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Isolated Cysticercosis of the Peroneous Longus Muscle: A Rare Pseudotumour

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Abstract: Human cysticercosis is endemic in the developing countries caused by the larvae of pork tapeworm Taenia solium. This infestation commonly involves the central nervous system, eyes, subcutaneous tissue and muscle. Isolated muscular cysticercosis is very rare and often difficult to diagnose. We present a rare case of isolated muscular cysticercosis involving the peroneous longus muscle in a 45 year old female. In endemic regions, it must be kept as a differential diagnosis in cases presenting with non specific symptoms. Adequate diagnosis, proper medications and complete surgical excision are the recommended treatment modalities.
Keywords: Isolated, Cysticercosis, Peroneous longus

INTRODUCTION

Human cysticercosis is a zoonotic disease caused by the larvae of pork tapeworm *Taenia solium*. This disease is endemic in the developing countries in South East Asia especially India [1]. The occurrence of the cysts in the order of frequency is in the central nervous system, the vitreous humor of the eye, the striated muscle, subcutaneous tissue and very rarely other organs. The central nervous system is involved in about 60-70% of cases and the most common presenting symptom is epilepsy. Muscular cysticercosis is generally associated with central nervous system involvement [1, 2]. Isolated muscular cysticercosis is very rare and often difficult to diagnose [2]. We present a rare case of isolated muscular cysticercosis involving the peroneous longus muscle in a 45 year old female.

CASE REPORT

A 45 year old female presented to the orthopaedics outpatient department with a history of pain and swelling over lateral aspect of left leg of 3 years duration. The pain was dull aching in nature and aggravated on walking and physical activity. The patient had no associated history of fever, weight loss, night sweats or any other constitutional symptoms. The general physical health of the patient was good and her medical history and family history were non contributory. However, the patient gave history of consumption of mixed diet with occasional intake of pork.

On physical examination there was no obvious swelling over the lateral aspect of the left leg. The skin overlying the swelling was normal. On deep palpation tenderness and localised swelling were located on lateral aspect of left leg over peroneus tendon. The swelling was not adherent to the overlying skin and was present in the deep muscular plane.

X ray of the left leg did not reveal any abnormality. The magnetic resonance imaging (MRI) revealed well defined space occupying lesion in peroneus longus muscle. The lesion characteristically contained rounded cyst of about 15mm by 23mm in size. The cyst revealed low signal intensity on T1 image and high signal intensity on T2 image indicative of fluid content (Fig. 1).

The routine haematological investigations of the patient revealed a haemoglobin of 10.2 gm/dl with a total leucocyte count of 5600/mm$^{3.}$ The erythrocyte sedimentation rate (ESR) was 15 at the end of the 1st hour.

An excision biopsy of the lesion was performed after anaesthetic clearance and adequate per-operative assessment. The excised tissue was sent for histopathology. The histopathological evaluation revealed a degenerated wavy membrane with a fibrous pseudo-capsule surrounded by intense inflammatory infiltrate. Multiple histopathological sections failed to reveal scolex and cuticle. Based on the MRI findings and in conjunction with histopathological evaluation, a diagnosis of degenerated cystic parasitic lesion possibly cysticercosis was rendered (Fig. 2). There was no evidence of disseminated or intracranial cysticercosis. The patient was prescribed anti-helminthic medications for 3 weeks duration. The patient responded very well

and is currently asymptomatic after 2 months of follow up.

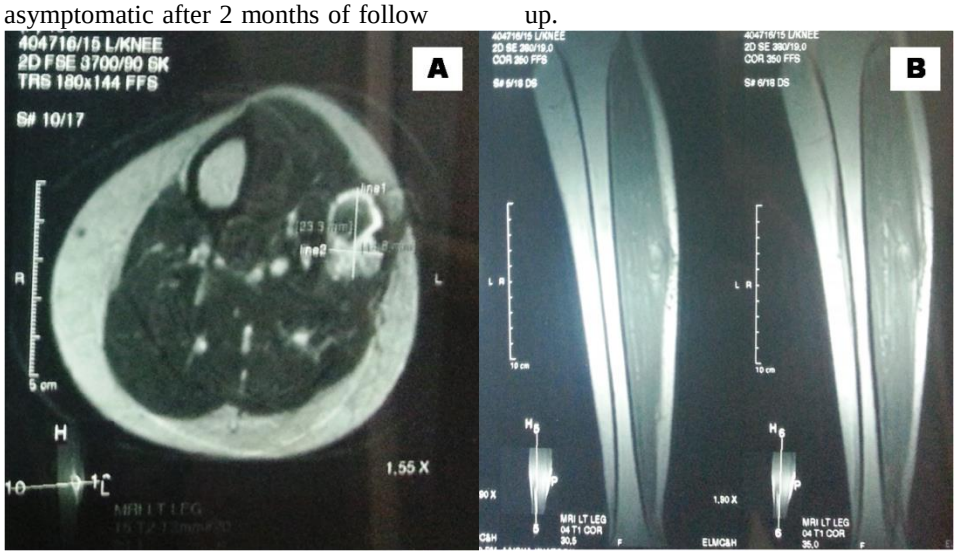

Fig. 1 (A and B): Magnetic Resonance Imaging (MRI): T1 and T2 weighed images in coronal and axial plane demonstrating a cystic lesion with fluid content.

