



Expert's Comment concerning Grand Rounds case entitled "Idiopathic spinal cord herniation: consideration of its pathogenesis based on the histopathology of the dura mater" by S. Shimizu et al. (Eur Spine J; 2017. DOI 10.1007/s00586-017-5147-y)

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The authors present a case of a 33 years gentleman presenting with leg spasticity and hyperesthesia, due to a ventrally displaced spinal cord at the T5/6 level [1]. This is an interesting case with histological assessment of the dural edges, which were biopsied at the time of repair of an idiopathic spinal cord herniation. The report is clear and well-presented.

One of the difficulties with such cases presenting with radiological displacement of the thoracic cord is knowing whether the cord is pushed (by a dorsal arachnoid cyst) or pulled (by a ventral herniation through a dural defect). Discriminating between the two diagnoses can be challenging, and may be aided by CT myelography, MRI constructive interference in steady state (CISS) sequences, or MRI CSF flow studies. This information may influence the surgical approach, by a posterior midline, posterolateral, or costotransversectomy approach. With a true ventral cord herniation, the spinal cord is usually displaced at the level of a thoracic disc, and there is no visible CSF on MRI or CT

myelography between the spinal cord and dura adjacent to the thoracic disc. In the case of a dorsal arachnoid cyst, however, the posterior spinal cord is sometimes seen to be scalloped or indented by the overlying cyst, and CSF may be seen anterior to the spinal cord.

The authors outline the possible etiologies of ventral dural herniation of the spinal cord, and probably the most plausible is a thoracic disc herniation causing weakness and defect in the dura, anterior to the spinal cord.

Treatment of ventral dural defects can be challenging, and recurrent herniations or adhesions can occur after surgery. Suturing the defect is often not possible without retracting the spinal cord, and the whole defect cannot be adequately visualised. It is more common to repair the defect by the application of a dural patch and tissue adhesives. Alternatively the spinal cord can be reduced and hitched by stitching the dentate ligament to the dura lateral to the spinal cord to maintain a central position of the cord within the spinal canal [2]. Otherwise CSF pulsations may continue to displace the spinal cord forwards, towards the repaired defect, and subsequently develop adhesions and tethering or re-herniation of the cord through the defect. Alternatively the "hammock procedure" [3], as adopted by the authors, may be used to minimise ventral displacement of the spinal cord after surgical repair.

At the time of surgery, dural biopsies from the edge of the defect were taken by the authors for histological assessment. The dura at the posterior surgical durotomy was histologically normal. However, the dura at the edges of the ventral defect revealed degeneration and loose arrangement of the collagen fibres with edema, macrophage infiltration and angiogenesis, assessed by haematoxylin and eosin histological stains.

Unfortunately the authors state that more rigorous immunohistochemical analyses were not possible. It would be

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interesting to assess the proportions of type 1 and 2 collagen around the defect compared to the normal dura, the proportions of elastin, immunostaining for macrophages, lymphocytes, and collagen/elastin to provide a more informative assessment of the inflammatory environment.

The inflammatory changes at the edge of the dural defect are likely to be due to chronic mechanical irritation from the spontaneous cord herniation and microtrauma to the dura. It is possible that primary dural inflammation could be the initiating factor of the spinal cord herniation, but this is difficult to substantiate on the evidence provided in this case report. As is often the case, it is difficult to say whether the histological evidence of inflammation is causative, or solely associated with the ventral cord herniation.

Compliance with ethical standards

Conflict of interest The author has no conflict of interest.

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