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"NEUROCYSTICERCOSIS WITH MULTILEVEL INTRAMEDULLARY CYSTICERCOSIS OF SPINAL CORD - A RARE CAUSE OF SEIZURE WITH PARAPLEGIA"

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KEYWORDS :

INTRODUCTION :

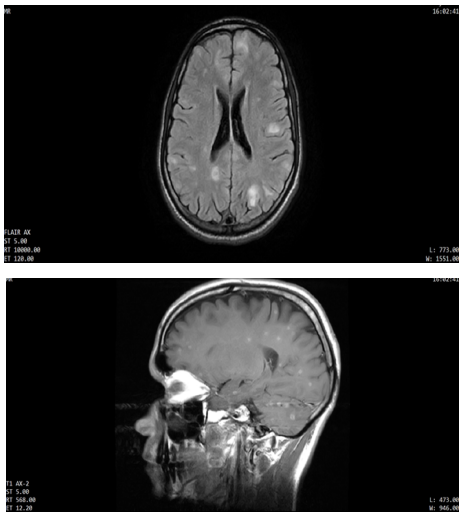
Cysticercosis is the disease caused by larval form of the tapeworm *Taenia solium*. Cysticercosis is the most common parasitic disease of the central nervous system. [1] Neurocysticercosis is a major cause of epilepsy in developing countries.Cysticercal involvement of the spinal cord is rare even in endemic areas and accounts for 0.7 to 5.85% of all cases.[2] Its prevalence may be underestimated, since brain cysticercosis which is more common condition, frequently occurs concomitantly.[3] The vast majority of neurocysticercosis are usually found at meningo-basal (30%), parenchymal (20%), intraventricular (17%), intraspinal (1%), or mixed locations (32%).[4] Intraspinal cases are extremely rare; incidence of spinal cysticercosis varies from 0.7% to 5.85%.[5] Spinal cysticercosis commonly affects the subarachnoid space compared to the spinal cord substance.[6] A purely cystecercosis at multiple level[C4-C5 and D9] of spinal cord as well as in the brain as reported in this paper is quite exceptional. We present a case report of 22 year young boy presented with seizures followed by bilateral lower limb weakness with spastic paraplegia with urinary retention. MRI brain reveals multiple small variable sized T1 hypointense and T2 hyperintense lesions , MRI cervical spine reveals a well defined T2 hyperintense lesion measuring 6x7mm in C4-C5level While MRI dorsolumbar spine revealed few well defined T2 hyperintense intrinsic signals are noted largest measuring 11.5x5.5mm in D9 vertebral level, possibility of NCC in brain and spinal cord at multiple level.

CASE REPORT

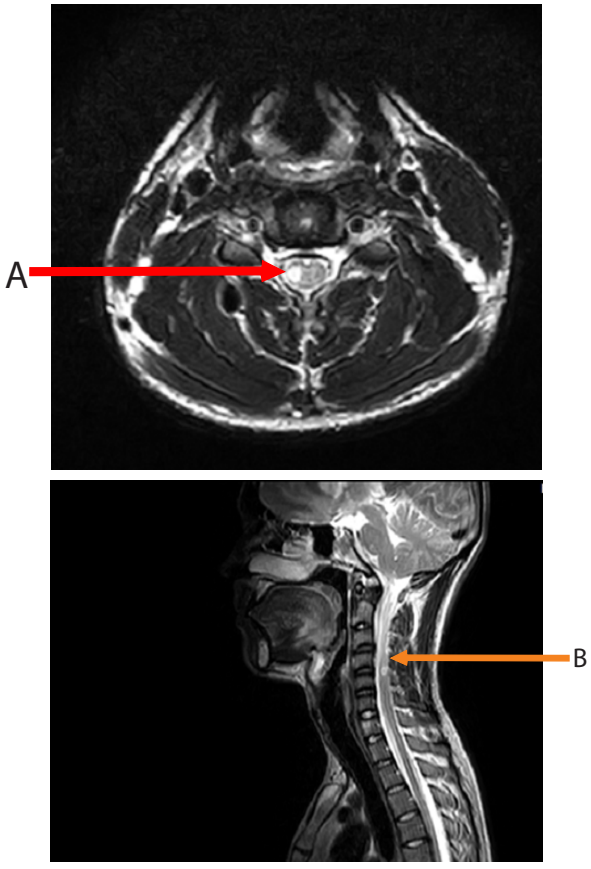
A 22-year-old man was admitted in Neuromedicine department of our institute, complaining of insidious onset, gradually progressive weakness of both lower limbs for 6 days. The weakness initially started in right lower limb followed by involvement of left lower limb within a day. He also complained of urinary retention for last 5 days. He had no history of fever, low backache or radicular pain. Patient had history of seizures 3 days before the onset of weakness, generalized tonic clonic seizures with uprolling of eyeballs and clenching of teeth. Similar episodes of seizures he had 3 years back, however patient had not taken any treatment for that and patient was asymptomatic afterwards for last 3 years. He did not complain of weakness in upper limbs. The patient is a resident in India [Madhyapradesh], which is an endemic region for neurocysticercosis. Clinical examination revealed a young man conscious, alert, oriented, and cooperative, higher mental functions are normal , speech normal, cranium is normal with no any spinal deformity. Neck rigidity,kernig's sign, straight leg raising test negative normal cranial nerve evaluation and intact higher mental functions. Examination of lower extremities showed increased tone with medical research council (MRC) power 3/5 at all major joints. Deep tendon reflexes were brisk with bilateral positive Babinski's sign. On sensory examination, there was decreased sensation for pain and touch in both lower extremities. Joint position and vibration senses were relatively preserved. Examination of upper extremities did not reveal any abnormality.

