Case report

Focal spinal intramedullary cysticercosis

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Abstract. Neurocysticercosis is the most common parasitic infestation of the CNS. Most authors have reported the incidence of spinal neurocysticercosis as only 1.5-3% of all cases, even in endemic areas. Spinal neurocysticercosis represents a distinct clinical entity that usually appears due to mass effect on the spinal cord. Most cases occur in the spinal subarachnoid space and cysticercosis in intramedullary location is extremely rare. The authors present a case of focal intramedullary spinal cysticercosis. The authors highlight the strategy for successful treatment of focal lesions in the intramedullary location. (www.actabiomedica.it)

Key words: Spinal, intramedullary, cysticercosis

Introduction

Worldwide, cysticercosis is the most common parasitic infection affecting the CNS (1). In endemic regions, the incidence may reach 4% of the general population. The disease is predominantly intracranial, even if in endemic areas the incidence of spinal cysticercosis has been reported as only 1.5-3% by most authors (1). Most cases of spinal cysticercosis are located in the subarachnoid space. Intramedullary location for cysticercus is extremely rare with less than 50 cases reported in literature (1-4). The location of mass lesion, its size, and the inflammatory response generated by cyst breakdown are important factors in the management of spinal cysticercosis. We operated a case of intramedullary cysticercosis at D5-6 disc level. Steroids and albendazole were administered.

Case record

A 35-year old male presented with gradual weakness in the lower limbs (right preceding left) for 2 years leading to inability to walk, numbness from lower chest to foot for $1^1/_2$ year, and difficulty in urinating for 3 months. On examination, the patient had power 2/5 in the lower limbs, wasting of his lower half of body with a sensory level at T7 dermatome, and exaggerated deep tendon reflexes in his lower limbs.

MRI (Magnetic Resonance Imaging) revealed a solitary focal well-defined rounded intramedullary mass lesion (size 11 x 9 mm) at D5-6 disc level. The lesion was rounded with well defined margins causing focal cord expansion. The lesion showed T2 hypointense rim and central T2 hyperintensity and was isointense on T1 weighted images. Mild perifocal T2 hyperintensity suggestive of edema, but no other focal lesions were observed. Rest of spinal cord is seen normally. Subarachnoid CSF space was normal. No evidence of cord compression was seen (Figure 1). Screening MRI of the brain revealed no abnormality. The diagnosis of intramedullary mass lesion was made.

Laminectomy with midline myelotomy was performed and a cystic swelling protruded from inside the cord. On opening, 1-2 drops of thick yellow colored fluid came out. The cyst was removed, irrigation performed and dura was repaired.

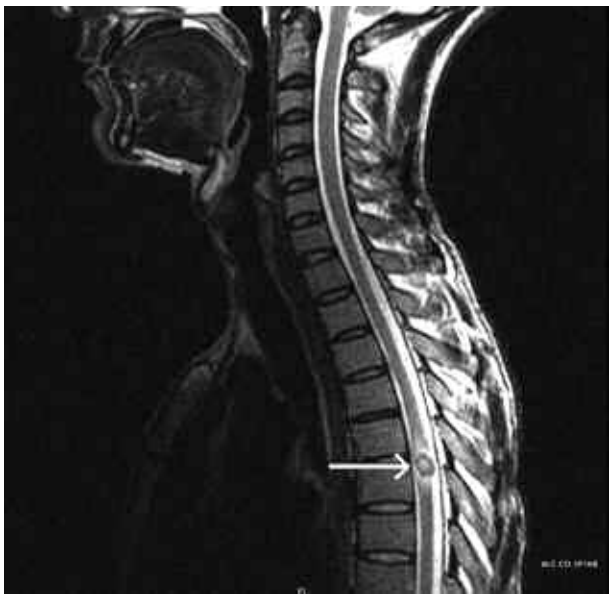

Figure 1. T2WI MRI showing a solitary focal well-defined rounded intramedullary mass lesion (white arrow) at D5-6 disc level with a thin hypointense peripheral rim, hyperintense core and mild perifocal edema causing focal cord expansion

Histology showed a wavy eosinophilic band showing degenerated tegument of *cysticercus cellulosae*. Underneath subcuticular cells with one or two suckers were seen. Some degenerated scolices were also seen (Figure 2).

Postoperatively, albendazole 400 mg BD with steroids were administered. At 12 months of follow-up, the patient satisfactorily responded to the treatment and power in his right lower limb improved to 3/5 and in left lower limb improved to 4/5. Bowel control was satisfactory and the patient was able to pass feces once per day. However, bladder control was not adequate and the patient still needs intermittent catheterization.

