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L A Heier and B C Lee

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A Dural Spinal Arteriovenous Malformation with Epidural Venous Drainage: A Case Report

Linda A. Heier[1] and Benjamin C. P. Lee

Thoracolumbar spinal angiomas are typically fed by inter-costal or lumbar arteries [1] and are drained exclusively by perimedullary veins [2]. Rarely, internal iliac artery branches may supply a spinal arteriovenous malformation (AVM). Epidural venous drainage has not been reported [2–4]. Only eight cases of spinal AVMs fed by internal iliac branches have been described in the literature [2, 5–11]. We report a lumbosacral AVM supplied by a lateral sacral artery and drained by left epidural veins that was successfully treated by isobutyl 2-cyanoacrylate (bucrylate) embolization. The unique venous drainage of this AVM raises controversial questions about the pathophysiology of the spinal angiomas.

Case Report

A 78-year-old man presented with a 4-year history of progressive leg weakness and loss of bladder control. Physical examination revealed wasting of the lower extremities, more marked on the left, with weakness involving extension and flexion of the left foot. The left hallux valgus had marked weakness as did the left hamstring. Deep tendon reflexes were hyperactive at both knees, brisk at the right ankle, and absent at the left ankle. The inferior and middle superficial abdominal reflexes on the left were absent. Sensory findings consisted of a zone of hypalgesia in the right perianal area. A left paraspinal bruit was heard at the level of L4.

A myelogram revealed serpiginous filling defects compatible with a midline dilated vessel extending from L5 to T6 (Fig. 1). Selective spinal angiography demonstrated that the artery of Adamkiewicz arose from the T11 intercostal artery. A retromedullary cauda equina AVM with no angiomatous mass was supplied by the right lateral sacral artery. Large left epidural draining veins were identified (Fig. 2).

Embolization of the AVM was performed with 0.2 ml of bucrylate diluted with an equal amount of Pantopaque contrast medium injected through a 3-French polyethylene catheter under fluoroscopic control. The malformation was successfully occluded (Fig. 3).

The left paraspinal bruit was no longer heard and the patient's absent left ankle jerk returned. There was progressive improvement in the patient's ambulation up to 8 months postembolization, but there was no change in bladder function.

Discussion

Spinal AMVs are divided angiographically into retromedullary and intramedullary lesions supplied by posterior and anterior spinal arteries, respectively [1]. Retromedullary malformations typically present as progressive neurological deficits in elderly men, while intramedullary lesions are characteristically seen in young patients who often have subarachnoid hemorrhage [1].

Retromedullary AVMs are supplied by posterior spinal arteries originating from the vertebral, deep cervical, intercostal, or lumbar arteries depending on the level of the lesion. Occasionally, anterior and posterior spinal arteries will anastomose with medullary branches from lateral sacral or other hypogastric arteries at the level of the cauda equina or conus medullaris [1, 12]. This anatomic variation allows an internal iliac branch to supply a spinal angioma. When an AVM is suspected on myelography and is not demonstrated with routine angiography, selective internal iliac angiography is necessary.

In recent years the majority of "retromedullary" malformations have been discovered to be extradural. The fistula or nidus is on the outer surface of the dura with venous drainage piercing the dura and ascending in the perimedullary space [2–4]. This type of malformation was first described by Kendal and Logue as "spinal epidural malformations draining into intrathecal veins" [2]. Merland subsequently termed these lesions "radiculomeningeal arteriovenous fistulae," as angiotomography identified the feeding arteries to be radicular or meningeal [3]. Included in his series was a case with internal iliac supply. Our case is likely a dural (radiculomeningeal) AVM although we could not ascertain whether the feeding vessel was radicular or meningeal in origin, as angiotomography was not performed.

Aminoff et al. [13] suggested that the neurological deficits associated with spinal AVMs result from cord ischemia secondary to raised pressure in the medullary veins and capillaries rather than to ischemia caused by an arterial steal [10].
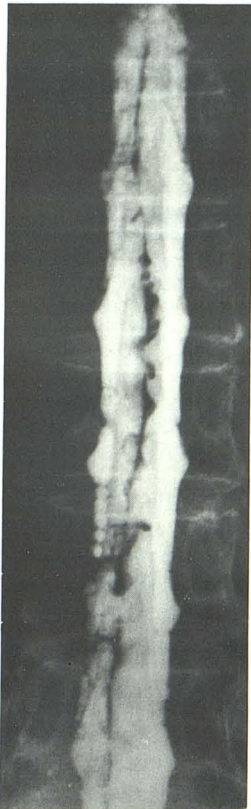
Fig. 1.—Pantopaque mye-
logram shows serpiginous
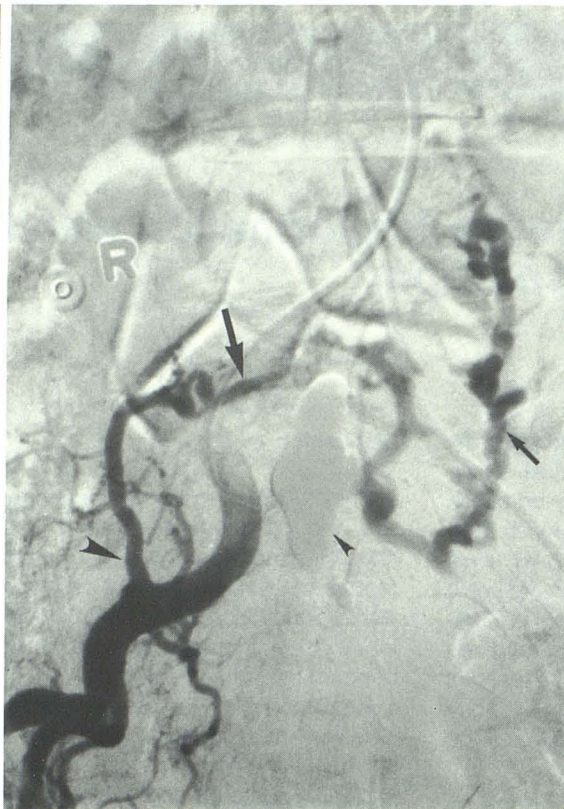filling defect compatible
with a dorsal draining vein.

