3 SSS-DAVFs treated in our institutions between September 2002 and September 2012. The clinical findings of the six patients are summarized in Table 1. There were 3 males and 3 females, with ages ranging from 71 to 86 years (mean age, 78.0). Symptoms were headache (n=1), cognitive disorder (n=2), disturbance of consciousness (n=1), hemiparalysis (n=1), and cerebral hemorrhage (n=2). Two patients had no symptoms. One patient had a history of clipping for an anterior communicating artery aneurysm, and one patient had a history of transvenous embolization for bilateral transverse sigmoid sinus (TSS) DAVFs.

Table 1: Symptoms and past history

<table>
<thead>
<tr>
<th>Age/gender</th>
<th>Symptoms</th>
<th>Past history</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 86 F</td>
<td>cognitive disorder, ICH</td>
<td>post clipping for cerebral aneurysm</td>
</tr>
<tr>
<td>2 74 F</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>3 84 M</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>4 71 F</td>
<td>cognitive disorder, headache</td>
<td>none</td>
</tr>
<tr>
<td>5 78 M</td>
<td>hemiparalysis, ICH</td>
<td>none</td>
</tr>
</tbody>
</table>
ICH, intracranial hemorrhage; TVE, transvenous embolization;
CSH, chronic subdural hematoma;
TSS-DAVF, transverse-sigmoid sinus dural arteriovenous fistula

All patients underwent selective cerebral angiography including biplane angiography and rotational angiography before endovascular treatment. Angiographic features were reviewed by two neuroradiologists with a special interest in the main feeding arteries, the presence of shunted pouch, drainage routes of the DAVFs, cerebral venous drainage from the affected areas of the brain, restricted changes of the other venous sinuses, and the coexistence of DAVFs in other locations. A shunted pouch was defined as a tubular or elliptical vascular structure that is separated from the main sinus lumen into which multiple feeding arteries converge and continue to the SSS.

All patients underwent endovascular treatment under local anesthesia. Selection of endovascular techniques was decided based on the angiographic features relating to occlusive changes in the other sinuses, cerebral venous drainage from the affected areas of the brain, accessibility to the shunted pouches, and the feeding arteries. Transarterial embolization was performed with a triple coaxial system (1.9F / 2.7F microcatheters and 5F guiding sheath, or 1.5F microcatheter/ 4F support catheter/ 5F guiding sheath) via a femoral arterial approach, while transvenous embolization was performed with a triple coaxial system (1.7 or 1.9F microcatheter and 4F support catheter) through 5F guiding sheath via a femoral venous approach. After endovascular treatment, all patients were followed up with angiography and/or MRA. We reviewed the angiographic results immediately after treatment, the complications, and the follow-up results.

Results

1. Angiographic features.

Angiographic features of the six patients are summarized in Table 2. The paramedian branches of the middle meningeal artery and the frontal and parietal branches of the superficial temporal artery supplied the SSS-DAVF bilaterally in all patients. The anterior falx arteries, which originate from the ophthalmic artery, supplied the SSS-DAVF in
4 patients, and the transosseous branches of the occipital artery supplied the SSS-DAVF in 2 patients. Shunted pouches were found in all 6 patients, which were located superolaterally or superomedially to the main lumen of the SSS. In all cases, the SSSs were occluded and the shunted blood drained retrogradely via the cortical venous anastomosis into the cavernous sinus and/or the transverse sinus, the posterior part of the SSS, and the inferior sagittal sinus. Cerebral venous blood from the affected areas of the brain drained via cortical venous anastomoses with significant congestion was seen in 4 patients. In the remaining 2 patients, cerebral venous blood from the affected areas of the brain partially drained via the isolated SSS into the epidural veins and/or emissary vein.

All cases showed occlusion of the SSS, and 3 cases showed bilateral TSS occlusion. 3 patients showed coexistence of DAVFs in other locations, namely the cavernous sinus (CS) in 1 patient, the TSS in 1 patient, and both CS and TSS in 1 patient.

Table 2: Angiographic features

<table>
<thead>
<tr>
<th>Feeder</th>
<th>Normal cerebral venous drainage via affected SSS</th>
<th>Patency of the other sinus</th>
<th>Coexistence of dAVF in other locations</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 MMA, anterior falx artery</td>
<td>no</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>2 MMA, STA, anterior falx artery</td>
<td>no</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>3 MMA, STA, anterior falx artery</td>
<td>yes</td>
<td>no</td>
<td>no</td>
</tr>
<tr>
<td>4 MMA, STA, anterior falx artery</td>
<td>no</td>
<td>yes</td>
<td>lt.CS</td>
</tr>
<tr>
<td>5 MMA, STA</td>
<td>no</td>
<td>no</td>
<td>lt.CS,lt.TS</td>
</tr>
<tr>
<td>6 MMA, STA</td>
<td>yes</td>
<td>no</td>
<td>rt.SS, bil.TS</td>
</tr>
</tbody>
</table>

MMA, middle meningeal artery; STA, superior temporal artery;
CS, cavernous sinus; TS, transverse-sigmoid sinus;
SS, sigmoid sinus;
2. Endovascular treatment.