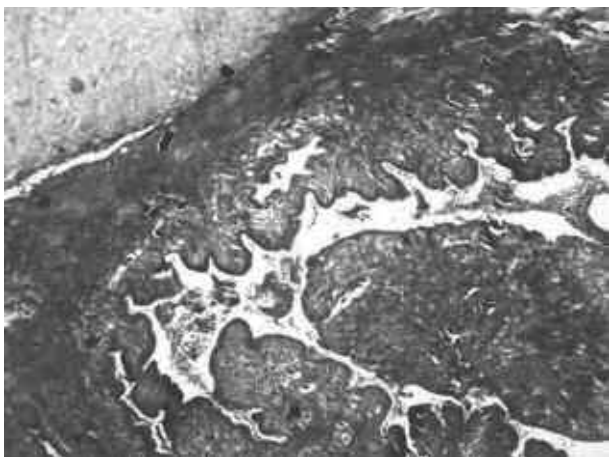

Figure 2. Wavy eosinophilic band showing degenerated tegument of *cysticercus cellulosae* and subcuticular cells with some degenerated scolices

Discussion

Spinal cysticercosis can be leptomeningeal, intramedullary or epidural. Leptomeningeal is the most frequent, intramedullary is quite rare and epidural is an extremely rare form (5). Spinal involvement is quite rare and the migration of the cysticercus through the ventriculo-ependymal pathway and hematogenous dissemination has been hypothesized to be the possible mechanism (1). However, Queiroz *et al.* did not find any evidence for ependymal route of spread of intramedullary cysticercosis (6). Rokitansky, in 1856, firstly described intramedullary cysticercosis (5). Because of limited space in the spinal canal, mass effect of these lesions is poorly tolerated necessitating for aggressive management (1).

Blood flow to the brain is approximately 100-fold greater than that of the spine explaining the lower incidence of spinal cysticercosis. In the spine, thoracic cord has higher incidence due to high blood flow in this segment (2, 3, 6). Queiroz et al. estimated the location of cysticerci in spine as: cervical-34%, thoracic-44.5%, lumbar-15.5% and sacral-6% (6).

MRI is the investigation of choice. Mathuriya et al. described MRI findings for various stages of intramedullary cysticercosis (4). Usually, MRI is described as hypointense rim with hyperintense core on T2WI and hypointense or isointense lesion on T1WI as is our case (4). However, these are not specific and the same changes can also be present in neoplastic, inflammatory, demyelinating, vascular, and granulomatous diseases (2, 7). The entire neuraxis should be evaluated to find additional lesions. In the present case, an isolated intramedullary cystic lesion was demonstrated at D5-6 with absence of cranial cysticercosis. This is in contrast with the previous hypothesis that concomitant intracranial lesions are present in all patients with spinal cysticercosis (7). Our

finding is supported by Parmar et al. who found only 2 patients with brain neurocysticercosis among 6 patients with intramedullary cysticercosis (8). Perifocal edema was present in all of their 6 patients as in ours.

As long as the cysticercus remains viable, there is relative host immune tolerance. Massive antigen exposure only occurs when the parasite dies. Then intensification of the immune response/inflammatory reaction occurs along with the appearance or worsening of symptoms. This may be the reason of partial recovery of our patient since dead parasite was recovered surrounded by moderate inflammatory reaction and partial response to steroids.

Surgery is the best method for the confirmation of diagnosis and treatment (5). It eliminates the compressive element and should be performed before cord changes take place (2, 4). Results of surgical outcome are mixed. Mohanty et al. reported improvement in 7/8 patients (9). Outcomes reported in other series have not been favorable. Sharma et al. found improvement in only 60%, no improvement in 25% and deaths in 15% (10). A variety of motor, sensory and sphincter related problems were seen in all survivors. They hypothesized that sensory and urological improvements were likely due to decreased mass effect following decompression, but the refractoriness of motor deficits might have been a result of parenchymal gliosis from toxic parasitic metabolites. However, our patient showed improvement in motor power. Sphincter control was variable with better control for bowel movements and worse for urinary sphincters.

Albendazole or praziquantel, with or without steroids are used. Albendazole is preferred because its blood levels are improved by corticosteroids, whereas those of praziquantel are diminished (5, 7).

Finally, we conclude that spinal intramedullary cysticercosis represents a diagnostic challenge and surgery is required to decompress the cord, confirm the diagnosis and provide a route for definitive therapy. Patient recovery may be variable. Despite promising reports, the safety and efficacy of medical treatment remains unproved.

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