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REVIEW

Oral Cysticercosis - Review of Literature

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ABSTRACT:

By oral infestation in man, their definite host, the large taenias (T. solium and T. saginata) of the Taeniidae family, Cyclophyllidea order, Cestoidea class, may cause two different types of disease: teniasis and cysticercosis. Although infestation in these parasitic diseases always takes place by ingestion, the manifestations or localizations within the oral region are extremely rare and, therefore, are hardly ever mentioned in studies of oral pathology.

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INTRODUCTION

The taenia solium parasite or tapeworm is found in the small intestine of man, the definitive host. The terminal segments of the parasite (proglottids) contain eggs, and these are excreted with the faeces. In areas where unhygienic conditions prevail, the faeces are dispersed on the surface of the ground and may be ingested by the pig, the intermediate host. The gastro-intestinal secretions of the pig dissolve the eggs and liberate the embryos or enchospheres, These embryos then penetrate the intestinal mucosa and gain access to either the vascular, or lymphatic circulation and are thus distributed to various tissues and organs, particularly muscle. Here they develop into the larval form known as cysticercus cellulosae. The eating of undercooked and contaminated pork by man results in the larvae reaching the intestine, where they develop into the adult stage of the taenia solium. This is the normal life cycle *of* the tapeworm.

In rare circumstances, and mainly as a result of poor hygienic conditions, man may ingest either the eggs or proglottids of the parasite. The larval stage, the cysticercus cellulosae, will then develop in the human. This is likely to happen when carriers of the taenia infect themselves by transferring eggs from the faeces to the mouth through their fingers. It 'has also been suggested that both the eggs and the gravid proglottids can gain access to either the duodenum or stomach through regurgitation. Subsequent penetration of the mucosa by the embryos results in cysticerci developing in the human organs and tissues. Man thus becomes the intermediate host.[1,2]

The brain, the skeletal muscles and the subcutaneous tissues arc the most common sites to be affected by the cysticercus cellulosae. Involvement

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of the central nervous system may result in epilepsy, intracranial hypertension and mental disorders. Implantation in muscle or subcutaneous tissues is usually symptomless. Superficial lesions present as subcutaneous nodules and are readily detectable. Deeply situated cysticerci are not evident unless presenting clinical symptoms". When the implanted cysticercus cellulosae dies the residual lesion undergoes calcification and is then evident radiographically.

Although there are no reliable statistics on the frequency of cysticercus in different areas in the world, it would appear that Latin America, India, Eastern Europe and Southern Africa are high incidence areas.[3,4,5]

Oral cysticercus is a rare condition and in the last 30 years only 23 cases have been published. Most of these cases were published in the Latin American and Indian literature where this condition is more frequent. The 8 oral cases mentioned by DIXON & LIPSCOMOO, although published in the English literature, were originally infected in India.

CLINICAL PRESENTATION

Variability is one of the most characteristic features of cysticercosis. Three sets of variables determine the clinical presentation, course and prognosis: (1) location, growth, size and number of cysts; (2) viability of lesions, stage of cyst degeneration and presence of calcifications; and (3) type and degree of host response (Nash and Neva, 1984).[6] Commonly, patients present with multiple lesions such as cysts of different sizes associated with varying degrees of host response, nodular enhancing lesions and residual calcifications are sometimes accompanied by perilesional edema. Besides detection of the characteristic scolex in cysts, it is the combination of these findings that is most helpful in suggesting the diagnosis since there are few processes that are present with multiple cystic lesions and/or characteristic punctate calcifications. Both computer tomography (CT) and magnetic resonance imaging (MRI) have played important and essential roles in recent understanding of cysticercosis. These radiological techniques have allowed easy, non-invasive ways to visualize lesions, establish the diagnosis in most cases, determine the types of treatments likely to be worthwhile and gauge the effectiveness of therapy (Martinez et al., 1995; Dumas et al., 1997).[7,8] MRI is the radiological technique that best reflects the pathological state of the cyst and host response (Suss et al., 1986; Teitelbaum et al., 1989; Martinez et al., 1995; Dumas et al., 1997).[9,10,7,8] Non-calcified granulomas in the absence of enhancement or the uncommon presence of perilesional edema are not visualized by MRI. The most common radiological finding in cysticercosis is punctate-calcified granuloma that can be present in about 14-20% (Garcia-Noval et al., 1996, 2001; Sanchez et al., 1999; Cruz et al., 1999)[11,12,13] of asymptomatic and/or serologically negative endemic populations. Calcified lesions are best visualized using non-enhanced CT imaging, but CT examinations fail to visualize a substantial number of cystic lesions and lesser degrees of enhancement and perilesional edema compared to MRI (Lotz et al., 1988; Teitelbaum et al., 1989).[14,10] The usual organs showing parasitic infestation are the subcutaneous tissues, skeletal muscles and brain. The oral cavity is a distinctly unusual site for these cysts and till now only 25 cases of oral cysticercosis have been reported. Many times, the most severe lesions dictate the type of treatment that should be offered.

