MRI Appearance of Lumbosacral Spine in a Patient with Ankylosing Spondylitis and Cauda Equina Syndrome

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Cauda equina syndrome is an unusual complication of ankylosing spondylitis. We present a case of a 68-year-old woman with long-standing ankylosing spondylitis who developed cauda equina syndrome. MRI revealed a characteristic widening of the lumbosacral thecal sac and numerous dorsal thecal diverticula. MRI is a powerful, noninvasive tool to confirm the diagnosis and exclude a treatable compressive lesion and provides a good alternative to myelography, an invasive and potentially dangerous procedure in such patients.

Key words: Ankylosing spondylitis; Arachnoiditis; Cauda equina syndrome; Magnetic resonance imaging

Cauda equina syndrome is a rare, late neurological complication of long-standing ankylosing spondylitis. Widening of the thecal sac and numerous dorsal thecal diverticula in the lumbosacral region are usually seen in such patients. It is important to distinguish this disorder from an intraspinal compressive lesion. The MRI findings are characteristic, and familiarity with them is helpful in making a correct diagnosis.

CASE REPORT

A 68-year-old woman had ankylosing spondylitis for many years. For several months, she complained of low back pain radiating to the left leg, numbness of the right leg, and intermittent claudication. There was also slight weakness in both legs. The patient denied problems with urination or defecation. Neurological examination revealed hyperreflexia in both legs and hypesthesia to pinprick over the saddle region (S3-S5 dermatomes).

A KUB and a lateral lumbar spine film showed typical changes of ankylosing spondylitis with fusion of both sacroiliac joints, squaring of the lumbar vertebral bodies, and syndesmophyte formation along the lumbar spine (Fig. 1). MRI examination at 1.5T demonstrated a wide thecal sac from L1 to S2, with extensive scalloping of the pedicles, laminae, and spinous processes of several vertebrae caused by numerous dorsal thecal diverticula (Fig. 2). Axial T2-weighted images (T2WI) showed clumping of the nerve roots of the cauda equina on the side of the ectatic thecal sac in both supine (Fig. 3) and prone positions (not shown). This finding suggested adherence of nerve roots to each other and to the arachnoid membrane. No mass or herniated disc impinging on the cauda equina was noted. The patient was treated with nonsteroidal anti-inflammatory agents for about 3 months without significant improvement.
This patient was thought to have cauda equina syndrome, but the MRI study did not disclose a compressive lesion impinging on the cauda equina. Instead, an ectatic dural sac and several dorsal thecal diverticula were seen. Clumping of nerve roots of the cauda equina on the side of the ectatic thecal sac was also found, similar to that seen in arachnoiditis.

The exact pathogenesis of cauda equina syndrome in long-standing ankylosing spondylitis is not fully understood. Based on previously reported operative and pathological findings in such patients, the process is thought to be due to arachnoiditis. The operative findings in a patient reported by Hauge were of a large thecal sac, thinning of the posterior elements, absence of peridural tissues, and atrophic sacral nerve roots adherent to a thickened arachnoid, which

**DISCUSSION**

This patient was thought to have cauda equina syndrome, but the MRI study did not disclose a compressive lesion impinging on the cauda equina. Instead, an ectatic dural sac and several dorsal thecal diverticula were seen. Clumping of nerve roots of the cauda equina on the side of the ectatic thecal sac was also found, similar to that seen in arachnoiditis. The exact pathogenesis of cauda equina syndrome in long-standing ankylosing spondylitis is not fully understood. Based on previously reported operative and pathological findings in such patients, the process is thought to be due to arachnoiditis. The operative findings in a patient reported by Hauge were of a large thecal sac, thinning of the posterior elements, absence of peridural tissues, and atrophic sacral nerve roots adherent to a thickened arachnoid, which
Cauda equina syndrome complicating ankylosing spondylitis

was in turn adherent to the dura [1]. Matthews’s reported postmortem findings of numerous arachnoid diverticula extending posteriorly into erosions of the laminae and spinous processes. The dura and arachnoid were not inflamed or thickened, but some of the free-lying roots of the cauda equina had fibrosis and loss of myelin [2]. Bartleson et al. noted similar findings at operation, namely atrophy of the peridural tissue, adherence of the dura to the surrounding periosseum and ligaments, small nerve roots exiting through foramina of ample size, and no active inflammation of the arachnoid [3]. Although no active dural or arachnoid inflammation could be seen on the histologic examination, Matthews suggested that arachnoiditis was the original insult but subsequently became inactive [2]. The imaging findings on myelography [4] and MRI are similar to those seen in arachnoiditis, which supports this hypothesis. The initial inflammation in the ligaments may lead to adjacent meningeal inflammation and arachnoiditis, with subsequent nerve root inflammation, degeneration, fibrosis, adhesion, and tethering, all resulting in cauda equina syndrome. The neurologic deficits seen in these patients cannot be explained by the dorsal thecal diverticula, which are located posterior to the intervertebral foramina and do not compress the exiting nerve roots [3].