A clinical diagnosis of myelopathy was made and magnetic resonance imaging (MRI) of cervical spine was performed [Figure 1], which revealed a well defined T2 hyperintense intrinsic signal noted in cord at C4-C5 level in right half measuring 6x7mm. MRI dorsolumbar spine revealed few well defined T2 hyperintense intrinsic signals are noted in spinal cord, largest measuring approx. 11.5x5.5 mm is seen at D9 vertebral level. Possibility of NCC of spinal cord. MRI brain [contrast] suggestive of multiple small variable sized T1 hypointense and T2 hyperintense lesions showing smooth post contrast ring enhancement with mild perilesional edema are noted diffusely involving brainstem, corpuscallosum, basal ganglia, bilateral cerebral and cerebellar hemispheres. Few eccentric nodules noted in few of lesions suggestive of NCC [scolex] of brain. The CSF examination shows increased proteins, a low or normal glucose, moderate lymphocytic pleocytosis and eosinophilia.7 serum ELISA for neurocysticercosis was found positive.

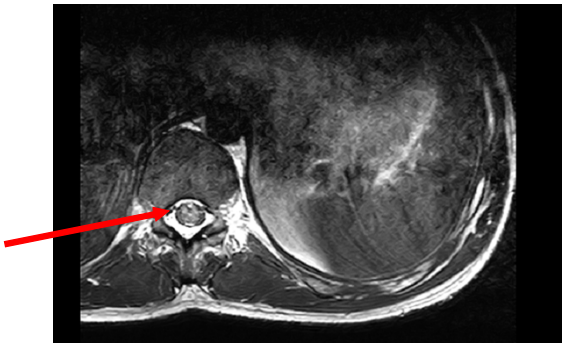
The patient recovered well from medical treatment and was ambulating with decreased tone and sensory improvement in both extremities.



Axail flair (A) & Sagittal post contrast (B) Magnetic resonance imaging (MRI) showing multiple small variable size T2 Flair hyperintense lesion showing smooth post contrast enchancement noted diffusely involving brain parenchyma eccentric nodule noted in few of the lesion



Axial (A) & Sagittal (B) T2 Weighted magnetic resonance imaging (MRI) of cervical spine showing well defined T2 Hyperintense lesion in the spinal cord at C4 - C5 vertebral level.



Axial T2 Weighted MRI image shows few well defined T2 hyperintense lesion in spinal cord at D9 Vertebral level

DISCUSSION

Spinal cysticercosis is an unusual form of cysticercosis with an incidence of 0.7% to 5.85% even in endemic regions. This low incidence may be due to inability of the cysticerci to travel to the spinal subarachnoid space. The spinal cord involvement is presumed to occur due to spread of the eggs of *Taenia solium* through arterial blood to the spinal cord,[7] which leads to local development of the larval form in the spinal cord manifesting as cysticercosis.CNS cysticercosis affects men and women equally. The peak incidence is between the third and fourth decades of life. It typically involves the brain parenchyma, intracranial subarachnoid space, or ventricles. Spinal cysticercosis is rare; it may be leptomeningeal, intramedullary, or epidural. Among these, leptomeningeal is the most common, intramedullary is rare, and epidural is extremely rare.[8] Most cases of spinal cysticercosis are usually associated with cerebral cysticercosis. Isolated spinal cysticercosis either intramedullary or extramedullary is extremely rare.[9]

