of metallic clips in the neural foramen would nullify the results.

According to our present case, CT myelography with MIP and 3D reconstruction could be considered the most reliable technique for assessing postoperative spinal CSF leaks. CT myelography provides high diagnostic accuracy by being able to demonstrate, despite surgical clips, the presence of a CSF leak, its location and extent, and its relationship to the surrounding anatomy, allowing the appropriate surgical planning.

References


Spinal cord herniation

Hernie médullaire

A 63-year-old woman had a 1.5-year history of rapidly progressing spastic paraparesis, with bowel and bladder disturbance. She also had sensory loss of the right leg, but no pain. There was no history of spinal trauma. Following a lumbar MRI, which revealed spinal stenosis, she underwent surgical decompression at the L4–L5 level. As this resulted in no improvement, thoracic MRI was performed, including sagittal T2-, sagittal T1-, frontal and axial high-resolution T2-weighted imaging and a sagittal 2D phase-contrast (PC) cine sequence (Fig. 1). MRI showed anterior displacement of the spinal cord at T4–T5 level with an abnormally thin cord in the anteroposterior view. The sagittal 2D PC cine MRI (with velocity encoding sequencing) showed interruption of CSF flux along the spinal cord and flow turbulence behind the herniation, thereby excluding a posterior arachnoid cyst. The clinical picture was interpreted as an idiopathic transdural spinal cord herniation. Surgery (posterior laminectomy) confirmed cord herniation through a defect of the ventral dural wall with minimal adhesions. The herniation was reduced and the defect repaired with an artificial dural patch. However, her condition worsened immediately after the operation, prompting a further MRI investigation, which showed the cord in a central position within the dura (Fig. 2), but severely narrowed (interpreted as myelomalacia due to hyperintensities on T2-weighted imaging). Three months later, there was no significant improvement and follow-up MRI revealed an identical recurrence of herniation. Considering the postoperative complications and with the patient’s agreement, no further surgery was planned.

Transdural spinal cord herniation is a rare pathology, with fewer than 100 cases described in the literature [1] since its initial description in 1973 [2]. It frequently presents as Brown–Sequard syndrome or myelopathy. It can arise spontaneously, posttraumatically or iatrogenically. In an analysis of 30 cases, spontaneous causes represented 57% [3]. There is a female predominance in spontaneous cases with a ratio of 2.4:1 [3], and a strong male predominance in posttraumatic and iatrogenic cases. Transdural herniation can be either ventral (if spontaneous) or dorsal. It occurs mainly at the cervical (often associated with iatrogenic origin) and mid-thoracic (T4–T7) levels.
Figure 1  Sagittal T2- (A) and T1-weighted (B) MRI shows focal atrophy and anterior displacement of the thoracic spinal cord at T4–T5 level. Sagittal MRI 2D phase-contrast cine (with velocity encoding sequences) (C) shows CSF flow turbulence just behind the herniated spinal cord. Axial T2 high-resolution (CISS) MRI (D) shows herniation of the spinal cord towards the anterior epidural space.

Les coupes IRM sagittales pondérées T2 (A) et T1 (B) montrent une atrophie focale de la moelle thoracique avec angulation et déplacement antérieur en T4-T5. La séquence sagittale en contraste de phase 2D et mode ciné (étude vélocimétrique à la phase systolique) (C) montre des turbulences du flux de LCS immédiatement en arrière de la moelle herniée. L'image T2 axiale en haute résolution (CISS) (D) montre une hernie du cordon médullaire vers l'espace épidural antérieur.