Matthews also suggested that the dorsal thecal diverticula and bony erosions result from CSF pumped by arterial pulsation. Under normal conditions, the meninges and especially the thecal sac expand promptly in response to increased CSF pressure, allowing absorption of CSF and dampening of transmitted pressure variations. Ankylosing spondylitis causes atrophy of the peridural tissues and adherence of the dura to adjacent structures, thereby reducing the elasticity and compliance of the thecal sac. This in turn would impair the sacs ability to dampen brief CSF pressure fluctuations, chiefly pulse pressure. Over a course of years, excessive pulse pressure could cause slowly enlarging thecal diverticula and secondary erosion of the dorsal bony elements of the lower spine.

Although the spine is usually extensively involved in ankylosing spondylitis, neurologic complications are uncommon. Cauda equina syndrome is a rare, late complication of long-standing ankylosing spondylitis, with an average age of onset of 57 years (39 to 70 years). The interval from onset of ankylosing spondylitis to cauda equina syndrome averages 35 years (17 to 53 years) [3]. Symptoms of cauda equina syndrome in patients with ankylosing spondylitis have prompted evaluation for a possible compressive lesion. Interestingly, there is usually none found.

We suggest that MRI is the investigation of choice in these patients because it is noninvasive can clearly demonstrates characteristic findings, and thus excludes a treatable compressive lesion. Myelography or computed tomographic (CT) myelography should be avoided if possible. Lumbar puncture in such a condition can be technically difficult and hazardous, owing to bony ankylosis and ossification of the spinal ligaments [3, 5]. Dural ectasia is also seen in Marfan’s syndrome, Ehlers-Danlos syndrome, and neurofibromatosis, but the bone erosions in ankylosing spondylitis predominantly involve the posterior elements rather than posterior aspect of the vertebral bodies, as typically seen in these other conditions [6].

The cauda equina syndrome in long-standing ankylosing spondylitis is progressive in most reported cases. In Bartleson’s study, 13 of 14 patients had a very slow but progressive course [3]. No treatment is known to be effective in this condition. No clinical improvement has been reported after treatment with corticosteroids or nonsteroidal anti-inflammatory agents [3, 4, 7]. Given the apparent fibrosis and tethering of nerve roots, surgical intervention also seems to play no role in the treatment for such patients. Although decompressive laminectomy has been reported in a small number of patients, almost none benefited from the procedure [1, 3, 8, 9, 10, 11, 12, 13]. Shaw reported one patient who was successfully treated in this way. He described some compression of nerve roots, perhaps by arachnoid cysts and attributed the success to early exploration and decompression of the arachnoid diverticula [13].

MRI is thus a useful, noninvasive diagnostic method for evaluating cauda equina syndrome in patients with ankylosing spondylitis. If it reveals a compressive lesion, prompt corrective action could be undertaken. However, if the findings are consistent with most cases reported to date, an appropriate diagnosis and prognosis can be determined without subjecting the patient to invasive diagnostic or therapeutic procedures.

REFERENCES

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長期僵直性脊椎炎病患併發馬尾症候群在腰薦椎之磁振造影表現：病例報告

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馬尾症候群是僵直性脊椎炎病患一個少見之併發症，我們在此提出一病例報告：一名68歲女性病患患有長期之僵直性脊椎炎併發馬尾症候群，其腰薦椎之磁振造影顯示具特徵性之硬膜囊擴張及許多的背側硬膜憩室。磁振造影為一強而有力又不具侵犯性之工具，不僅可以幫助確立診斷並且可以排除可治療之壓迫性病兆。脊髓腔X光攝影術對於此種病患可能難以施行，並且可能造成病人的傷害，故應避免。

關鍵詞：僵直性脊椎炎，蜘蛛膜炎，馬尾症候群，磁振造影