Tubercular Infection of Hydatid Cyst

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Abstract

Tubercular infection of hydatid cyst of the chest wall in an immunocompetent individual is rare. Immune modulation for symbiosis between host cells and the parasite - Echinococcus granulosus favors tubercular infection. In this case report, we describe a case of both these chronic diseases coexisting together, to present as chest wall mass.

Keywords: Helminthiasis, hydatid cyst, hydatidosis, tuberculin test, tuberculosis

Introduction

Both tuberculosis and hydatidosis are common in India, but tubercular infection of a hydatid cyst in an immunocompetent individual is rare. Hydatidosis is seen in people who ingest dog feces containing eggs of the dog tapeworm - Echinococcus granulosus, either while handling dogs or consuming water and/or vegetables contaminated with viable eggs. The eggs can, not only survive in adverse conditions for long periods of time but also are capable of entering a secondary host immediately. In the intestines, the eggs hatch into oncospheres that penetrate the mucosa and enter the portal circulation or lymph vessels. The first capillary filter before reaching the heart is in the liver, and this makes it the most common site of hydatid cyst followed by the second capillary filter of the lungs making it the second most common site. Oncospheres entering the lymph channels bypass the capillary filter of liver and thus can be deposited anywhere. Oncospheres entering the systemic circulation after bypassing the liver and lung filters are uncommonly trapped by other body parts resulting in a small percentage of all hydatid cysts. Most of the infections are acquired in childhood and present in adulthood as the rate of growth of hydatid is very slow and its growth may be halted temporarily.

Case Report

A 44-year-old male shepherd presented with a painless swelling on the left side of the chest wall for the last 10–12 years. It was slow growing, but for the past few months, its growth had increased significantly. There was no history of fever, weight loss, night sweats, or swelling(s) over other parts of the body. There was a history of contact with sheep and dogs. The patient gave no history of deworming the dogs. There was no history of tuberculosis or contact with someone afflicted with tuberculosis. On examination, his abnormal finding was an oblong, subcutaneous swelling of 14 cm × 10 cm in size. It was freely mobile in all directions, without any signs of inflammation. The swelling was not translucent. His abnormal biochemical finding was an erythrocyte sedimentation rate of 48 mm in 1st h by Wintrobe’s method. His abnormal radiological finding was a lobulated heterogeneous peripheral enhancing soft tissue mass in the left chest wall in the subcutaneous plane arising from serratus anterior with axillary lymphadenopathy on magnetic resonance imaging [Figure 1]. Fine-needle aspiration cytology revealed chronic inflammatory cells, and a tru-cut biopsy imprint smear revealed lymphocytes and acid-fast bacilli positivity on Ziehl–Neelsen staining. His tuberculin test was negative with 5 tuberculin units. Based on this, he was started on antitubercular therapy (ATT).

He came back after 3 weeks with serosanguinous discharge from the tru-cut biopsy site. The swelling was 10 cm × 6 cm. Swelling was explored with a wide excision of the mass. A 10 cm × 6 cm mass was seen overlying the 9th to 11th ribs on the left chest wall with multiple discharging sinuses. Multiple small cystic masses typical of hydatid daughter cysts were also
seen within the mass [Figure 2]. Histopathology confirmed the diagnosis. Albenzaole with fatty foods was added for 4 weeks, and ATT was continued for 6 months.

Discussion

Based on the natural history of the disease, we believe the patient acquired chest wall hydatidosis during childhood which subsequently got infected by *Mycobacterium tuberculosis*. The rate of growth of the cyst is very slow and may remain asymptomatic for decades depending on the site. Symptoms are usually because of the mass *per se* or because of the pressure of this mass if it is in a confined cavity. Bacteria may enter the cyst and convert it into a pyogenic abscess. Rupture of the cyst can cause severe anaphylactoid reaction resulting in shock, hypotension, syncope, and rarely even death. Only a few cases of tuberculosis and echinococcal infection are reported. Most of these are pulmonary tuberculosis with either pulmonary echinococcal disease or hydatidosis of the axillary lymph nodes.[1-4] We believe that this is the first case report of chest wall hydatid cyst infected with tuberculosis as even after a detailed search of the literature, using PubMed and Google scholar, we found no case reports of such an unusual occurrence.

The oncosphere of *Echinococcus* results in the formation of a cyst. At an early stage, the complement system is activated, resulting in a complement-mediated inflammatory response, which leads to the death of the host cells surrounding the oncosphere. With the development of laminated and germinal layers, the exposure of the former to the host cells decreases the strong complement-mediated immune response.[5] A symbiotic relationship is developed between the host and the parasite. There is a generation of T-suppressor cells and impaired macrophage and lymphoproliferative response. This changing pattern of the immune system during the development of hydatid has been demonstrated by the changing positivity of the tuberculin test, indicating a change from Th1 to Th2 cells.[6] With the suppression of Th1 cells, the ability of the host to respond to pathogens is impaired.[7] This predisposes the individual to develop tuberculosis by suppressing the tubercular-specific Th1 (interleukin [IL]-12 and interferon gamma) and type T helper (IL 23 and IL 17) responses which are closely linked to the cytotoxic T lymphocyte antigen-4 and programmed cell death protein 1, as both of these proteins downregulate immune responses.[8]

Echinococcal infection predisposes to the development of tuberculosis.[9] We do not know the reason for rarity of concomitant tuberculosis and echinococcal disease. It is either underdiagnosed or underreported. Echinococcal infections are more frequent in the sheep- and goat-rearing areas where perhaps diagnostic facilities are not available or the patients are asymptomatic, and the disease goes unrecognized. Even in the presence of coinfection, only one of these diseases may be treated, and the other may not look for at all.

Conclusion

Our aim of reporting this combination of tuberculosis and hydatid disease is to create awareness of this coexistence and the need to evaluate histopathological specimens of hydatid cysts routinely for tuberculosis. Similarly, tubercular patients should also be evaluated for echinococcal infection if clinical history is suggestive. The presence of any helminthic infection may give a false tuberculin test including the common Mantoux test.

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Conflicts of interest
There are no conflicts of interest.

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