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Dural Arteriovenous Malformation of the Major Venous Sinuses: An Acquired Lesion

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Arteriovenous malformations of the dura are thought to be congenital. However, arteriographic investigations of four patients who, after a head injury, developed dural arteriovenous fistulae with features of congenital malformations suggest that these abnormal communications may also be acquired. Thrombosis or thrombophlebitis in the dural sinus or vein may be the primary event in their formation. The pathogenesis is probably "growth" of the dural arteries normally present in the walls of the sinuses during the organization of an intraluminal thrombus. This may result in a direct communication between artery and vein or sinus, establishing an abnormal shunt. Ultimate fibrosis of the sinus wall and intraluminal thrombus may be the factors responsible for the spontaneous disappearance of such malformations.

Most dural arteriovenous malformations (AVMs) that involve the major venous sinuses present either spontaneously or as incidental findings during arteriography performed for other reasons. They occur predominantly in women over age 40 years [1]. The angiomatous network, multiple feeding arteries, numerous arteriovenous (AV) shunts, and occasional association with cerebral angiomas [2], as well as a few cases reported in children [3], suggest that these AVMs are congenital. Thrombosis of the draining sinus or vein is thought to be responsible for the occasional spontaneous disappearance of these lesions [4, 5].

Our experience with four patients who, after a head injury, developed dural AV fistulae with features of congenital malformations prompted a review of the literature and this report. In two of our four cases, transverse and sigmoid sinus abnormalities were demonstrated angiographically before the AV fistulae developed. In case 3, angiography 2 years earlier for unrelated reasons was normal; no AV shunt was seen. Although in case 4, there was no angiographic evidence of a dural fistula before head injury, the posttrauma onset of symptoms and the angiographic findings strongly suggested that this lesion had been acquired.

Cases 1 and 3 were studied by serial angiography with selective internal, external, and common carotid injections, while stereo magni
giography with selective and superselective catheterization of th
was performed in cases 2 and 4. The first three patients v
periodic angiography for up to 4 years, and the AV shunts we
spontaneously.

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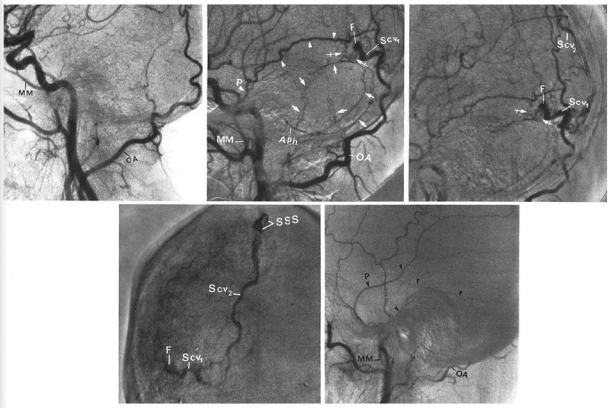
Case Reports

Case 1

A 56-year-old woman was examined for headaches and dizzy spell
raphy to rule out transient ischemic attacks was normal (figs 1A and
mild diabetes caused her headaches and dizzy spells to disappear.

She was brought to the emergency room after a head injury 2 mont
bruise over the right occipitomastoid area, was confused and disorien



Bulging Lumbar Intervertebral Disk: Myelographic Differentiation from Herniated Disk with Nerve Root Compression

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Deformities of the lateral margins of the contrast material-filled lumbar thecal sac are common findings at myelography in patients with low back pain, but not all such deformities are due to herniated disks. Differentiation at Amipaque myelography between a diffusely bulging disk (unlikely to cause nerve root compression) and a herniated disk (which typically causes nerve root compression) is based on the curvature and extent of the extradural deformity of the anterolateral margin of the contrast-filled sac and on the presence of fusiform widening of the most distal part of the affected nerve root. The deformity caused by a bulging disk is rounded, usually symmetrical (although occasionally more prominent on one side), and does not extend above or below the disk space; the nerve root is uniform in caliber and normal in size. The deformity caused by a herniated disk is angular and extends cephalad and/or caudal to the level of the disk space; the affected nerve root is usually widened in its most distal visible part. A consecutive series of 33 patients with clinically suspected lumbar disk herniation and no previous history of back surgery underwent laminectomy. Using the criteria listed above for differentiation of bulging from herniated disk on Amipaque myelography, the myelographic diagnosis was correct in all six operatively confirmed bulging disks and in 26 (96%) of 27 operatively verified disk herniations.

In 1934, Mixter and Barr [1] first described the clinical picture and anatomic findings of herniation of a lumbar intervertebral disk causing nerve root compression with resultant low back pain, sciatica, and weakness of the affected lower extremity. Only 2 years later, in another classical paper, Hampton and Robinson [2] described the myelographic criteria for recognizing this lesion using an oily contrast medium.

Deformities of the lateral margins of the contrast-filled lumbar thecal sac are a common finding at myelography in patients with low back pain, but not all such deformities are due to herniated disks [3–5]. Perhaps the most difficult task for the myelographer is to differentiate the extradural deformity produced by a bulging disk (unlikely to cause nerve root compression) from that produced by a herniated disk (which typically causes nerve root compression).

Amipaque (metrizamide, Winthrop), a nonionic water-soluble contrast medium for myelography, reliably provides delineation of the lumbar nerve roots both within the thecal sac and in the root sheaths [6, 7]. Myelography with Amipaque can directly demonstrate distal widening of a nerve root compressed by a herniated disk, thus permitting more accurate differentiation of disk herniation from disk bulging.

Materials and Methods

A consecutive series of 33 patients with clinically suspected lumbar intervertebral disk herniation and no previous history of back surgery underwent laminectomy. Amipaque lumbar myelography had been performed on all 33 shortly before laminectomy. The concentration of contrast medium introduced into the lumbar subarachnoid space varied