Postoperative reversible deterioration in a spinal dural arteriovenous fistula

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This 61-year-old man had a one-year history of progressively worsening gait disturbance, dysesthesia in both legs and urinary retention. On admission, neurological examination revealed diminished strength in both legs involving the proximal and distal muscles; manual muscle testing (MMT) returned a score of 4/5. His response to tactile and pinprick stimulation was reduced bilaterally at the level of Th10. His gait was spastic, ataxic and broad-based. His lower extremities were hyper-reflexic; Babinski reflex was positive on the left. Magnetic resonance imaging (MRI) revealed abnormal flow voids dorsal to the spinal cord in the middle thoracic and lumbar subarachnoid space and edema in the cord [Figure 1A]. T1-weighted images showed abnormal heterogeneous enhancement [Figure 1B]. Spinal arteriography revealed an SDAVF fed by the left sixth intercostal artery with dorsal PD in the caudal direction [Figure 2]. Electromyography disclosed bilateral reduced motor unit potentials in the proximal leg muscles, absence of the patellar deep tendon reflex and thigh pain. No radiological findings explaining this deterioration were obtained. He was treated conservatively and all segmental symptoms and signs subsided by the fifth postoperative day. Although the precise mechanisms underlying the dramatic but often reversible deterioration after radical SDAVF treatment remain to be determined, we postulate that this was attributable to postoperative segmental venous hemodynamic changes based on the neurological changes.

Key words: Complication, hemodynamics, spinal dural arteriovenous fistula, surgery

Spinal dural arteriovenous fistulas (SDAVFs), also known as Type I spinal arteriovenous malformations, are treated by endovascular embolization or by surgical division of the draining pathway that connects with the perimedullary drainage (PD) adjacent to the fistula. Favorable treatment results have been shown in both modalities and surgery seems to be superior to embolization because of a high recurrence rate in embolization.[1-3] Known potential complications are migration of embolic material and cerebrospinal fluid leakage.[1,2] In both modalities, however, few patients experienced delayed transient post-treatment deterioration thought to be the result of impaired venous drainage.[4,5] We present a patient who suffered from this complication and discuss the underlying pathophysiological mechanism.

Case Report

This 61-year-old man presented with weakness and sensory disturbance in the legs. There was a spinal dural arteriovenous fistula (SDAVF) fed by the left sixth intercostal artery with dorsal perimedullary drainage. Surgical division of the perimedullary drainage led to rapid neurological improvement. However, on the second postoperative day he experienced transient deterioration of second neuron function in the left upper lumbar segment resulting in motor weakness of the proximal leg muscles, absence of the patellar deep tendon reflex and thigh pain. No radiological findings explaining this deterioration were obtained. He was treated conservatively and all segmental symptoms and signs subsided by the fifth postoperative day. Although the precise mechanisms underlying the dramatic but often reversible deterioration after radical SDAVF treatment remain to be determined, we postulate that this was attributable to postoperative segmental venous hemodynamic changes based on the neurological changes.

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Figure 1: A. Preoperative T2-weighted MRI showing flow voids dorsal to the edematous cord. B. Preoperative enhanced T1-weighted MRI discloses heterogeneous enhancement of the cord. Dominant enhancement on the left side at the Th10/11 level (inset) is shown. C. T2-weighted MRI obtained two days after postoperative deterioration shows disappearance of the flow void and improvement of the edema in the cord.
quadriceps femoris, tibialis anterior and gastrocnemius. Upon Th5 and Th6 laminectomy we found an SDAVF in the dorsal aspect of the left sixth dural root sleeve. A dilated PD from the fistula ascended, formed a hairpin curve and then descended [Figure 2]. We divided the PD just distal to the fistula. After PD division, the arterial spectrum of the draining vein disappeared on Doppler ultrasonographs. The procedure produced no changes in the somatosensory evoked potential. At the end of the surgery, the draining vein had lost its tension and redness.

Postoperatively, his motor and sensory symptoms were clearly improved. However, on the second postoperative day he complained of difficulty raising his left knee [Figure 3]. There was weakness of the left iliopsoas and quadriceps femoris, MMT was 3/5, he manifested no patellar tendon reflex and there was dysesthesia in the frontal aspect of the thigh. On the other hand, he manifested full strength of the bilateral distal muscles (tibialis anterior, digital flexors and extensors and gastrocnemius), his Achilles tendon reflex was unchanged. The MRI performed two days after deterioration onset showed disappearance of the flow void in the dilated PD and improvement of the cord edema [Figure 1C]. On T1-weighted images there was no high-intensity signal suggestive of venous thrombosis. Additional spinal arteriography was refused. He received 8mg of betamethasone/day for three days, IV; all segmental symptoms and signs subsided by the fifth postoperative day.

