

Cysticercosis of conus medullaris: A case report and literature review

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ABSTRACT

“Neurocysticercosis” - involvement of the central nervous system (CNS) by taenia solium, is one of the most common parasitic diseases of the CNS. However, spinal involvement by neurocysticercosis is uncommon. Spinal intramedullary cysticercosis involving the conus medullaris is an uncommon clinical condition, which may mimic an intramedullary tumor and can lead to irreversible neurological deficits if untreated. Here, we report a 31-year-old male patient with cysticercosis in the conus medullaris of the spinal cord. Magnetic resonance imaging revealed a well-defined round intramedullary lesion at D12-L1 vertebral levels, which was homogeneously hypointense on T1WI and hyperintense on T2WI with peripheral edema. Since the patient had progressive neurological deficits, surgery was performed to decompress the spinal cord. Histopathology examination of the removed lesion proved it to be cysticercosis. In this report, we also discuss the principles of diagnosis and treatment of intramedullary cysticercosis in combination with literature review.

Key words: Cysticercosis, intramedullary, spinal cord

INTRODUCTION

Neurocysticercosis, caused by taenia solium, is the most common parasitic infection affecting the central nervous system. However, the spinal cysticercosis is rare, representing 1.2-5.8% of all cases of neurocysticercosis.^[1,2] According to the cysticercus location in spine, cysticercosis has been classified anatomically as extraspinal (vertebral) or intraspinal (epidural, subdural, arachnoid, or intramedullary), of which the intramedullary type is quite rare and only 54 cases have been reported until 2012.^[3-8]

CASE REPORT

This was a case report of a 31-year-old male patient who was transferred to our department from Urology Department with the complaints of voiding dysfunction and constipation for last 1½ years. Neurological examination revealed spastic paraparesis with decreased motor power of grade 3/5 in both lower limbs, impaired sensations below

L1 dermatome, brisk tendon jerks and positive Babinski signs on both sides. Magnetic resonance imaging (MRI) revealed a 4.1 cm × 1.9 cm × 1.3 cm well-defined round expansile lesion at T12-L1 vertebral level, in conus medullaris with cystic degeneration and septae within, heterogeneously hypointense on T1WI [Figure 1a] and hyperintense on T2WI [Figure 1b] with peripheral edema and mild contrast enhancement [Figure 1c], mainly at the antero-inferior limit of cystic part and around it, with the septae also exhibiting enhancement. The subarachnoid space from T12 to L1 vertebra was narrow due to the marked expansion of spinal cord. There were no abnormalities at cervical or lumbar levels or within the brain parenchyma. The diagnosis of intramedullary mass lesion was made. The patient underwent laminectomy from T11 to L2 and the spinal cord was found swollen. When a midline myelotomy was performed at T12-L1 level, a white cystic lesion was seen and clear fluid was then aspirated. The cyst wall of which slightly stuck to the surrounding the spinal cord. In order to dissect the cyst with minimal injury to the peripheral tissue, the cystic liquid was partly withdrawn first and the slackened cyst was removed totally. The liquid was yellowish and transparent. Histological examination [Figure 2] of the resected sample showed cysticercosis.

Post-operatively, the patient refused to be treated with anticysticercal agents and steroids. The patient's

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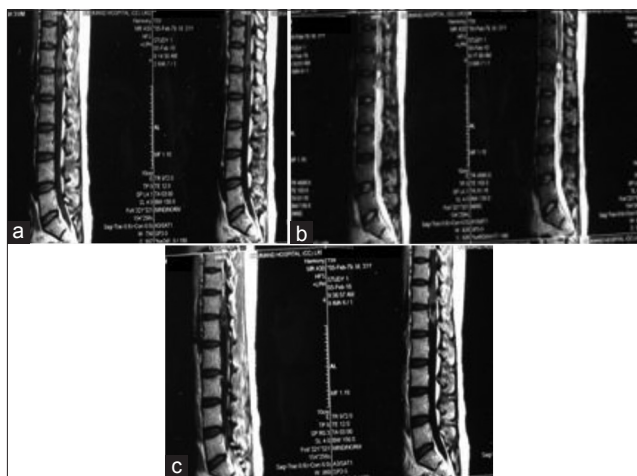


Figure 1: Sagittal T1 (a), T2-weighted (b) Contrast (c) Magnetic resonance image of thoracic spine showing a relatively well-defined cystic intramedullary lesion with hypointense on T1WI and hyperintense on T2WI with minimal contrast enhancement

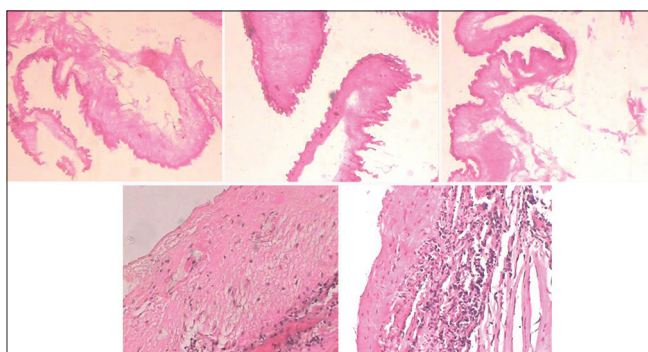


Figure 2: Photomicrographs of the histological specimen showing the cysticercosis cyst wall with neutrophil granulocyte lymphocyte and necrosis cell (H and E, $\times 100$)

neurological function in the immediate post-operative period was not changed from his pre-operatively status and he was discharged 2 weeks later. At 6 months of follow-up, the motor power of his lower limbs recovered to grade 4/5 and he could ambulate without special support. The function of the anal sphincter and bladder regained to extent that within 14 months of surgery patient was off “clean self-intermittent catheterization” of bladder with no significant postvoid residual volume. The patient to have constipation with defecation every 2nd-3rd day without compromise of the activities of his daily living.

