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Case Report *Volume 3 Issue 1*

Reversible Thoracic Cord Edema Secondary to Morphine Intrathecal Catheter Granuloma – A Case Report

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Abstract

Introduction: Long term back pain has been treated with intrathecal drug delivery since 1970. Formation of inflammatory masses at the tips of intrathecal catheters are a known complication and can result in permanent neurologic consequences. Our case represents an uncommon intramedullary granuloma at the T10 level from a morphine pain pump.

Methods: Imaging was acquired using a 3 Tesla MRI scanner (GE HDx, software 14.M5) with spinal 8 channel phase array coils (USA Instruments). Key imaging included pre-contrast axial 3 mm T1 and T2-weighted images, sagittal 5 mm T1, T2 and STIR images, post contrast (20 ml of Multihance gadolinium, IV) axial and sagittal T1-weighted images and high resolution, non-contrast 3D sagittal 3 mm T2-weighted images.

Results: A 43-year-old man with increasing chronic low back pain presented to the ER with additional leg weakness and anterior thigh paresthesias. Physical examination revealed thoracic point tenderness, no skin findings and a normal gait. An outside hospital report described a possible cord mass vs a granuloma. The patient was referred for a neurosurgical evaluation and an MRI of the spine was obtained. An intrathecal catheter entered at the L2 level and extended cephalad to T10. An enhancing mass was located around the distal catheter tip, with enhancement and T2 hyperintensity within the adjacent intramedullary cord. Abnormal high T2 cord signal and generalized cord swelling extended from the T6-T12 levels. Surgical catheter retraction to the T12/L1 interspace and medication dose reduction by 10% resulted in partial resolution of imaging findings. The patient's symptoms improved on follow-up.

Discussion: Proposed explanations for granuloma formation include abnormal CSF dynamics, catheter positioning and medication-induced inflammation. Histologically, these granulomas are highly vascular, have a peripheral margin of inflammatory cells surrounding a central area of necrosis, and originate from the arachnoid and possibly the dural layers.

Keywords: Reversible Cord edema; Intrathecal morphine catheter; Catheter granuloma

Abbreviations: MRI: Magnetic Resonance Imaging; STIR: Short TI Inversion Recovery.

Introduction

Long term back pain has been treated with intrathecal drug delivery since 1970 [1]. Although uncommon, formation of inflammatory masses at the tips of intrathecal catheters is a known complication. As it can be devastating and potentially result in permanent neurologic consequences, appropriate radiological diagnosis is of great importance in promptly treating this condition [1]. The following case report is an uncommon intrathecal granuloma at the T10-11 level, developing on the tip of a morphine pain pump with associated reactive inflammation of the spinal cord.

Case Report

A 43-year-old man with a history of chronic low back pain presented to the ER with increasing lower back pain, leg weakness and anterior thigh paresthesia. The physical exam revealed thoracic spine tenderness, no skin findings and a normal gait. An outside hospital Magnetic Resonance Imaging [MRI] report described a possible cord mass vs a granuloma. The patient was referred for a neurosurgical evaluation, and an MRI of the thoracic spine with and without contrast was obtained (Figure 1). The intrathecal catheter entered the thecal sac at the L2 level and extended cephalad to T10 level. A 1 cm enhancing intradural mass was located at T10-11 around the distal catheter tip. It abuts and displaces the spinal cord to the right. Abnormal high T2 cord signal at the contact level, extended cephalad to the T6 level and caudad to the T12 level. The T2 cord hyperintensity was associated with generalized cord swelling. The patient was then treated with surgical catheter retraction, with the tip of the catheter brought down to the T12-L1 interspace. The morphine medication dose was also reduced by 10%. A repeat MRI of the thoracic spine with and without contrast was repeated 3 weeks after this treatment (Figure 2). The imaging demonstrated decreased cord enhancement and edema, with residual mild abnormal high T2 cord signal, at the T10-11 level. The patient's symptoms improved on subsequent office visits.

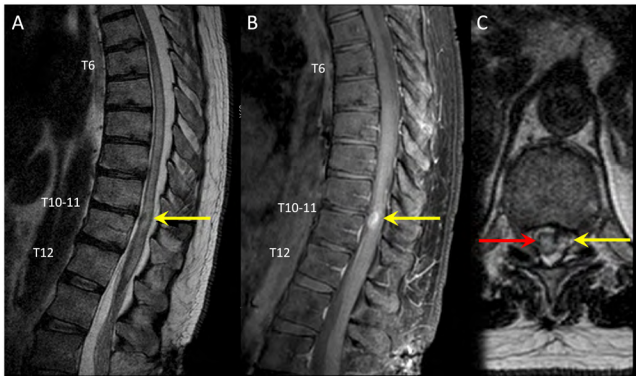

Figure 1: A) Sagittal T2-weighted image. Abnormal high T2 cord signal extends from the T6-T12 level. Associated generalized cord swelling is readily apparent. Arrow points to the lesion.
B) Post contrast Sagittal T1-weighted image. An enhancing mass (arrow) is located at T10- 11 level, around the distal catheter tip, touching the adjacent intramedullary cord.
C) 3D axial T2-weighted image. Mixed T2 signal left sided intradural mass (yellow arrow) is located at T10-11, around the distal catheter tip, abutting the cord and displacing the cord (red arrow) to the right. High T2 cord signal is present.