At one year after the operation, the muscle strength and deep tendon reflexes in both legs were normal, however, dysesthesia in the distal portion persisted.

**Discussion**

Although no MRI findings explaining the deterioration were observed, neurological deterioration limited to the proximal part of the left leg suggested that the spinal second neuron in the left upper lumbar segment was involved in a postoperative hemodynamic event.

It was suggested that collateral venous pathways developed in the area irrigated by the descending PD to compensate for the existing cord congestion [Figure 3 bottom left]. Surgical division of the PD stopped the high-pressure arterial blood flow overload into the descending PD, a pathological form of the posterior spinal vein, resulting in the re-acquisition of the ascending flow in the normal direction of venous return [Figure 3 bottom center]. This hypothesis is supported by the following considerations. Anatomically, normal venous drainage at the middle thoracic level can be in the ascending direction.[6]

The cephalad venous channel at the tip of the PD hairpin curve we observed intraoperatively [Figure 2, inset] may have functioned as an ascending vein. The direction of the PD, cephalad or caudal, can change after surgical treatment.[7] Intraoperative hemodynamic study has shown that the pressure in the PD of an SDAVF decreased to normal venous levels following division.[8] Decreased venous blood inflow results in stagnation and insufficient venous drainage in limited areas [Figure 3 bottom center]. Histological arterialization of the PD, i.e. thickening of the wall with abundant elastic fibers induced by a continuous arterial pressure load,[9] resulting in poor vessel compliance, may lead to stagnation. Focal blood brain barrier (BBB) disruption due to venous congestion, demonstrated as abnormal...
gadolinium enhancement of the cord\textsuperscript{[10]} may have contributed to our patient's postoperative deterioration. In fact, BBB disruption dominant on the left side at the Th11/12 level [Figure 1B, inset] corresponded well with our findings of postoperative neurological deterioration in the left upper lumbar segment. We posit that upon the development of sufficient ascending venous drainage, the stagnated flow may have returned to normal levels, resulting in his recovery [Figure 3 bottom right].

Most previously reported series of SDAVF, consisting of six to 70 patients treated by direct or endovascular surgery, had favorable outcomes.\textsuperscript{[1]} The reported incidence of post-treatment deterioration is 7.7-8.3%.\textsuperscript{[4,5]} The interval between treatment and deterioration ranged from one to three days and improvement was observed several weeks after the conservative treatments [Table 1].

The degree of recovery varied from full recovery to stabilization at the new segmental level. Others speculated that a sudden decrease in the venous load resulted in perimedullary or intramedullary venous thrombosis.\textsuperscript{[4,5]} We have treated 12 patients by simple division of the PD; of these, only the present patient experienced postoperative deterioration. Thus, at 8.3%, our incidence of post-treatment deterioration is comparable to previously reported series.

The precise mechanisms underlying such an event remain to be determined. Awareness of the risk for deterioration makes it possible to obtain the proper informed consent for treatment and allows the planning of perioperative management.

### Table 1: Details of cases of SDAVF deteriorated after treatment

<table>
<thead>
<tr>
<th>Author (year)</th>
<th>Age/sex</th>
<th>Feeder</th>
<th>Symptomatic duration</th>
<th>Treatment</th>
<th>Post-treatment deterioration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Logue (1979)</td>
<td>65/M</td>
<td>ND</td>
<td>18 months surgery</td>
<td>3 days</td>
<td></td>
</tr>
<tr>
<td>Logue (1979)</td>
<td>55/M</td>
<td>ND</td>
<td>18 months surgery</td>
<td>3 days</td>
<td></td>
</tr>
<tr>
<td>Ushikoshi (1999)</td>
<td>63/M</td>
<td>Rt. L2</td>
<td>9 months ES</td>
<td>1 day</td>
<td></td>
</tr>
<tr>
<td>Present case</td>
<td>61/M</td>
<td>Lt. Th6</td>
<td>1 year surgery</td>
<td>2 days</td>
<td></td>
</tr>
</tbody>
</table>

*Period after the treatment. ES: Endovascular surgery, M: Mannitol, ND: No description, S: Steroid.

### References


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