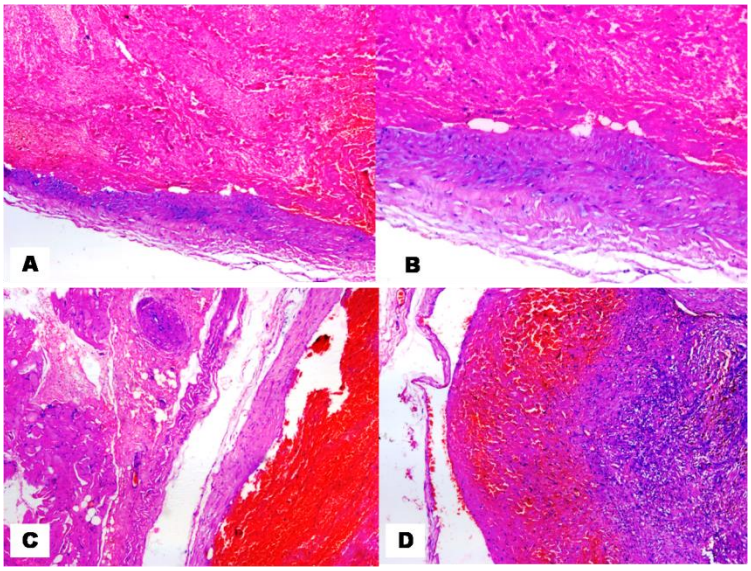

Fig. 2: (A and B): Histopathological section demonstrating degenerated wavy membrane with a pseudo-capsule [A: Hematoxylin and Eosin x 100; B: Hematoxylin and Eosin x 200] (C): Histopathological section demonstrating the intramuscular location of the cystic lesion [Hematoxylin and Eosin x 100] (D): The fibrous capsule surrounded by intense chronic inflammatory infiltrate [Hematoxylin and Eosin x 100]

DISCUSSION

The ingestion of *T. solium* eggs occurs by consumption of fecally contaminated vegetables, food or water, as well as self-contamination by reflux from the intestine into the stomach or by contaminated hands. Humans are the definitive host for this parasite [1, 3]. After ingestion of the parasite, the gastric enzymes lyse the outer shell of the parasitic cyst leaving the scolex (head) behind. The scolex has suckers and hooks (rostellum) that aid in attachment to the intestinal wall. Once the parasite attaches itself to the intestinal wall, the scolex proliferates and produces an adult tape worm. The adult tapeworm becomes gravid and produces eggs. These eggs then detach from the adult tape worm, pass through the anus and are excreted in faeces. When pigs ingest the eggs, the life cycle begins again. Normally pigs are the intermediate host. Rarely when humans become the intermediate host, they manifest the clinical signs and symptoms of cysticercosis. When humans ingest the eggs, the eggs are lysed in the stomach to release oncospheres. These oncospheres then penetrate the small bowel mucosa and enter the blood stream to reach various organs like the brain, subcutaneous tissue, eyes and muscle [3-5].

Most cases of isolated muscular cysticercosis remain asymptomatic and unnoticed for years. Rarely after death of the worm in the cyst or antecedent trauma

that leads to release of antigens responsible for inflammation, the lesion becomes symptomatic [5]. In our case there is no history of trauma so the possible cause of symptomatic presentation was probably the death of the worm within the cyst.

There are 3 different clinical manifestations of muscular cysticercosis that include the myalgic or myopathic type, the nodular or mass forming type or the pseudohypertrophy type in which there is formations of single or multiple cysts [1, 5, 6].

In the myalgic type, manifestations generally occur when there is trauma causing sudden release of antigens responsible for inflammation. When there is death of the worm within the cyst there is slow release of antigens that leads to fibrosis and collection of chronic inflammatory cells that forms a pseudotumour causing pseudohypertrophy. In our case, the death of the helminth within the cyst was possibly responsible for formation of a pseudotumour with chronic non specific symptoms. This also explains the absence of scolex and cuticle in the histopathological evaluation. The dead worm was possibly degenerated by the host mediated immune response [1, 6].

The important differential diagnosis in cases of muscular cysticercosis presenting as pseudotumours includes lipomas, neurofibromas, epidermoid cysts and tubercular masses. These lesions can be differentiated by radiological and histopathological evaluation. Routine laboratory testing is generally unremarkable in cysticercosis. The diagnosis rests on characteristic imaging findings supported by serologic tests such as enzyme-linked immunosorbent assay (ELISA) and enzyme-linked immunoelectrotransfer blot (EITB). In most cases, the diagnosis is established based on history along with radiological findings [1, 6, 7].

X ray findings are generally non contributory in cases of cysticercosis. Computed tomography (CT) scan is generally useful in cases of neurocystecercosis. The best radiological modality for soft tissue and muscular cysticercosis is MRI which is generally useful in visualisation of peri-lesional edema and degenerative changes with the cyst. The typical MRI features are fluid equivalent signal and peripheral rim enhancement. The findings may vary according to the growth stage of the parasite and the immune response of the host [5, 6]. In our case a well defined cystic lesion with fluid content was identified with the absence of scolex that was indicative of a degenerated parasitic cyst.

Treatment of cysticercosis depends on the site of involvement. Isolated muscular or subcutaneous cysticercosis require no specific treatment unless it is painful, which may necessitate excision. In our case excision was done as the lesion formed a pseudotumour associated with pain and swelling. Recently, case reports have advocated non-operative management, even for painful masses, with antihelminthic medication and oral steroid therapy. Albendazole acts by inhibiting microtubule formation. The loss of cytoplasmic microtubules blocks glucose uptake in the larval and adult stages of the parasites, thereby depleting their energy reserves and causing death. Steroids are commonly used as a cover for the inflammatory response to the necrotic pathway [4-8].

CONCLUSION

Isolated muscular cysticercosis is a diagnostic dilemma for treating physicians and may present in a variety of forms. We present a rare case of isolated cysticercosis in the peroneous longus muscle. In endemic regions, it must be kept as a differential diagnosis in cases presenting with non specific symptoms. Adequate diagnosis, proper medications and complete surgical excision are the recommended treatment modalities.

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