Progressively worsening paraesthesia of the left leg, gait disturbances and constipation in a 63-year-old man: diagnosis and discussion

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Diagnosis

Ventral arachnoid diverticula associated with cauda equina syndrome in ankylosing spondylitis.

Discussion

Cauda equina syndrome (CES) is a well-known but rare and poorly understood complication of ankylosing spondylitis (AS). CES manifests late in the course of AS, often at a time when the AS is no longer active [1]. MRI characteristically shows an enlarged caudal thecal sac and dorsal arachnoid diverticula that fill the erosions of the bony spinal canal [2, 3]. Our patient had the typical features of CES in AS, i.e. sensory changes in lumbosacral dermatomes, constipation and moderate motor deficits in the left lower limb. However, what made this case unique was the presence of ventral arachnoid diverticula. MRI of the lumbosacral spine demonstrated localised scalloping of the posterior aspect of the L1 and L2 vertebral bodies and ventral arachnoid diverticula without neural tissue herniation. Ventral shift and adhesion of the conus medullaris and

the cauda equina were also seen (Figs. 1, 2 in the case presentation). To date, only three cases have been reported regarding ventral dural abnormalities associated with CES in AS (Table 1) [1, 4, 5].

Arachnoid diverticula formation in AS is thought to result from an inflammatory process affecting the vertebral column [6]. In our case, the L1/2 intervertebral disc showed a high signal, similar to that of bone marrow or perivertebral fat on T1- and T2-weighted images (Figs. 1, 2 in the case presentation). These findings are thought to be a reflection of normal disc tissue being replaced by fatty bone marrow following an ossification process after severe discitis in the past [7]. This process was accompanied by destruction of cartilaginous endplates and subchondral bone plates, resulting in fusion of the L1 and L2 vertebral bodies.

The direction of arachnoid diverticula formation is thought to be dependent on the direction of the force of increased CSF pulse pressure and the “give” of the structures surrounding the spinal canal [1]. If structures posterior to the canal are firm and there is a relatively “soft” spot in the posterior aspect of the vertebral body, erosion could occur anterior to the spinal canal. In our case, we speculate, severe enthesitis of the discovertebral junction at the posterior aspect of the L1/2 vertebral bodies occurred in the past and created a “soft” spot. After the inflammatory process ceased to be active, formation of arachnoid diverticula through a dural defect at this spot occurred over many years.

Several possible mechanisms for nerve root damage have been proposed. These include inflammatory injury from arachnoiditis, gravitational traction injury resulting from the presence of arachnoid diverticula, and traumatic damage from excessive CSF pressure fluctuations caused by reduced elasticity and compliance of the caudal sac exposed to inflammatory changes [6, 8, 9]. In our case, we

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Table 1 Comparison of cases with ventral arachnoid diverticula in patients with cauda equina syndrome associated with long-standing ankylosing spondylitis

Case	Patient age and sex	Time since onset of CES (age at onset)	Presenting complaints	Level of dural defects	Description of the lesion	Neural tissue herniation	Imaging studies performed
1 [5]	71, male	2 years (69)	Difficulty walking, impotence, bladder disturbance	L2–L3	Intradural cyst (believed to be arachnoid cyst)	Yes	Myelography: multilobulated cyst at L2–L3 level, bony erosion of the posterior aspect of vertebral bodies, laminae and pedicles
2 [1]	65, male	9 years (56)	Numbness and weakness in both legs, right buttock and posterior thigh pain, bladder and bowel disturbance, impotence	T12–L1	Arachnoid diverticula	Yes	MRI: dural ectasia at the TLJ ventrally, expansion of the central spinal canal, marked erosion of the posterior aspect of T12–L1
3 [4]	50, male	2 years (48)	Numbness in left buttock and feet, bladder disturbance, faecal incontinence	T12–L1	Ventral dural defect	Yes	CT: scalloping of the posterior aspect of T12–L1 vertebral bodies. Myelography: bony defect at T12–L1 level filled by extravasated contrast material. Postmyelographic CT and MRI: anterior displacement of CM and CE fibres into the bony defect and a relative enlargement of the dorsal subarachnoid space
4, present case	63, male	2 years (61)	Numbness and tingling left leg posteriorly, difficulty walking, constipation	L1–L2	Arachnoid diverticula	No (ventral shift and adhesion only)	MRI: localised scalloping of the posterior aspect of L1–L2 vertebral bodies and ventral arachnoid diverticulae

TLJ = thoracolumbar junction; CM = conus medullaris; CE = cauda equina

think chronic arachnoiditis at the level of L1/2 vertebral bodies in the past led to ventral shift and adhesion of the conus medullaris and the cauda equina, causing neural tissue injury and hence CES.

In conclusion, we have presented a case of CES in AS with formation of rare ventral arachnoid diverticula without neural tissue herniation. These changes seem to occur commonly at the level of the thoracolumbar junction and upper lumbar vertebrae.

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