Fig. 2.—Internal iliac angiogram (*large arrowhead* = right
lateral sacral artery; *small arrowhead* = pantopaque in distal
thecal sac; *small arrow* = left extradural intervertebral vein;
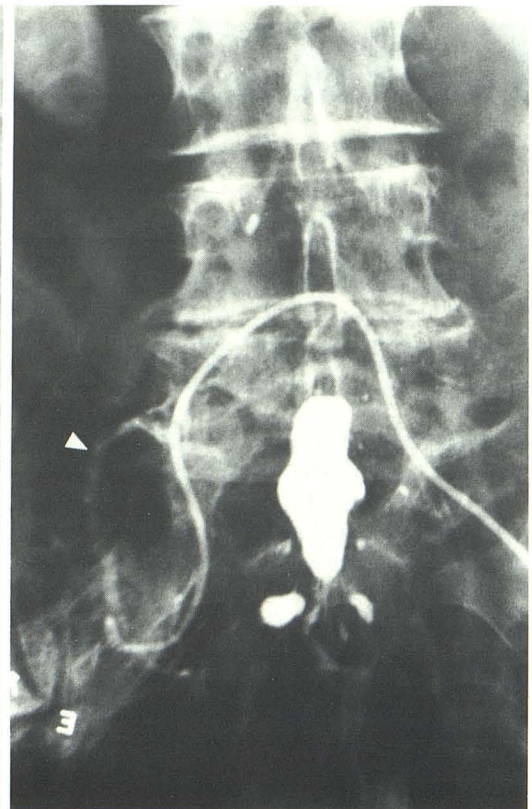*large arrow* = arteriovenous fistula).

Fig. 3.—Postembolization (*arrowhead* = bucrylate cast
of the arteriovenous malformation).

This explanation is likely in cases such as ours where the malformation is supplied solely by the right lateral sacral artery that does not provide any arterial supply to the cord. There is no connection between the lateral sacral artery and the spinal arterial system by which any steal could take place.

Thoracolumbar AVMs characteristically drain into dorsal veins that extend over a long length of cord distal to the nidus of the AVM (Fig. 1) [3, 9]. Why the venous drainage of the angioma ascends rather than decompresses into the extra-dural venous plexus via the abundant anterior and posterior medullary veins is not clear. One possible explanation is that AVMs are developmental vascular lesions that have a blood supply separate from the normal spinal cord vasculature [9]. However, if this is true, the theory that spinal cord dysfunction is caused by venous hypertension in medullary veins is im-plausible. How can there be raised pressure in the medullary veins if there is no connection with them to the arterialized coronal vein draining the AVM? Another possibility is that the usual medullary veins draining to the epidural space are absent and that the medullary venous drainage is confined to the arterialized coronal vein [3]. In this setting venous hyper-tension is likely.

Merland et al. [3] were unable to demonstrate any dorso-lumbar medullary veins draining into the epidural space in 13 cases of dural AVMs, even with anterior spinal artery injec-tions. In our case a dorsal coronal vein extending from L5 to T6 was demonstrated on myelography but not on the angio-gram. Instead, the angiogram showed a direct communication into the left extradural intervertebral veins. This suggests that the draining vein on the back of the cord was thrombosed. Clotted vessels have been seen in AVMs at surgery by several authors (9, 14). Perhaps the thrombosis of the coronal vein diverted venous drainage into the left extradural system. However, if angiomas are truly separate from the normal blood supply of the cord, or if the normal medullary venous drainage is supposedly absent, this should not have hap-pened.

Epidural venous drainage has not been described with any retromedullary spinal angioma to our knowledge. Although our case may be an isolated incident of communication with the epidural venous system, it supports the possibility that spinal AVMs may be acquired lesions. The dilatation of the left extradural veins may have caused root compression accounting for the left-sided symptoms in this patient.

This unique case illustrates some aspects of the controversial pathophysiology of spinal AVMs and reminds us not to forget the possibility of internal iliac artery supply.

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