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Oral Cysticercosis: review article

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Abstract

Cysticercosis is caused by the tapeworm, Taenia solium. It is a common disease of developing world. Larvae of this parasite can be found in the muscles of pig, which is the intermediate host of this disease. Humans are generally the definitive host and infection occurs with ingestion of undercooked pork containing cysticerci, but they can also serve as the intermediate host if eggs of T. solium are ingested accidently by them. Larvae can lodge in any part of the body including subcutaneous tissue, brain, muscle, liver, lungs, heart or eye. They are generally asymptomatic, except when ocular region or brain is involved. Oral cysticercosis is a rare occurrence, which presents as a firm nodule clinically. Radiological tests and immunological investigations can help in making diagnosis of this disease, but for definitive diagnosis histopathological examination is required. Surgical excision of the lesion is generally done for treatment and prognosis is good for these cases. Improvement of sanitary conditions is very important for stopping propagation of this disease.

Keywords: Cysticercosis, Taenia solium

Introduction

Cysticersosis is caused by the tapeworm Taenia solium, which is prevalent in Latin America, South East Asia, India, South Africa and East Europe. Although cysticercosis can affect many different organs in the body, yetbrain and eye involvement are symptomatic. Neurocysticercosis is the most dangerous type of all these afflictions. It can present with fever, headache, seizure, obstructive hydrocephalus, meningitis, and mental disorders. According to WHO estimates, neurocysticercosis causes around 50,000 deaths per year.[1,2,3] Cysticercosis is linked to insanitary conditions, and uncontrolled pig breeding. Human being is the definitive host of this parasite. Adult worm lives in the small intestine of man, who gets this disease by ingestion of undercooked pork containing cysticercus larvae.[4]

Cysticercus contains inverted scolex, which becomes everted when the larva gets into small intestine, with the help of bile juice and digestive enzymes of the host. Scolex attaches to the small intestine of the hosts and with its help cysticercus larvae gets its nutrition and grows in size, thus becoming an adult worm in 10 to 12 weeks time inside small intestine of human host. Adult worm can survive for years inside human host and reproduces by producing embryonated eggs known as oncospheres. Each T. solium is composed of head portion, known as scolex and caudal part consisting of proglottids. Each gravid proglottid can have as many as forty thousand to sixty thousand eggs.[5,6,7] These gravid proglottids are shed in the intestine of definitive host in the groups of four to five, and move out of the body along with faeces. They can survive in environment for two months.[8]

Pigs serve as intermediate host to T. solium. Human faeces containing T. solium eggs can contaminate food and water, which is consumed by the pig. By the action of stomach enzymes, the embryonated membranes of eggs are removed, and the eggs hatch into motile oncospheres in the intestine of pigs. The oncosheres attach to the intestinal wall and burrow inside intestinal mucosa before entering blood vessels and lymphatics. Along these blood and lymphatic channels, these oncospheres reach distal organs of the body including liver, muscle tissue and brain. Once inside these tissues, they form cystic structures called cysticerci. The central part of a cysticercus is filled with fluid, so it is also known as a "Bladder worm". A cysticercus contains in vaginated protoscolex and is 1 to 2 cm in diameter.[9,10,11]

Human beings are generally infected by consumption of contaminated pork. But, less commonly, they can also become a secondary host of this disease by ingesting eggs. This can occur by ingestion of faecally contaminated water or food for example vegetables, which are infested with T. solium eggs. This can also occur by mechanism of self contamination, in which reflux of scolices containing eggs occurs from intestine to stomach, and in stomach coating of eggs are dissolved by action of gastric juices and upon reaching intestine again, eggs develop into oncospheres, which penetrate into intestinal wall. They develop into cysticerci upon reaching the target organs by similar mechanism as discussed in pigs. In humans the cysticerci get deposited preferentially into muscles and brain tissue, trunk, subcutaneous tissue, liver and lungs.[12,13]

Cysticercosis affects oral cavity rarely in humans, possible because of higher muscular activity and

increased metabolic rate of oral tissues, which act as deterrent for lodgement of larvae.[14] Out of the tissues of oral cavity, tongue is most commonly involved, followed by labial and buccal mucosa. According to Hosur et al. out of 69 cases of oral cysticercosis reported, 32 cases involved tongue.[15,16] Yet, the frequency of involvement of tongue is much as compared to other organs including other striated muscles in humans. It is interesting to note that tongue muscles are commonly involved by this parasite in pigs.