DISCUSSION

Cysticercosis is widely endemic in Brazil, Peru, Mexico, Korea and India.^[1,2] Intramedullary cysticercosis often presents in the patients between 20 and 45 years old, with the youngest one reported being 5 years old and the oldest one 45 year's old.^[9] The common clinical manifestations included pain, paraparesis, spasticity, bowel and bladder incontinence and sexual dysfunction.^[2,3] Most patients

experienced a progressively worsened course from a week to 10 years.^[20]

Inflammatory reaction against the dead parasite is associated with perilesional edema, which can damage medullar parenchyma and therefore, worsen symptoms.^[4] Inside the spinal cord, cysticercus usually distributes in the thoracic cord, with a few cases involving the cervical and the lumbar cord. This distributional mode of cysticercus supports the hypothesis that intramedullary cysticercus comes from the blood circulation, because thoracic cord has a richer blood supply than the other parts of the spinal cord.^[2,10] However, it is also thought that intramedullary cysticercus could migrate to the spinal cord via the ventriculo-ependymal pathway.

On MRI, intramedullary cysticercosis usually show a cystic lesion within the spinal cord, which appears hypointense on T1WI with hyperintense scolex identified inside the cyst cavity, hyperintense on T2WI in vesicular stage, a subtle hypointense rim may surround the intramedullary cyst on T2WI. In the colloidal stage the thickened cyst capsule is hyperintense on T1WI and hypointense on the T2WI. Cyst contents appear hyperintense on T1WI resulting in scolex is not seen. There is an amount of surrounding edema. If cyst degeneration is present peripheral ring enhancement may be present.^[3,4,9,11]

The differential diagnosis of an intramedullary cystic lesion is extensive, including some other cysts such as arachnoid cyst,^[12] ependymal cyst,^[13] neuroenteric cyst,^[14] sarcoidosis,^[15] neoplasms such as ependymoma and infections such as abscess.^[16]

If patient has a history of cysticercosis and/or hails from an endemic region and MRI reveals a cystic spinal cord lesion, the diagnosis of intramedullary cysticercosis should be suspected and be further verified by serologic alterations, subcutaneous nodules and/or changes in the cerebrospinal fluid. The CSF examination often shows increased proteins, a low or normal glucose, moderate lymphocytic pleocytosis and eosinophilia.^[17] Cysticercal antibodies found in CSF either by ELISA or in serum by enzyme-linked immunoelectrotransfer bolt assay have good sensitivity and specificity in cysticercosis diagnosis.^[17,18]

However, our patient lacked history of neurocysticercosis and was not from an endemic region. Therefore, it was difficult to clinically suspect intramedullary cysticercosis prior to treatment. The diagnosis of neurocysticercosis was established based on the histopathological examination. In our case, owing to

increasing neurological deficit and bowel and bladder involvement, surgical treatment was considered a good choice for removing the mass which was producing progressive spinal compression and also to confirm the diagnosis. No significant post-operative neurodeficit was noted in our patient compared to his pre-operative status. We attribute the same to decompression of cord from mass effect of cysticercus and accurately placed midline myelotomy. Our patient showed improvement in motor power and partial recovery of function of the anal sphincter and bladder over a period of next 14 months. However, the results of surgical outcome are mixed. Mohanty *et al.*^[19] in their study have reported a 75% satisfactory outcome after surgery and cysticidal treatment. Early diagnosis and treatment can improve the outcome. Outcomes reported in other series have not been favorable. Sharma *et al.*^[2] have reported that 60% patients acquired improvement after surgery, 25% did not improve and 15% died respectively. In the reports published in recent years,^[3,4,9,20,21] surgical outcome has significantly improved; with no reported death and the majority of patients living a life without special support. Surgery is considered the procedure of choice only when diagnosis is in doubt otherwise medical treatment has its advantages.

Albendazole is medicine that has been proved to be effective in patients with intramedullary cysticercosis since 1996.^[22] Pre-operative adjunctive treatment with albendazole is thought to be helpful to consolidate the lesion and thus induce a clear plane of dissection during surgery. Albendazole is normally used post-operatively as a regular treatment (15 mg/kg/day) for 4-6 weeks, according to the idea that cysticercosis is a generalized disease with focal manifestation. Moreover, albendazole is often used with corticosteroids, because its blood level could be synergistically increased by the latter.^[23] Apart from being used after surgery, Albendazole may also be used independently in the conservative treatment for patients who are clinically suspected as intramedullary cysticercosis and whose neurological course is stable. The potential advantages of medical therapy alone include avoidance of surgery and treatment of surgically unreachable and multifocal cysticercus.^[4,5,11,17]

CONCLUSION

In conclusion, we think that intramedullary cysticercosis represents a diagnostic challenge and neurocysticercosis should also be strongly considered for intramedullary cystic lesions. Surgery is required to facilitate extirpation of the lesion, decompress the cord, confirm the pathological diagnosis and provide a route for definitive therapy.

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