CASE REPORT

An Unusual Presentation of Cysticercosis as Cold Abscess in Axillary Region: A Case Report

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ABSTRACT

Introduction: Cysticercosis is a common tropical disease caused by dissemination of the larval form of the pork tapeworm, *Taenia Solium*. It is the most common parasitic infection of soft tissue and have predilection for central nervous system, skeletal muscles and eyes.

Case presentation: We report a case of cysticercosis which presented as a cold abscess in right axillary region in immunocompetant female patient.

Conclusion: An isolated axillary abscess can be manifestation of cysticercosis which can be diagnosed by FNAC and ultrasonography and poses a diagnostic challenge clinically, as being a differential diagnosis of cold abscess. Surgical excision is treatment modality for such cases.

Keywords: Cysticercosis, *Taeniasolium*, Axilla.

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INTRODUCTION

Cysticercosis is a parasitic infection caused by the larval form of pork tapeworm, *Taenia solium*. Prevalence of cysticercosis/ taeniosis is high in most of the developing countries like Africa, Brazil, Mexico, Korea, China, India and other South Asian countries. Coexistence of poor sanitary condition and domestic pig raising without veterinary control or surveillance system is main reason for high prevalence in developing countries.¹ Cysticercosis is categorized as either neurocysticercosis or extraneuralcysticercosisis involving subcutaneous tissue, striated muscles, heart, liver, lung, eyes and other soft tissues. Neurocysticercosis is the most common parasitic infection of the central nervous system (CNS). Presentation of cysticercosis as a solitary muscular and soft tissue involvement without central nervous system involvement is rare and often presents a diagnostic challenge. Role of Imaging like ultrasonography and MRI, is crucial for establishing the diagnosis in cysticercosis of subcutaneous tissue and muscles.² We report a surgically treated case of an isolated axillary cysticercosis for its uncommon site and presentation as a cold abscess in axilla posing a diagnostic challenge, as in India tuberculosis is common and frequently manifest as tubercular lymphadenitis and cold abscess.

CASE PRESENTATION

A 26 year old female presented with a history of gradually progressive lump in the right axillary region for the past four months duration. There was no history of fever, chronic cough, tubercular contact, trauma or other breast problems. On physical examination, an oval 4x3 cm size mass was palpable which was firm, freely mobile and non-tender in the right posterior axillary region. The skin overlying the swelling and local temperature was normal.Contra-lateral axilla and bilateral cervical examination not revealed palpable lymph nodes. On bilateral breast examination no abnormality was detected. A clinical diagnosis of lipoma/ cold abscess was suspected and routine investigations were advised along with ultrasound scan and FNAC. All the routine blood investigations (TLC/ DLC/ ESR) and chest X-ray (PA- view) were normal. Ultrasound scan revealed a well defined cystic lesion measuring 3.5x3 cm along the posterior axillary fold muscles with pericystic inflammation.

FNAC was performed in pathology lab and fluid was aspirated which was smeared over the slides, alcohol fixed and stained with Hematoxylin and Eosin. Smears
revealed numerous eosinophilic/ basophilic rounded structures, few of which appear calcified suggesting inflammatory pathology ruling out clinical diagnosis of lipoma/ cold abscess.

Later, the mass was excised and sent for histological evaluation. Grossly, the mass was brown, soft to firm and filled with pus, measuring approx 3x2.5x2.5 cm³. On microscopic examination it showed parasitic cyst wall of having a convoluted appearance (Fig. 1; H&E x 40) and is composed of three layers, thick eosinophilic outer cuticular layer with knob like projection, thin middle cellular layer and innermost loose reticular layer (Fig. 2; H&E x 100). Higher magnification of cyst wall showed, tissue reaction comprising of eosinophil, lymphocytes, histiocytes and fibrosis (Fig. 3; H&E x 200) and pale fibrillary sheath of cysticercus cellulose with granular nuclei and few entrapped histiocytes (Fig. 4; pap x 200).

**Figure-1:** Parasitic cyst wall (H&E X 40); **Figure-2:** Parasitic cyst wall (H&E x 100)

**Figure-3:** H&E X 200; **Figure-4:** Wall (Papanicolaou stain x 200)

**DISCUSSION**

Human cysticercosis is caused by dissemination of embryos of *Taenia solium*. Humans are the only definitive host while both humans and pigs can act as intermediate hosts. The infestation of human intestine with adult tape worm is known as taeniasis. Adult worm sheds gravid segment laden with eggs in the stool which can be ingested by free-ranging pigs feeding on human feces. The embryo from pig gut migrates to muscles and other sites through systemic circulation, where it develops into infective larval stage, the cysticercus, which is consumed by human in pork meat. In the alimentary canal of human, cysticercosis releases the scolex which anchors in gut wall and develops in the adult tapeworm.