The techniques and treatment results of the six patients are summarized in Table 3. Coil embolization of the affected sinus or fistulous pouch was performed in 4 cases wherein the isolated sinus was not a normal cerebral venous drainage route. Sinus packing was performed in three patients via a transvenous approach through the occluded sinus (n=2), or via a transarterial approach (n=1). One patient underwent selective transvenous embolization of the shunted pouch combined with transarterial embolization. The SSS-DAVF disappeared immediately after treatment in 4 patients.

In 2 cases wherein the affected sinus received normal cerebral venous drainage, selective transvenous embolization of the shunted pouches was initially attempted but failed due to poor maneuverability of the microcatheter/microguidewire caused by the occlusion of the transverse/sigmoid sinuses in addition to the SSS occlusion. These 2 patients were then treated by transarterial embolization of the shunted pouches using a mixture of diluted (17%-20%) n-butyl-2-cyanoacrylate (NBCA) and lipiodol. Angiography immediately after treatment showed that the SSS-DAVF had disappeared in 1 patient, and was minimally residual in the other patient.

No complications were observed during and after the treatment in all patients. Follow-up angiography and/or MRA showed no findings suggesting recurrence or reaggravation for 4~42 months (mean 13.2 months).

Table3: Treatments results

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Angiographic results</th>
<th>Complications</th>
<th>Recurrence/aggravation (follow-up months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Selective TVE + TAE (NBCA)</td>
<td>disappeared</td>
<td>no</td>
<td>no (12months)</td>
</tr>
<tr>
<td>2 Sinus packing (transvenous)</td>
<td>disappeared</td>
<td>no</td>
<td>no (44months)</td>
</tr>
<tr>
<td>3 TAE(NBCA)</td>
<td>markedly regressed</td>
<td>no</td>
<td>no (4months)</td>
</tr>
<tr>
<td>4 Sinus packing (transarterial)</td>
<td>disappeared</td>
<td>no</td>
<td>no (6months)</td>
</tr>
<tr>
<td>5 Sinus packing (transvenous)</td>
<td>disappeared</td>
<td>no</td>
<td>no (30months)</td>
</tr>
<tr>
<td>6 TAE(NBCA)</td>
<td>disappeared</td>
<td>no</td>
<td>no (8months)</td>
</tr>
</tbody>
</table>

TAE, transarterial embolization; TVE, transvenous embolization;
Fig. 1: SSS-DAVF treated by transvenous sinus packing (Case 5) a) Lateral view of left external carotid angiogram shows an SSS-DAVF with SSS occlusion. The SSS DAVFs are supplied by the left middle meningeal artery and superior temporal artery, which drains retrogradely into the cortical veins. b) Lateral view of left internal carotid angiogram at arterial phase shows that the left anterior falx artery supplied the SSS-DAVF. c) Lateral view of left internal carotid angiogram at venous phase shows venous congestion at the frontal area. The affected SSS (anterior portion of the SSS) does not work as a drainage routes of the cerebral venous blood. d) Frontal view of left common carotid angiogram shows coexistence of lt.TSS-DAVF. e) Fluoroscopic image during transvenous embolization shows coils packed in the isolated SSS introduced via a microcatheter advanced through the occluded portion of the SSS. f) Lateral view of left external carotid angiogram after transvenous embolization shows disappearance of the DAVF.
**Fig. 2:** SSS-DAVFs developed following clipping operation of the cerebral artery aneurysm, which were treated by selective transvenous embolization combined with transarterial embolization using NBCA. 

a) Frontal view of right external carotid angiogram shows SSS-DAVFs with cortical venous reflux and SSS occlusion. Anterior branches of the right middle meningeal artery supplied SSS-DAVF and were converged to a shunted pouch located on left side of the SSS (arrow). 

b) Frontal view of left external carotid angiogram shows that anterior branches of the left middle meningeal artery supplied the SSS-DAVF and converged to another shunted pouch (arrow). 

c) Lateral view of right internal carotid angiogram shows that there was no normal cerebral venous drainage via the affected sinus. 

d) Fluoroscopic image during treatment showed coils were selectively placed into the shunted pouches via a microcatheter that had been advanced through the occluded SSS. 

e) Frontal view of selective angiography of the left middle meningeal artery after selective transvenous embolization shows residual arteriovenous shunts through the coil mesh. 

f) Digital subtraction angiogram obtained during the injection of 25% NBCA shows NBCA-lipiodol mixture fills into the shunted pouch. 

g) Lateral view of left external carotid angiogram post transarterial embolization shows disappearance of the DAVFs. 

