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Letter to the Editor

Incarcerated spinal cord: A preventable surgical debacle

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Dear Editor,

Herniation followed by incarceration of spinal cord has increasingly been reported in the postmagnetic resonance imaging (MRI) era with various causes such as idiopathic, iatrogenic, traumatic, congenital, and inflammatory.[1] However, majority of the cases described have ventral herniation.[1] Primary dural repair, augmented duraplasty or simply widening of dural defect are the various techniques described so far.[1] Here we describe a rare case of posteriorly incarcerated spinal cord following a failed attempt at removing an intradural extramedullary (IDEM) lesion (shwannoma) at D11 level.

A 27-year-old male patient presented at a private clinic with back pain and mild spastic paraparesis. His MRI showed an anteriorly placed IDEM lesion at D11 level [Figure 1a and b]. He underwent D11 level laminectomy but due to excessive bleeding surgery was abandoned and dura was left open with layers of oxidized cellulose (Surgicel®). Postoperatively his pain worsened and he developed progressive weakness of both lower limbs with bowel bladder involvement, over a month. On presentation to our neurosurgical service after 4 weeks of first surgery, he had paraplegia with complete sensory loss below L1 level. MRI showed a herniated spinal cord through a dorsal dural defect with cord signal changes [Figure 1c-e]. A postero-lateral approach (costotransversectomy) was chosen to remove the lesion and reposition the herniated cord. Intraoperatively there was a herniated spinal cord through a narrow dural neck leading to its incarceration [Figure 2]. We excised the IDEM and reposited the cord in the thus created space with primary repair of dura. Postoperative MRI showed complete excision of lesion with cerebrospinal fluid (CSF) space surrounding the reposited cord [Figure 1f-i]. Patient improved partially at 3 months follow-up.

Ohnishi *et al*. described a similar case of dorsal cervical herniated cord following laminectomy for ossified yellow ligament in the presence of ossified posterior longitudinal ligament compressing the cord from its ventral aspect.[5] The mechanism of such spinal cord herniation leading to myelopathy has excellently been described by Kumar *et al*.[4] They have proposed two prerequisites, namely, (1) a dural defect (congenital or iatrogenic) and (2) concave surface of spinal cord (e.g., dorsal for cervical, ventral for thoracic).[4] The normal flexion-extension or pulse synchronous sagittal displacement of cord has been well documented.[4] The spinal cord has the maximum mobility at areas with most severe pathology.[4] With each pulsations or movement arachnoid herniates out of dural defect with subsequent herniation of spinal cord.[4]

In our case herniation was posterior, that is, on the dorsal surface of thoracic cord. It might have been exacerbated by the fact that an anteriorly placed IDEM was forming the dorsal vector of displacement. Besides the concave surface of cord at the level of lesion was ventral, thereby supporting the theory proposed by Kumar *et al*.[4] Moreover, the narrow defect leads to an early incarceration. It is analogous to small craniectomies and duratomies for significant raised intracranial pressure thereby causing herniation and incarceration of brain tissue especially when the intracranial pathology is un-tackled. In such

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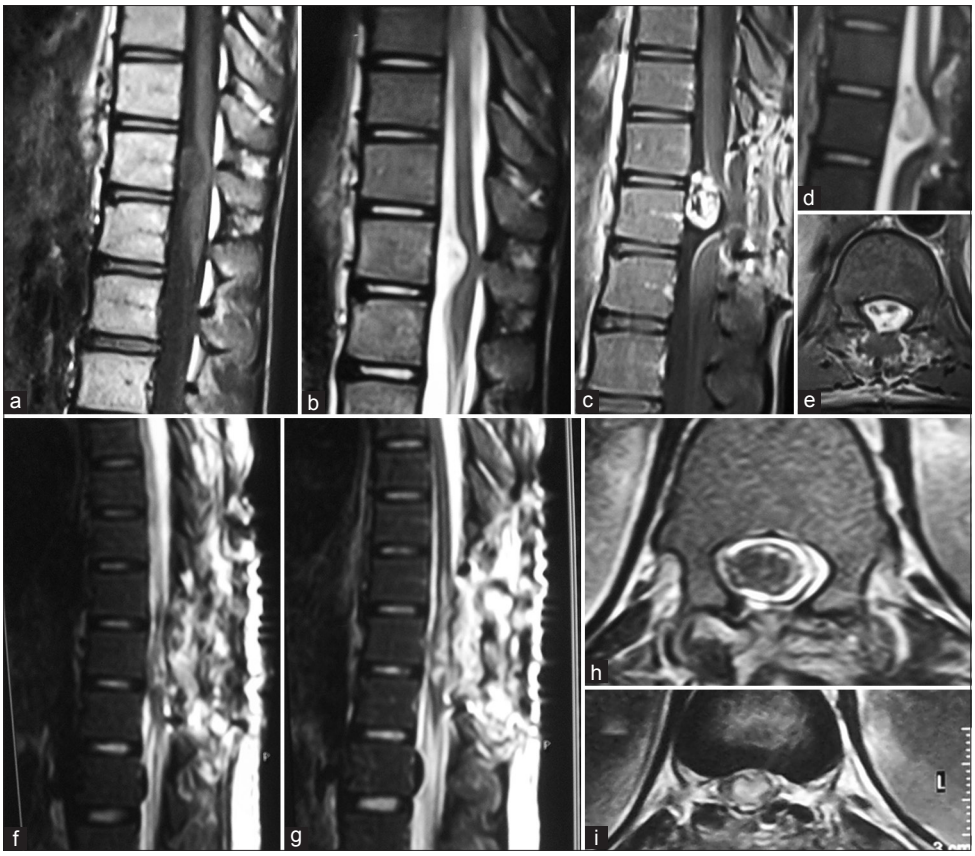