Oral cysticercosis lesions have a high intraluminal pressure and they present as firm nodules.[17] Many times it resembles a mucocele, or it can mimic mesenchymal benign tumour like lipoma, fibroma, hemangioma, granulose cell tumour or a minor salivary gland tumour on clinical examination. Careful clinical examination can help in differentiating this disease from other similarly presenting lesions. The lesion in cysticercosis is a firm nodule, while lipoma is soft and fluctuant and divided by fibrous tissue into lobules. A flat or raised presentation is common for hemangioma and is usually bluish red or purple in colour. Lymphangioma is generally present on body since birth. Sometimes, a granular cell tumour which is a slow growing, painless, smooth mucous swelling, can present in a very similar way as a cysticercosis lesion.[18-22] In these cases histopathology is needed to confirm the diagnosis. Microscopic examination of granular cell tumours show polygonal cells with eccentrically placed nucleus and abundant eosinophillic, pale granular cytoplasm.[23]

The clinical diagnosis of oral cysticercosis can be confirmed by radiological tests, serological testing or by histopathology. X rays can help make a diagnosis; these images may show elongated calcified cysticerci in soft tissues. Many times, they appear as distinct opacities, which are elliptical in shape and appear similar to grains of rice. Computerized tomography (CT) and magnetic resonance imaging (MRI) are better diagnostic tools than conventional imaging. In fact MRI is now considered as the best method for detecting degenerating and viable cysticerci, especially in cases of neurocysticercosis. Lately high resolution ultrasonography has also shown promise in diagnosing cysticercosis in muscle tissue.[24,25,26] Immunological diagnosis can be made by analysing serum, saliva or cerebrospinal fluid with the help of enzyme linked immunosorbant assay (ELISA), or enzyme linked immune electro transfer blot. While enzyme linked immune electro transfer blot has very good sensitivity and specificity in detecting multiple intracranial lesions, its usefulness is limited in detecting a single or calcified lesion.[27-29] CT and MRI tests are considered better than serological tests.[30] Fine needle aspiration cytology is also sometimes used in making diagnosis of this disease. Its sensitivity is between 45 to 100%.[31]

The definitive method of diagnosis is histopathological examination (HPE). The suspected cystic nodule is resected and enucleation of cyst is done, and is sent for HPE. On cut section, it contains water like clear fluid.The inner surface of the enucleated cyst is generally yellowish to pale in colour and contains scolex of the larva which is attached as an evagination from the cystic wall. The scolex has 4 suckers and two crowns of rostellar hooklets. Microscpic exam shows a well defined capsule. A double eosinophillic membrane with an outer acellular layer and an inner cellular layer outlines the capsule. Invaginations and inflammatory cell infiltrates are seen in the host capsule. After about 3 years, larva dies and it takes about 5 years for calcification to appear. Cytological examination shows clear fluid with white flakes, mixed inflammatory response with eosinophills and plasma cells, palisading histiocytes with calcific structures on microscopy.[1,32]

Treatment of oral cysticerciosis is simple excision and complete removal of the lesion. These lesions are easy to excise without any postoperative complications. In case there are multiple lesions, it is not required to remove all lesions, if the definitive diagnosis of cysticercosis is established.[17] Oral cysticerciosis patients should undergo investigations to rule out presence of lesions in other parts of body too. In symptomatic patients with disseminated disease, especially in unresectable areas, antiheliminthics such as praziquantel and albendazole should be given.[33]

Improvement of hygiene in a community and proper sanitation are two very important strategies to control cysticerciasis. This disease can be potentially eradicated from the world because of absence of any other reservoirs except humans and pigs. It is very important to propagate high level of sanitation, especially proper human faecal disposal around pig farms so as to break mode of transmission from humans to pigs. Human cysticercosis can be prevented by encouraging hand washing before handling food, and by avoiding eating undercooked pork. Concurrent chemotherapy programs for humans and pigs have been tried with some limited success to control this disease. Lately a few genetically engineered vaccines are being tested on pigs in hope to find a way to eliminate this disease.[34-38]

Conclusion

Although cysticercosis affects oral cavity rarely, it should still be considered as a differential diagnosis while treating solitary intraoral swellings, especially in areas where this disease is endemic. CT scan, MRI, high resolution ultrasonography and immunological investigations are some of the ways of diagnosing this disease, but histopathological examination remains the gold standard for diagnosis. The role of improving sanitation and effective faecal disposal cannot be overemphasized for prevention of this disease.

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