h) Lateral view of left external carotid angiogram post treatment shows the preservation of normal cerebral venous drainage.
Fig. 3: SSS-DAVs developed after transvenous coil embolization of bilateral transverse-sigmoid sinus DAVFs. The SSS DAVFs were treated by transarterial embolization using NBCA. (Case 6) a, b) Lateral view (a) and frontal view (b) of right external carotid angiography show SSS-DAVs with SSS occlusion. The SSS-DAVs are fed by right middle meningeal artery and superficial temporal arteries, and they drain retrogradely into the cortical veins. c, d) Frontal view (c) and lateral view (d) of right internal carotid angiography show blood flow from the normal cerebral vein drained via the affected SSS into the emissary or epidural veins (arrow). Selective transvenous embolization was difficult because of the occlusion of bilateral TSS, and transarterial embolization using NBCA was performed.
Fig. 4: SSS-DAVs developed after transvenous coil embolization of bilateral transverse-sigmoid sinus DAVFs, The SSS DAVFs were treated by transarterial embolization using NBCA. (Case 6) e) Frontal view of selective angiography of right middle meningeal artery shows a shunted pouch fed by small multiple branches. Transarterial embolization with 20% NBCA-lipiodol mixture was subsequently performed. f) Fluoroscopic image after transarterial embolization NBCA cast in the feeding artery and the shunted pouches. g) Lateral view of right external carotid angiogram after transarterial embolization shows disappearance of the SSS DAVF. h, i) Frontal (h) and Lateral (i) view of right internal carotid angiography after transarterial embolization show the preservation of the normal cerebral venous drainage via the anterior part of the SSS.
Conclusion

Halbach\(^{(2)}\) et al. reported that SSS-DAVs are relatively rare (11% in their series), and are highly associated with head trauma and sinus thrombosis. Thrombus formation due to several causes such as operation and trauma can cause proliferation of vessels at the venous sinus wall and arteriovenous shunts. However, sinus thrombosis was observed less frequently in histopathologic examinations\(^{(5)}\). A histopathologic study by Nishijima et al.\(^{(6)}\) showed that arteriovenous fistulas were located in the dural sinus wall and/or the adjacent dura mater. Restrictive changes in the sinuses were mainly due to the intimal thickening of sinuses and the development of abnormal vascular networks within the sinus walls. Terada et al.\(^{(7)}\) showed venous hypertension can cause DAVFs in an animal model. A few studies\(^{(8-9)}\) suggested that angiogenic factors contribute to the cause and progression of DAVFs. In our cases, 2 cases had previous open head surgery and trauma, 2 cases showed coexistence of DAVFs in other locations, and 1 patient had a history of transvenous embolization of the bilateral TSS-DAVFs. This would suggest that SSS-DAVF can be caused by angiogenesis promoted by venous hypertension and/or traumatic events.

Bavinzski\(^{(4)}\), et al. reported that SSS-DAVFs were supplied by the middle meningeal artery, the occipital artery, the superficial temporal artery, and the anterior falx artery, which is in line with our cases. Some studies\(^{(4)}\) reported that SSS-DAVFs were also supplied by the middle cerebral artery in addition to the branches of the external carotid artery. Careful attention should be paid to transarterial embolization in cases where DAVFs are supplied by the cortical branch of internal carotid artery because embolic materials injected from the external carotid artery may migrate into the cortical branches. Regarding the shunted pouches, some authors\(^{(10-12)}\) described cases of selective transvenous embolization of shunted pouches for the treatment of DAVFs including SSS-DAVF. Piske et al.\(^{(13)}\) demonstrated a dural sinus compartment (shunted pouches) in 12 of 40 cases (30%) of DAVFs. In our 6 cases, at least one shunted pouch can be identified, and 1 case could be successfully treated by selective transvenous embolization of the shunted venous pouch combined with transarterial embolization with NBCA.

Normal cerebral venous drainage is important to determine whether the affected sinus can be sacrificed. It is generally thought that occlusion of the affected sinus with coils or liquid embolic materials can be acceptable for cases of DAVF involving an isolated sinus (Borden type-3) because the isolated sinus does not work as a drainage route for normal cerebral venous blood in most cases. However, 2 cases in our series showed that cerebral venous blood from the brain partially drained via the isolated SSS into
the epidural veins and/or emissary vein. Sinus packing for these cases may cause disturbance of normal cerebral venous return resulting in venous infarction even if they are Borden type-3 DAVFs. Therefore, selective transvenous embolization or transarterial embolization of the shunted pouch with liquid embolic materials while preserving the main sinus lumen would be more preferable for the treatment of such cases.

In conclusion, SSS-DAVFs are highly associated with the coexistence of DAVFs in other locations. Borden type-3 SSS-DAVFs could be successfully treated by endovascular techniques selected based on angioarchitecture and hemodynamics.

References


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