Clinical manifestations of spinal cysticercosis depend on the number and topography of lesions, individual's immune response to the parasite, and the presence or absence of previous infestations. Clinically, the most common manifestation of spinal neurocysticercosis is root pain and progressive weakness in contrast to the parenchymal neurocysticercosis that manifests with epileptic seizures; subarachnoid neurocysticercosis that manifests with headache while intraventricular neurocysticercosis manifests as subacute or intermittent syndrome of intracranial hypertension. Neurological damage in spinal intramedullary cysticercosis is attributed to the following factors: (i) mechanical compression caused by the cyst, (ii) due to the cord edema as a result of inflammation caused by degenerating larva remnants, and (iii) gliosis.[10]

Sotelo and Carpio had defined three clinical stages of neurocysticercosis, namely, active, transitional, and inactive neurocysticercosis. Escobar has also defined four pathological stages of neurocysticercosis. They are vesicular, colloidal vesicular, granular nodular, and nodular calcified. Vesicular is active form, colloidal vesicular and granular nodular represent transitional stages while nodular calcified stage is an inactive stage of neurocysticercosis.[11] Clinical suspicion of spinal cysticercosis is difficult, especially when there is neither previous history of any parasitic infestation nor associated cerebral neurocysticercosis. In patients with clinical suspicion of neurocysticercosis, cerebrospinal fluid (CSF) examination should be done as it provides a reliable evidence of inflammation; further immunoblot test may be performed to confirm the diagnosis of neurocysticercosis.12] Apart from CSF studies, MRI is one of the most useful diagnostic tools providing useful information in the evaluation of spinal neurocysticercosis patients. MRI, in addition to the diagnosis also provides precise information about the disease activity and its location, carrying important therapeutic implications.[13]. In cases of spinal cysticercosis, the entire neuraxis should be evaluated to detect any additional lesion. In according to the existing literature , in our case cysticercosis is noted both in brain and in multiple level of spinal cord.

The clinical features of spinal cysticercosis may be paraparesis or quadriparesis, sensory loss, autonomic dysfunction involving bowel and bladder, radicular pain and paresthesias. Clinical presentation is dictated by the location of lesions within spinal cord, number of lesions, size of lesion and presence or absence of local inflammation. Our patient presented with features of spinal cord compression, without any inflammatory features. The clinical features were indistinguishable from any other intramedullary mass like astrocytoma or tuberculoma. MR imaging can help in diagnosis of these lesions on which the cysticercal cysts appear homogeneously hypointense on T1 and hyperintense on T2 with minimal surrounding edema.

Similar cases have been described previously in literature. Guedes-Corrêa *et al*. described a women with an intramedullary cyst of the conusmedulllaris region, who presented with low backache in the absence of any other neurological features.[14] Agale et al. presented a patient with intraduralcysticercosis which resulted in spinal cord compression, causing spastic paraparesis.[15] Singh et al. noted cysticercosis involving cervico-dorsal spinal cord in a young man with seizures due to cerebral neurocystiercosis.[16] Torabi et al. described a 35-year-old man with cerebral neurocysticercosis who presented with caudaequina and Brown-Sequard syndrome due to multilevel intramedullary spinal cord cysticercal lesions and cerebrospinal fluid findings of eosinophilic meningitis.[17]

There are various therapeutic options for spinal neurocysticercosis. Medical treatment can be considered in those cases with stable neurological status or diagnosed preoperatively by CSF and serum enzyme linked immunoelectric transfer blot assay for antibody.[18] There are a few anecdotal case reports of successful medical therapy of spinal cysticercosis with albendazole and steroids.[19,20] .Albendazole is a medicine that has been proved to be effective in

the patients with intramedullary cysticercosis.Albendazole is normally used postoperatively as a regular treatment (15mg/ kg/ day) for 4 to 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[21]. Except for being used after surgery, Abendazole also could be used independently in the conservative treatment for the patients whom are highly suspected as intramedullary cysticercosis and whose clinical courses are stable. The potential advantages of medical therapy alone include avoidance of surgery and treatment of surgically unreachable and multifocal cysticercosis[22] Our patient was managed by medical treatment with iv dexamethasone and albendazole patient improved during hospitalization from power 3/5 to 4/5.

Spinal NCC should be considered in differential diagnosis of spinal lesion, though isolated cysticercosis of spine is difficult to suspect; however, correlation of clinical findings, history of endemic areas origin and typical MRI findings can help in making early diagnosis.

CONCLUSIONS

We conclude that cysticercosis involving spinal cord is an unusual manifestation of neurocysticercosis, and its diagnosis needs a high index of suspicion. This diagnosis should be considered in a patient from endemic region with a cystic spinal cord lesion. The CSF examination often shows increased proteins, a low or normal glucose, moderate lymphocytic pleocytosis and eosinophilia. Cysticercal antibodies found in CSF either by ELISA or in serum by enzyme-linked immunoelectric transfer bolt assay have good sensitivity and specificity in cysticercosis diagnosis

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