Its physiopathology remains unclear, whatever its origin. There is no association with congenital anomalies of the spine or cord, but is an acquired condition with predisposing factors (such as congenital cyst, degenerative disk prolapse, duplication of the dura and clinically occult minor trauma) [4]. It can originate from a dural or CSF flow pathology. Normally, both cord and dura are mobile, with motion in both craniocaudal and anteroposterior directions. Dilatation of the CSF space at the herniation level is usually observed, and may be related to an arachnoid cyst. Such associated cysts have been found on surgery in up to 30% of cases [3] as either a cause or consequence of the herniation. When there is no cyst, widening of the CSF space may be responsible for turbulence in CSF flow, as shown by cine PC MRI sequences, which could be increasing the pressure over the area of cord herniation and making it worse. It may also contribute to cord narrowing. Conventional MRI findings can usually make the diagnosis, although high-resolution T2 MRI and 2D-PC cine imaging can be helpful in determining the presence of an arachnoid cyst [4,5].

Surgical release and repair of the dural defect can lead to improvement, but total recovery is rare. Various surgical techniques [1,6] have been proposed to improve the results, including closing the dural defect, widening the dural defect or wrapping a dura graft around the myelum.

References

Calcified senile scleral plaques

Plaques sclérales séniles calcifiées

To investigate the prevalence of calcified senile scleral plaques (CSSP), all cranial CT scans acquired at Braunschweig Teaching Hospitals between 1st and 16th November 2007 were retrospectively evaluated for the presence of CSSP (N = 300 patients; mean age 61.7 years, range: 10–93; 50.3% female; axial CT with 3 mm slices of the posterior fossa, including the orbits, 6 mm supratentorially). Indications included focal neurological deficit (33.3%), headache (13.3%), head injury (11.7%), reduced vigilance (10.3%), psychiatric states (10.3%), vertigo (9.3%) and staging (9.3%).

CSSP were identified in 18 patients (6%; mean age 80.6 years, range 51–93; 83.3% female). Prevalence increased from 2% in patients aged less than 70 years to 7.2% in those aged 70 to 79 years and to 22.6% in those aged more or equal to 80 years. The plaques most frequently involved the insertions of the rectus muscles (77.7%) and were symmetrical in 55.5%, appearing as ovoid hyperdensities (length 1–5 mm, width about 1 mm; bone window settings) (Fig. 1). The lateral recti were involved in 27.7% of cases (one patient had medial and lateral recti involvement) and no plaque was identified at the insertions of the superior and inferior recti.

Scleral calcification has a differential diagnosis that includes major pathologies such as inflammation, lymphoma and hypercalcemic states [1], but is not infrequently encountered in asymptomatic patients. In such cases, like dystrophic calcification elsewhere in the body, calcium salts are deposited in plaque-like areas of hyaline degeneration, usually anterior to the insertions of the rectus muscles. While usually asymptomatic, plaque sequestration and expulsion with ulceration may occur [2,3]. No association with systemic conditions has been observed [4] and prevalence of between 3% and 6.2% has been recorded (randomly selected scans, ophthalmological indications [4–6]).

CSSP were present in 6% of cases in our general patient population, which is similar to that in a previous report of an ophthalmological population (6.2%, N = 145 [5]). In contrast, a lower prevalence of 3% was recorded in one study (N = 100 [6]), although that population was considerably younger (mean age 35 years). We found that the prevalence of CSSP increases considerably with age, which compares favorably with previous published age distributions: Gordon et al. [5] reported a prevalence of 22.6% for patients more than 70 years and Moseley [4] recorded a prevalence of 4% for those aged 70 to 79 years old and 22% for patients more or equal to 80 years. As documented by Alorainy [7], we also found a higher prevalence in women, which may be partly explained by the higher mean age of the women in our cohort (65.8 years vs. 57.3 years for men). Due to scanning in a transverse plane in our study, an under-recognition of plaque presence in the superior and inferior recti may have occurred, although this is unlikely, given that Alorainy [7] found that only one of 109 plaques (0.9%) in their series was located at the insertion of the superior rectus — with none at the insertion of the inferior rectus.

In conclusion, around 6% of subjects undergoing cranial CT scanning for unrelated indications showed calcified senile scleral plaques, with a prevalence increasing with age. Radiologists should be aware of the appearance and location of this "don’t-touch" lesion to distinguish it from high-density foreign bodies and clinically relevant scleral calcifications.

References