II Post Treatment Imaging

MRI obtained 3 weeks after retraction of the catheter.

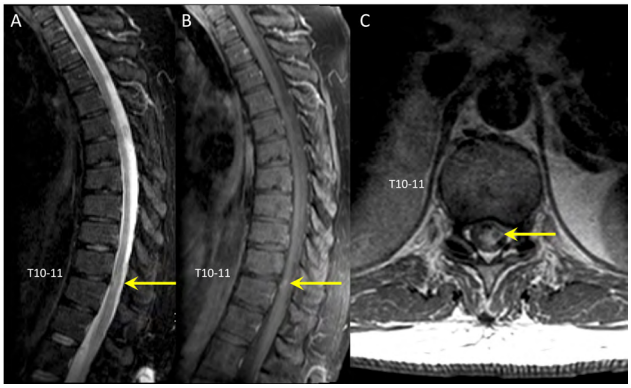

Figure 2: A) Sagittal T2 FLAIR image. Residual mild high T2 signal at T10-11 cord (arrow). Note that the cranial and caudal extents of cord T2 hyperintensity have significantly decreased.
B) Sagittal Post contrast T1-weighted image. Residual enhancement (arrow) is smaller at the initial site of inflammation.
C) Axial T2-weighted image. There is resolution of the previously-seen left lateral intradural mass. There is no more mass effect on the cord. Cord has moved to its central location in the thecal sac (arrow).

Methods

The patient was imaged using a 3T MRI (GE HDx, software version 14.M5, Milwaukee, WI), and a spinal 8 channel phased array coil (USA Instruments, Aurora, Ohio). Key imaging sequences included pre-contrast axial 3 mm and sagittal 5 mm T1-weighted and T2 weighted images, sagittal 5 mm STIR images, and post contrast (20 mL IV Multihance, Bracco Diagnostics, Milan, Italy) axial 3 mm and sagittal 5 mm T1-weighted images. Additional higher resolution, non-contrast 3D sagittal 1 mm T2-weighted sequences were obtained through the granuloma (reformatted as 3 mm axial and sagittal images). 3 weeks after treatment, an MRI of the thoracic spine with and without contrast was repeated with the same parameters, sequences and contrast.

Discussion

Intrathecal granulomas have developed following intradural drug delivery system of opioids like fentanyl, morphine, diamorphine, hydromorphone, tramadol, Sufentanil and non-opioid baclofen [1,2]. Though reporting of this complication is voluntary, based on available data the incidence of granuloma formation is about 0.04% after 1 year and 1.15% after 6 years of therapy [3,4]. Duarte et al. tested the longstanding hypothesis that the drug concentration played a role in the development of intrathecal granuloma and confirmed it [2]. Hence, they recommended decreasing the opioid dose and the concentration intrathecally, from the recommended maximum of 15 mg/day and 20 mg/mL to 10 mg/day and 15 mg/mL respectively [2].

To avoid formation of an intrathecal granuloma, Deer et al. cautioned against ultraslow rates and positioning of catheter tip between Thoracic 7 and Thoracic 10 levels [5]. Kratzch, et al. also found correlations between granuloma formation and catheter tip placement at Thoracic 5- 8 levels and prior spinal surgery [6]. Previous hypotheses for granuloma formation include abnormal CSF dynamics resulting from catheter placement, catheter positioning and properties of the drug [7]. Histologic evaluation of granulomas demonstrated highly vascularity with a peripheral margin of inflammatory cells and fibrosis that surround a central area of necrosis [3,8,9]. Granuloma formation has been proposed to originate from the arachnoid layer by some authors, while others have described involvement of both the dural and arachnoid layers [4,10].

Histologically, it has been argued that the phlegmon is not a true granuloma by histopathology criteria, as they have not been found to contain giant cells amidst the granulation tissue [4,11]. MRI with and without contrast is the imaging of choice for diagnosing intrathecal lesions and cord abnormalities. The peripheral enhancement is understood to represent capillary leakage and presumed to result from neo-vascularity containing fenestrated capillaries. We propose that this is responsible for the enhancement in our case, and this would also explain the intramedullary edema we observed that resolved with catheter repositioning.

We also propose that the hypointense T2 peripheral rim of this lesion is due to fibrosis, while the hyperintense T2 central signal of the phlegmon is due to necrosis, as intrathecal granulomas and intramedullary abscess formation have been reported with an intrathecal morphine pump [3,12]. Finally, prompt treatment of intrathecal catheter granulomas is important as the efficacy of intrathecal medication decreases once granuloma forms [2,13]. Drug tolerance and increase in drug infusion can occur following failure of prompt diagnosis of a granuloma [2,14,15]. In our case, catheter repositioning and changing the catheter dosage improved symptoms.

Conclusion

Formation of intrathecal inflammatory granuloma at the tips of intrathecal drug delivery catheters is an uncommon manifestation but a known complication. Awareness of this entity and prompt radiological diagnosis is of great importance in timely treating these patients, as it can be consequential and potentially result in permanent neurologic deficits.

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