Dural Ectasia and Arachnoid Diverticula Causing Cauda Equina Syndrome in Long Standing Ankylosing Spondylitis

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Learning objectives

To illustrate the imaging findings of the Dural Ectasia causing Cauda Equina Syndrome as neurological complication in Ankylosing Spondylitis

Background

Neurological complications of ankylosing spondylitis (AS) are rare, occur in advanced stages of the disease, with a prevalence of 2.1% of patients (Figure 1).

Among the most frequent complications are including solitary nerve root lesions, spontaneous atlantoaxial subluxation with associated myelopathy, cauda equine compression secondary to lumbar spinal stenosis, and traumatic fractures of the fused vertebral column with nerve root of spinal cord compression.

Another complication that affects these patients is the cauda equina syndrome (CES), which is a rare neurological complication, described in 1961 by Bowie & Glasgow, and Hunge independently. Is a progressive loss of function of the spinal canal neurological structures below the termination of the spinal cord. It is associated with ectasia of the dural sac and arachnoidal diverticula formation, most often at the level of the lumbar spine, but there are reported cases with involvement of cervical and thoracic spine.

The most common initial neurological symptoms are sensory disturbances in lower limbs and perineum or rectal voiding dysfunction, pain and weakness in the lower extremities.

In the literature, several cases have been reported in which urinary symptoms, with debuts that most of these patients have been confused with symptoms derivates of prostatic hyperplasia, which underwent unnecessary prostatic resection, without any improvement of urinary symptoms.

Because in our institution we had a case of a patient with long standing Ankylosing Spondylitis who came to our center for study of cauda equina syndrome, we made research about this pathology. We describe the typical radiological findings of this rare complication of the AS, as well as the possible causes that lead to the widening of the dural sac and the development of a CES.
NEUROLOGICAL COMPLICATIONS OF THE ANKYLOSING SPONDYLITIS

• Medular compression caused by:
  – Traumatic fractures of the fused vertebral column
  – Atlantooccipital y atlantoaxial subluxation
  – Spinal canal stenosis
  – Pseudarthrosis
• Nerve root lesions
• Cauda equina syndrome (CES)

Fig. 1
Imaging findings OR Procedure details

We describe the typical radiologic findings of Dural Ectasia (DE) as rare neurological complication of the Ankylosing Spondylitis based on the different imaging techniques.

**Magnetic Resonance Imaging (MRI):**

- Typical findings of the AS.
- Changes related to the dural ectasia *(figure 2,3,4).*
  - Expansion of the spinal canal, dural ectasia sac at different levels of the spine, with arachnoidal diverticula formation with the same signal intensity to CSF and communicated with the dural sac.
  - Accumulation of nerve roots within the diverticula and adherence and convergence of the cauda equina within the arachnoid sac, causing a dural sac empty image, most noticeable on T2-weighted images.
  - Scalloping of the anterior aspect of the vertebral posterior elements (pedicles, laminae and spinous processes) that is characteristic of a long process of evolution. There are also some cases reported with erosions/scalloping in the posterior aspect of the vertebral bodies.

**Computerized Tomography (CT) :**

- Is more effective to show bone changes typical of AS.
- Changes related to the dural ectasia: *(figure 5,6,7,8)*
  - Widening of the lumbar spinal canal, with presence of erosions on the anterior aspect of the posterior elements of the vertebrae.
  - Scalloping on the anterior aspect of the laminae, pedicles and spinous processes.
  - Some cases have been reported with dural calcifications seen on CT.

**Plain radiographs of the spine: (figure 9,10)**

- Typical changes associates to AS.
- Changes related to the dural ectasia:
  - The lateral view may show widening of the spinal canal and bone erosions.

**Pathogenic mechanisms:**

The pathogenic mechanisms that lead to the expansion of the dural sac and cauda equina syndrome are unknown. Several hypotheses have been described:
• A chronic inflammation with arachnoiditis may lead to fibrosis of the dura mater, with adhesion of the meninges to vertebrae, which could compromise the reabsorption of CSF. In addition, fibrotic meninges may be unable to dampen brief CSF pressure fluctuations secondary to breathing and arterial pulsations. Therefore a combination of stagnation of the CSF, diminished meningeal elasticity and compliance, bone fragility secondary to osteoporosis spinal stiffness may lead to the widening of the dural sac and the diverticula formation. (Figure 11)

• According to another hypothesis, the pathogenesis of CES related to AS, may involve injuries of the apophyseal joints and enthesis, such as yellow and interspinous ligaments and joint capsules. These injuries can lead to epidural tissue inflammation, arachnoiditis, arachnoid thickening, meningeal fibrosis and adhesion of the arachnoid to the dura mater and periosteum.