Figure 1: (a and b) Preoperative T1 and T2 images of MRI of dorsal spine showing intradural extramedullary lesion anteriorly placed lesion at D11 vertebra. (c), (d and e) Dorsally herniating spinal cord through a dural defect at laminectomy site, one month after failed attempt for removal of lesion as seen on MRI images. (f, g, h and i) Postoperative MRI images after removal of lesion and reposition of cord. CSF space is seen all around the repositioned cord. Altered signal intensities are noticed in the cord parenchyma, probably secondary to incarceration

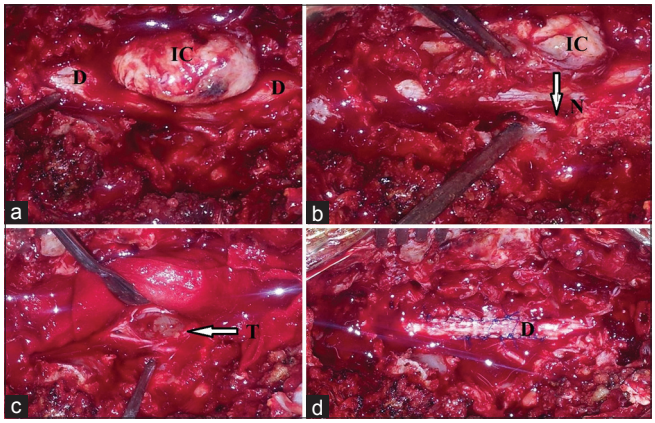

Figure 2: Intraoperative images. (a) Incarcerated cord (IC) seen herniating though the dural (d) defect. (b) Nerve root (n) is seen alongside the dural defect with herniated cord. (c) Tumor (t) is seen though a separate dural incision. (d) Primary closure of dural (d) defect after excision of lesion and reposition of cord

cases a large decompressive craniectomy and duratomy does not cause incarcerations.[2] Similarly a narrow defect left on spinal dura is more hazardous than longer defect for the chances of early incarceration especially when the pathology is untouched. Based on the same principles, Isu *et al*. described a surgical strategy of widening the dural defect to prevent cord strangulation and minimize its handling.[3] Else the narrow duratomy defect needs to be repaired either primarily or with a graft especially for an untreated lesion.

Once incarceration or herniation has been diagnosed, it needs to be surgically treated as soon as possible to arrest the ongoing myelopathy. Prevention of herniation would obviously be the best strategy. In a situation where the lesion cannot be excised and dura needs to be left open for fear of handling the bulging cord, it is advisable to open the dura widely. The usual time for cord herniation and myelopathy is 2-4 weeks as seen in a case described by Ohnishi *et al*. as well as ours.[5] Therefore it is preferable to tackle the lesion through a different surgical approach as early as possible (<2 weeks) so as to prevent the cord herniation and ensuing myelopathy.

This report highlights the importance of early surgery through a different approach to prevent the herniation and incarceration of spinal cord, which is likely to occur following failure of removal of the lesion and inability to close the dura due to cord bulge. Awareness about this complication is essential both in terms of taking measures for its prevention and early suspicion and

treatment, which should rectify the neurological deficits, at least partly.

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Commentary

Although a rare clinical condition, idiopathic spinal cord herniation has been reported with increasing frequency in recent years. This is likely due to heightened awareness about this uncommon disorder as well as refinement of MRI techniques that more ably demonstrate the classic radiographical features of this abnormality. Surgical intervention is frequently recommended to relieve the spinal cord incarceration and halt the progression of neurological symptomatology. The majority of published reports point toward a likely congenital or traumatic etiology, yet the cause in many cases is not completely known. In a subset of herniated spinal cord cases, the etiology is clearly iatrogenic, and results from a breech in the dura following surgery. This could potentially be related to an incidental or intentional durotomy, directly or indirectly repaired. In the present case, the authors present a patient that previously underwent an attempted resection of a ventral T11 IDEM tumor, which was eventually aborted due to excessive bleeding. The dura was not able to be closed primarily, and therefore was left open with layers of oxidized cellulose as covering. Unfortunately the patient soon developed spinal cord herniation through the dorsal dural defect. The herniation led to the onset of progressive paraplegia, as well as a revision surgery to remove the tumor and repair the spinal cord herniation. The authors should be congratulated on their management of this difficult case. The radiographical outcome was excellent, and it appears as though the patient has made some partial neurological recovery at 3 months follow-up. I am interested in knowing whether continued neurological improvement will be achieved with longer clinical follow-up time. One of the emphasized learning points of the article is that early postoperative spinal cord herniation is more likely to occur with a smaller dural defect than a larger defect. The implication is that in cases of an unresectable large ventral tumor, it is better to leave the dura open with a long dural opening than a narrow one. Although I agree with this thought in principle, my first choice is not to leave the dura open at all if possible, but rather perform an expansile duraplasty

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