A Rare Case Of Oral Cysticercosis

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Abstract

Cysticercosis is caused by the larval stage of Taenia Solium. Taenia solium has a complex 2-host life cycle. It is a hermaphrodite cestode that inhabits the human small intestine of those individuals who have ingested raw or inadequately cooked pork infected with their viable larvae (cysticerci). Although oral involvement by cysticercosis is common in swine, this location is rare in humans. We present a case of cysticercosis on the tongue of a 25 year old Indian male.

INTRODUCTION

Cysticercosis is caused by the larval stage of Taenia Solium. Taenia solium has a complex 2-host life cycle. It is a hermaphrodite cestode that inhabits the human small intestine of those individuals who have ingested raw or inadequately cooked pork infected with their viable larvae (cysticerci). The scolex of the larva evaginates from the cyst inside the small intestine and attaches to the bowel wall. After 3 months, the adult tapeworm develops within its human definitive host, producing a condition known as taeniasis and thereafter begins forming proglottids, which are frequently detached from the distal end of the worm and are excreted in the feces. Each proglottid contains 50,000 to 60,000 fertile eggs, which can remain viable for a long time in water, soil, and vegetation. Cysticercosis develops when these eggs are ingested by humans and pigs (intermediate host), and oncospheres (embryos) are liberated by the action of gastric acid and intestinal fluids and actively cross the bowel wall, enter the blood stream and infest various other tissues and organs where they develop into larval vesicles or cysticerci. In humans, this potentially fatal parasitic disease mainly occurs as a result of the ingestion of contaminated food or polluted drinking water, but it may also develop by fecal-oral contamination in tapeworm carriers.1

Although the disease is more common in endemic areas like Latin America, Asia, Africa and Easter Europe, its incidence is also increasing in developed countries as a result of migration of infected persons and frequent travel to and from endemic areas.2 In humans, cysticerci are most commonly located within the central nervous system (CNS), where it produces a pleomorphic clinical disorder known as neurocysticercosis (NCC), but it may also localize primarily in a variety of tissues, including muscle, heart, eyes, and skin. Although oral involvement by cysticercosis is common in swine, this location is rare in humans.3,4 We hereby present a case of cysticercosis on the tongue of an Indian male.

CASE REPORT

A 25 year old male presented with a swelling on the right lateral border of the tongue (Figure 1). During anamnesis, the patient reported that the lesion was present since six months with no associated pain. Intra oral examination revealed that the lesion was spherical in shape, 1x 1 cm in size, firm, compressible, smooth surfaced and movable with the overlying tissue. A clinical differential diagnosis of mucocele, sialocyst, lymphangioma and minor salivary gland tumour was given. The lesion was aspirated and surgically excised under local anaesthesia.
Microscopic examination of the aspirate showed RBCs without any host response (Figure 2). Histopathology of the excised tissue revealed a thin capsule of fibrous connective tissue surrounding a cystic cavity, which contained cysticercosis cellulosae (larval form of Taenia solium). The larva composed of a scolex towards the cephalic end caudal to which a duct like invagination segment was lined by a homogeneous membrane (Figure 3). Areas of dystrophic calcifications were also present (Figure 4). Cyst wall had a variably thick tegument raised into projections (Figure 5). Based on these findings, a diagnosis of cysticercosis was made.
DISCUSSION

Cysticerci are uncommon in the oral cavity of humans where they appear as cystic nodules that may rupture and heal uneventfully. In swine this location is common. Authors suggest that a high muscular activity and metabolic rate of oral tissues in humans might act against the lodgement and development of cysticercosis in this location.

According to literature, oral cysticerci usually elicit a clinical diagnosis of mucocele, or a benign tumour of mesenchymal origin, such as lipoma, fibroma, hemangioma, granular cell tumour, or a minor salivary gland tumour. Delgado et al. through experience proposed that oral cysticerci are firm nodules on palpation because of its high intraluminal pressure, and therefore considered neither lipoma nor hemangioma as clinical possibilities.

Routine sections stained with haematoxylin and eosin may be all that is required for diagnosis, although in later stages only an inflammatory response to dead larvae may be seen. Fine needle aspiration cytology (FNAC) can also aid in diagnosis as reported in some series, but is subject to sampling error and may be difficult to confirm the diagnosis. Studies have demonstrated that parts of the parasite have been identified in 45% to 100% of the aspirates, particularly when the aspirated material showed a speck of pearly white content that was confirmed to be the larva in acute and chronic inflammatory background by microscopic examination, which may include some eosinophils and palisading histiocytes. The larva has been identified by its lightly stained outer wavy membrane and multiple tiny ovoid nuclei in the fibrillary stroma beneath.

In our case the aspirate revealed only RBCs. Histopathological examination makes up a diagnosis of cysticercosis by the detection of a cystic space containing the cysticercus cellulosae. The scolex has four suckers and a double crown of rostellar hooklets. A duct-like invaginated segment, lined by a homogeneous anhistic membrane, composes the caudal end. The eosinophilic membrane that lines the capsule is double-layered, consisting of an outer acellular and an inner sparsely cellular layer. After a period within three and five years the larva dies and the cyst undergoes calcification.

Cysticerci may remain alive for many years. The first stage of involution of cysticerci is the colloidal stage, in which the transparent vesicular fluid is replaced by a viscous, turbid fluid. Additionally the scolex shows signs of hyaline degeneration. Thereafter, the cyst wall thickens and the scolex is transformed into coarse mineralized granules. This stage, in which the cysticercus is no longer viable, is termed the granular stage, which anyway does not seem to modify its clinical outcome. Finally, a granulomatous reaction develops that is characterized by histiocytes, epithelioid cells, and foreign body giant cells, leading to fibrosis of the supporting stroma and calcification of the parasitic debris. This pattern of reaction is the same, regardless of the organ involved, and this evolution indicates the age of the infestation.

Laboratory findings in patients with cysticercosis reveal eosinophilia, raised immunoglobulin E (IgE), and most importantly, a positive enzyme linked immunosorbent assay (ELISA) test against cysticercus cellulosae. Anti cysticercus cellulosae antibodies are important in the immunodiagnosis of the disease. This procedure may be performed in serum or cerebrospinal fluid, the latter is considered a diagnostic test for neurocysticercosis.

Drugs as albendazole and praziquantel are potent antihelminthics used in the treatment of cysticercosis, replacing niclosamide, which was the drug of choice for the treatment of the disease for a long time. Drugs should be used especially in cases where surgical treatment is risky or not possible, as in neurocysticercosis.

CONCLUSION

In summary, we have showed the clinical and histopathological findings in a man with oral cysticercosis, emphasizing the need to consider cysticercosis along with other causes of cystic lesions, particularly in areas with a
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high incidence of this condition.

References

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