Both hypotheses explain pathogenic dural sac dilation and diverticula formation but not the clinical presentation or the presence of atrophic demyelination of the nerve roots seen in autopsy studies. To explain the spinal cord and nerve roots lesion:

• Some authors postulate that is probably due to a mechanical narrowing and compression of the nerve roots (by dural ectasia, arachnoidal cysts or secondary to an arterial pulse wave); a reduced compliance of the dural sac, or secondary to demyelination due to ischemia.

• Another scenario described by Levine posits that the spinal cord and nerve roots lesion occurs secondary to arterial pulse wave transmitted to the LCR, in a dural sac with reduced elasticity due to chronic inflammation and adhesions. This theory would explain the beneficial effect of the lumboperitoneal bypass with reduction of the intradural pressure shocks.

Images for this section:
DURAL ECTASIA: RADIOLOGICAL FINDINGS

- Widening of the spinal canal
- Ectasia of the dural sac
- Arachnoidal diverticula
- Herniated nerve roots within the diverticula
- Displacement of the cauda equina within the dural sac
- Scalloping of the posterior wall of the vertebrae and the anterior aspect of the posterior elements.

Fig. 2
Fig. 3: MRI lumbar spine. Images A-C show the marked widening of the spinal canal and dural ectasia sac (opposite blue arrows in B). Note the shift of the cauda equina to the posterior aspect of the dilated dural sac (orange arrow B). Arachnoidal diverticula are observed with the same CSF signal (green arrows in C).
Fig. 4: MR Axial images of the lumbar Spine. Superior row using T1WI correlates with the bottom row of T2WI. A) Displacement of the cauda equina to the dorsal aspect of the widened dural sac (orange arrow). B, C) arachnoidal diverticula producing scalloping of the anterior aspect of the laminae and spinous processes (red arrows). Note the posterior displacement of the roots of the cauda equina within an ectatic dural sac (blue arrow).
DURAL ECTASIA: RADIOLOGICAL FINDINGS

- Widening of the spinal canal
- Scalloping / bone erosions of the anterior surface of posterior elements (laminae, spinous processes and pedicles) and scalloping on the posterior surface of the vertebral body
- Dural calcifications

Fig. 5
Fig. 6: CT lumbar spine. Sagittal sections from right to left. The images show the widening of the lumbar spinal canal (opposite blue arrows in C), with presence of erosions in the anterior aspect of the posterior elements of the vertebrae L1-L5 (purple arrows). Note the squaring of the vertebral bodies, calcification LLA (red arrow in B), interspinous ligament (orange arrow) and apophyseal (green arrow). In some levels disc calcification can be noticed.
Fig. 7: Lumbar spine CT. The axial sections (A-D) show the marked widening the spinal canal (opposite blue arrows in A) and the striking bone erosions of the laminae and spinous processes (purple arrows). The coronal view (E-G) show the typical findings of the "bamboo spine". The red arrow in E shows the interspinous ligament ossification.
Fig. 8: Images of MR and CT at the same level. Notice the arachnoid diverticula producing scalloping of the right laminae and the herniation of the nerve roots within the diverticulum (orange arrow). The CT image show marked thinning of both laminae and in the ventral aspect of the spinous process (blue arrows). The green arrows show the position within the dural sac of the lumboperitoneal shunt catheter, which can be used as a treatment to normalize CSF flow in these patients.
• In the lateral view widening of the spinal canal and bone erosions can be identified.
**Fig. 10:** X-ray cervical, thoracic and lumbar spine. A-D images show the typical bone changes in a patient with an advanced AS: squared of the vertebral bodies, anterior longitudinal ligament calcification (red arrows A, C) and interspinous ligament (blue arrows in B, D), intervertebral disc calcification (green arrows in A), and ankylosis of the joints facets (purple arrows in A).
Fig. 11: Hypothesis regarding the pathogenesis of dural sac enlargement in long-standing ankylosing spondylitis. Chronic primary arachnoiditis or arachnoiditis secondary to recurrent enthesitis results in adhesion of the arachnoid dura mater to the periosteum. Meningeal fibrosis and epidural space obliteration lead to impaired resorption of the cerebrospinal fluid. The fibrotic meninges are inelastic and therefore fail to dampen brief CSF pressure fluctuations secondary to breath and arterial pulsation, which lead to dural sac enlargement and diverticula formation.
Conclusion

- The CES is a rare late complication of longstanding AS that is associated with dural ectasia and arachnoidal diverticula.

- It is important to remember the diagnosis of CES in AS patients presenting mechanical low back pain clinic or neurological symptoms, especially urinary symptoms suggestive of prostatic disease.

- Early diagnosis can be made by MRI, which is able to demonstrate the typical findings described previously.

References


Personal Information