TREATMENT

Because of the variability in presentation, the consideration for treatment should be individualized. Inherent in any clinical decision is reasonable proof that treatment will be of clinical benefit. This implies that there is adequate knowledge of the natural history of cysticercosis and its various types of presentations. Unfortunately, the natural history of most presentations of cysticercosis is at best incompletely described and therefore prognosis is difficult to judge with certainty. In addition, most treatment trials have not been optimally designed or executed so that, for the most part, treatment regimens are based on nonrandomized trials and investigators' observations. Nevertheless, although imperfect, there is a body of knowledge on which to base treatment decisions. There are four treatment modalities that can be offered to patients: (1) larvicidal agents to kill the cystic larvae and/or tapeworm; (2) corticosteroids or other immunosuppressive agents to decrease or prevent inflammation; (3) surgical-based therapies including emergent measures to decrease the mass effect of cysts with or without accompanying inflammation, and (4) general supportive measures in impaired individuals or symptomatic treatments. Albendazole and praziquantel are the larvicidal drugs used in the

treatment of cysticercosis in human. A summary of treatment series has been reviewed by Garcia et al. (2002).[15] Cure rates range from 60 to 85% in the usual dosing with most series showing albendazole yielding slightly higher cure rates. Praziquantel at the usual dosing of 50-75 mg/kg per day in three divided doses was the first drug available and has few side effects. It was initially administered for 30 days but in later studies employed for 14 days. Because of the high cost associated with a long course of treatment, 1-day therapy using 75 mg/kg every 2 h for three doses (Corona et al., 1996)[16] has been advocated. Praziquantel blood levels are decreased with common anti-seizure medications such as dilantin and carbamazine (Bittencourt et al., 1992)[17] and corticosteroids (Vazquez et al., 1987).[18] Albendazole has largely supplanted praziquantel because of slightly greater cure rates, decreased cost and increased availability. Albendazole is converted to its active metabolite, albendazole sulphoxide, in the liver. It is usually given at 15 mg/kg per day with a maximum of 400 mg/bid (higher doses have been given) for 7-30 days with repeated dosing as clinically warranted. Absorption of albendazole is increased with fatty foods (Lange et al., 1988).[19] Drug interactions are well documented with praziquantel but are not well studied with albendazole even though similar compounds demonstrate significant interactions with anti-seizure medications. Few side effects directly related to the drugs themselves have been documented in the treatment of cysticercosis. It is unusual to experience serious side effects with praziquantel; but serious side effects, including agranulocytosis, liver function abnormalities and balding have been documented with long-term albendazole administration in the treatment of hydatid disease. Corticosteroids are commonly used to suppress and/or prevent ongoing or treatment-induced inflammation that usually occurs 2 - 5 days after initiation of therapy. Its usage has not been standardized. Although some wait until symptoms develop, others begin corticosteroids with, before or just after, administration of larvicidal drugs. There is a general consensus that corticosteroids should be used prophylactically when patients have numerous, large or critically located lesions. Corticosteroids decrease blood levels of praziquantel and theoretically could result in decreased efficacy (Vazquez et al., 1987).[18] Elimination of albendazole sulphoxide is decreased by corticosteroids but it is unknown if there is increased efficacy due to this (Takayanagui et al., 1997).[20] The duration of corticosteroid treatment is 10 - 16 mg per day of dexamethasone in divided doses and taper the dose following therapy over 1- 3 months depending on the MRI findings. The need for surgically based therapies has decreased dramatically over the past two decades. In addition, and have removed easily approachable cysts that impinged on critical structures before larvicidal treatment to prevent serious complications following treatment and to decrease the duration of corticosteroid use. Decompression of life-threatening lesions is another indication.

Discussion

Cysticercus cellulosae is a rare condition in man and in the majority of cases the larvae are implanted in the central nervous system, in muscle, and in subcutaneous tissues. Other organs may be affected, but relatively infrequently. Its occurrence within the oral cavity is rare and is mainly distributed in muscle. The tongue is most commonly involved,[21] followed by the upper and lower lip, the cheeks and in one reported case, the alveolar mucosa. The age of discovery varies from 5 to 70 years and there is no sex predilection. In most cases the infestation is massive and many foci may be found in the same individual. In a series of 450 cases, mentioned by DIXON & LIPSCOMBG,[22] 8 involved the tongue, and in all these cases cysticerci were also present in the brain. Some of these 8 cases showed subcuticular nodules. Similar findings of multiple foci have been mentioned by other authors.[22]

The symptomatology depends on the organ involved. In cerebral cysticercosis the symptoms are mainly related to the anatomical location of the cysticerci, since the lesions can produce pressure on the different brain centres. They act as space occupying lesions, and many have been mistakenly treated over long periods of time as primary brain lesions mimicking cerebral neoplasm.[22] The deeply situated muscular lesions are usually symptomless and, at the present time, are impossible to detect unless they become calcified, when they can be demonstrated radiographically. Both the oral and subcutaneous forms of cysticercosis are relatively easily detected clinically because of their superficial location.

The high incidence of multiple foci is of significance, and every case of oral and subcutaneous cysticercosis should be regarded as part of a generalized involvement and should be thoroughly investigated to determine whether or not other organs are involved.

The differential diagnosis of oral cysticercosis depends on the location of the lesion. Nodules on the lips and cheeks may be considered as fibroma, lipoma, mucocele, pyogenic granuloma or pleomorphic adenoma. Nodules on the tongue may be considered as fibroma, pyogenic granuloma, granular cell myoblastoma or rhabdomyoma. Laboratory tests with cysticercus cellulosae antigen give a relatively high degree of reliability.[23] This method should be used in all suspicious cases, but a definitive diagnosis can only be made on histological evidence. Although the incidence of oral cysticercus cellulosae is rare, it should always be considered as a possibility when nodular formations appear in the mouth, and especially when the affected individual Jives or came from a geographical area of high incidence. The treatment of oral cysticercus cellulosae is surgical excision and the biopsy specimen will allow confirmation of the tentative diagnosis. The treatment in other locations is dependent on the symptomatology and the accessibility of the lesion to surgical intervention.

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