Cysticercosis is acquired as a feco-oral infection when a human accidentally swallows eggs of *T. solium* excreted in the feces, through ingestion of contaminated food, water or alternatively by auto-infection by contaminated fingers in a tapeworm carrier.³ The egg hatches in the small bowel of the human host, and the oncosphere penetrates the intestinal mucosa and are then spread through the systemic circulation to all parts of the body. Almost any tissue may be infected, but preferably central nervous system and skeletal muscle. In around 3 months, the oncospheres get established in the tissues as fluid-filled cysts thereby evading the immune response of the host.⁴

Most of the patients with extracranial cysticercosis present as solitary or multiple subcutaneous nodules on the trunk, upper arm, neck, tongue, face, and breast has been reported in this order of frequency. The intramuscular and subcutaneous cysticercosis is characterized by multiple, mobile, firm, subcutaneous nodules of size 1 to 2 cm with normal overlying skin and most of them are usually asymptomatic.⁵ During the death of the larva, there is leakage of fluid from the cyst. The resulting acute inflammation may result in local pain and myalgia. There may be intermittent leakage of fluid of cyst during degeneration of cyst, which leads to chronic inflammatory reaction with collection of fluid around the cyst. The differential diagnosis of soft tissue cysticercosis includes lipomas, epidermoid cysts, tuberculous lymphadenitis, neuroma, neurofibroma, pseudoganglion, sarcoma or myxoma.⁶

The intramuscular and subcutaneous cysticercosis is quite difficult to differentiate from benign mesenchymal tumors and lymphadenitis on clinical ground alone. The role of ultrasonographic studies in diagnosis of isolated soft tissue cysticercosis has been confirmed. The most common ultrasonographic feature is presence of an oval or rounded cystic lesion with well defined margin, containing a scolex within and with surrounding abscess. On CT scan or MRI, active cysticerci can be identified as hypodense lesion with central hyperdense scolex.¹ FNAC may be diagnostic when the fragments of larval cuticle and parenchyma are identified in the smears. Presence of scolex in cytology smear is an uncommon finding. But most of the time, suspicion about parasitic lesion starts with the presence of eosinophils, neutrophils, palisading histiocytes and giant cells in an aspirate.
Histology shows the outer cuticular layer of the parasite which appears smooth, hyalinized and rounded wavy folds and scolex with hooklets with hyaline membrane surrounding it. The inflammatory reaction consists mainly of eosinophils with varying proportions of other polymorphs and epithelioid cells and giant cells. Serodiagnosis is not much helpful in diagnosis of cysticercosis unlike other parasitic disease, due to cross-reaction to other parasite and nonspecific binding. There are two available serological tests to detect cysticercosis, the enzyme-linked immunoelectrotransfer blot (EITB), and commercial enzyme-linked immunoads. The preferred test is immunoblot, because its sensitivity and specificity have been well characterized in some studies.

Medical treatment for tapeworm carriers is praziquantel as such person are common source of infection in both human and pigs. For asymptomatic, nonviable cysticercal lesion in subcutaneous tissue and muscle, no treatment is required. For solitary symptomatic lesion, surgical excision is the preferred modality of treatment. As solitary muscular and soft tissue cysticercal involvement is a rare disease per se, therefore central nervous system should be ruled out if systemic involvement is suspected. For neurocysticercosis, symptomatic therapy is the mainstay for the treatment and includes analgesics, corticosteroid and antiepileptic drugs. Anti-helminthic therapy, either albendazole or praziquantel for neurocysticercosis may lead to exacerbation of the neurological symptoms acutely because it kills viable cysts and provokes an intense inflammatory response. To mitigate these effect, it is recommended to prime the patients with corticosteroids before starting cysticidal drugs.

CONCLUSION

Cysticercosis is more common than usually thought, especially in endemic areas. Differential diagnosis of any soft tissue nodular swelling should include cysticercosis cellulose. Presentation of axillary cysticercosis as cold abscess is rare and poses a diagnostic challenge clinically. Early diagnosis can be made by use of FNAC and ultrasonography, however in inconclusive cases; excisional biopsy is the diagnostic and mainstay of treatment for solitary lesion.

REFERENCES

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