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Case Report

A case of symptomatic spinal dural arteriovenous fistula after high-volume lumbar puncture

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Abstract

Background: Spinal dural arteriovenous fistulas (DAVFs) are rare lesions that lead to venous congestion and ischemic injury resulting in neurologic deterioration. Here we present a patient diagnosed with glioblastoma multiforme (GBM) who became symptomatic from a spinal DAVF after a diagnostic high-volume lumbar puncture (LP).

Case Description: When a 72-year-old female developed partial seizures in her left upper extremity without other focal neurological deficits, she underwent a magnetic resonance imaging (MRI) scan of the brain. The MRI revealed a right frontal/posterior corpus callosal lesion. She next had a MR-guided high-volume LP. A GBM was diagnosed following a biopsy. Postoperatively, after the LP, she was noted to have bilateral deltoid and bilateral 4/5 lower extremity weakness, with diffuse hyperreflexia. The MRI and magnetic resonance angiogram (MRA) of the cervical spine demonstrated a large venous varix at the C5-C6 level within the left neural foramen. She underwent successful complete embolization of two thyrocervical branches with direct communication to an enlarged anterior spinal artery. One month later, her neurological examination returned to baseline; she was walking independently with only 4+/5 residual weakness in her left lower extremity.

Conclusions: Here we report a patient with a cranial GBM and an incidental cervical spinal C5-C6 DAVF that became symptomatic after a high-volume LP. It is possible that the high-volume LP increased vascular congestion, thus precipitating the onset of cervical myelopathy.

Key Words: Arteriovenous malformation, dural arteriovenous fistula, glioblastoma multiforme, lumbar puncture, magnetic resonance angiography, magnetic resonance imaging

INTRODUCTION

Dural arteriovenous fistulas (DAVFs) are the most common vascular malformations of the spinal cord (1 out of 100,000 per year). Initial neurological symptoms include gait imbalance, numbness, and paresthesias.[1] Most are located at the thoracolumbar junction.[7] Some present with a progressive myelopathy attributed to an increase in venous congestion.[5,13]
An acute decrease in cerebrospinal fluid (CSF) pressure attributed to a lumbar puncture (LP) may lead to the sudden engorgement of dural and epidural veins, resulting in medullary/cord ischemia. Here we present a patient with a cranial glioblastoma multiforme (GBM) whose cervical spinal DAVF became symptomatic after a diagnostic high-volume LP.

**CASE DESCRIPTION**

A 72-year-old female presented with partial seizures in her left upper extremity, but no other focal neurological deficit. Magnetic resonance imaging (MRI) of the brain revealed a right frontal/posterior corpus callosal enhancing lesion involving the internal capsule and crossing the midline [Figure 1]. GBM was diagnosed after biopsy. Her workup next included an MR-guided high-volume LP following which she immediately developed bilateral deltoid and 4/5 lower extremity weakness with diffuse hyperreflexia. The subsequent cervical MRI and magnetic resonance angiogram (MRA) demonstrated a large venous varix (DAVF) at the C5-C6 level extending into the left neural foramen [Figures 2 and 3]. She underwent successful complete embolization of two thyrocervical branches with direct communication to an enlarged anterior spinal artery; no residual feeders were noted [Figures 4 and 5]. She was discharged to rehabilitation with no worsening of her strength. One month later, she was back to her original neurological baseline, and was walking independently with only mild residual 4+/5 left lower extremity weakness.

**DISCUSSION**

This case highlights the emergence of a subacute neurological deficit following a high-volume LP in a...
Table 1: Cases of dural arteriovenous fistulas (DAVFs) post high-volume lumbar puncture (LP)

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<thead>
<tr>
<th>Author</th>
<th>Spinal level, etc</th>
<th>Presentation post-LP</th>
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</tr>
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<tbody>
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<td>Roullet et al., 1988</td>
<td>T6, T7</td>
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<td>One patient remained paraplegic after operative intervention, one patient resolved paraplegia after embolization</td>
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Figure 5: In the delayed phase minimal venous drainage into the varices is present, filled from tiny collaterals too small to individually catheterize

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patient with a cranial GBM. Both the MRI and MRA of the cervical spine ultimately demonstrated a large venous varix in the left neural foramen at the C5-C6 level. The most likely etiology of this patient’s myelopathy was acute/subacute increased vascular congestion of the DAVF following the high-volume LP.

This phenomenon has been previously reported with thoracic arteriovenous malformations (AVMs). Roullet et al. described a patient whose T6/7 AVM became symptomatic after LP,[12] one patient remained paraplegic after surgical intervention, and the other’s paraplegia resolved after embolization. Awad et al. described a patient with a T5/6 AVM who, after surgical treatment was antigravity in all muscle groups on postoperative day 10.[1] Aloui-Kasbi et al. described a 1-year-old patient who remained paraplegic after treatment.[1] Koerts et al. reported in 2013 of an AVM at the L2/3 level.[7] The patient made a full motor recovery with an ataxic gait requiring a cane and experienced some continued hyperalgesia [Table 1].

DAVFs involve a single feeding artery connected to intradural veins. The decrease in CSF pressure caused by the LP may have exacerbated vascular congestion, as also documented by Monro-Kellie leading to focal hyperemia.[8-10] Nonhemorrhagic subacute or acute myelopathy may occur as a complication of DAVF (Foix-Alajouanine syndrome). This is attributed to the evolution of a necrotic myelopathy due to spinal vein thrombosis resulting from AVM.

The diagnosis of DAVFs can be established with MRI and confirmed with MRA-angiography.[14] Two key MR features include enlargement of the spinal cord at the level of the DAVF lesion and a hyperintense signal on T2-weighted images. Angiography as a confirmatory tool is indicated if T2 hyperintensity and flow voids are present.[6]

CONCLUSION

Here we report a patient with a cranial GBM and an incidental cervical spinal DAVF (C5-C6) that became symptomatic after a high-volume LP. Likely, the high-volume LP caused an increase in vascular congestion thus precipitating her cervical myelopathy.

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Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES