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Disseminated Abdominal Hydatidosis

Sir,

Human hydatid disease results from infection with larval form of Echinococcus granulosus. Rupture of Hydatid cyst into abdominal cavity, causing multifocal dissemination, is uncommon complication affecting 2-12% of patients. A case of disseminated abdominal hydatidosis is presented along with a brief review of literature pertaining to life cycle, epidemiology and management of echinococcus.

25-year-old female, sheep and dog rearing by occupation, presented to OPD with complaints of anorexia, fatigue, weight loss, abdominal lump and intermittent high-grade fever for 3 months. She had undergone abdominal laprotomy for retroperitoneal hydatid cyst 10 years back. Physical examination revealed a large mass arising from pelvis measuring (15 cm x 10 cm x 8 cm) rising up to the umbilicus, with a dull note on percussion over it. There was hepatomegaly (8 cm below costal margin). Laboratory examination revealed Hb-6.8 gm/dl, total leukocyte count-5600 cu/mm with 15% eosinophils. All other hemorrheological parameters were within normal limits. Computer tomogram of abdomen (Figs. 1, 2) showed multiple well-defined cystic areas with internal septations spreaded all over abdomen. In view of the investigations diagnosis of disseminated abdominal hydatidosis was made. The patient was prescribed Albendazole and was advised admission for further management. She refused admission and was lost to follow up.

Echinococcus is a cosmopolitan parasite, with highest prevalence in Mediterranean countries, North and East Africa, Australia and South America. It is widely prevalent in Indian subcontinent as well, and is frequently reported from states of Tamil Nadu, Andhra Pradesh, Gujarat, South Maharashtra and North Karnataka. Treatment options for cystic hydatid disease are surgery, drug therapy and percutaneous drainage. Disseminated echinococcus is an absolute indication for anthelminthic drug therapy. Albendazole (10mg/kg) is most commonly used drug, administered for at least 3-6 months, causes death of parasite by preventing glucose absorption through cell wall, but a favorable response is obtained only in 29% patients and in 48% there is no change in cyst dimensions. The main disadvantages of use of albendazole are its hepatotoxicity, teratogenecity and prolonged treatment duration. Surgery has the potential to remove the cyst and result in complete cure. Surgical procedures include cystectomy with removal of the germinal and laminated layers and preservation of pericyst. Operative mortality varies from 0.5-4%. Cyst fluid spillage can occur during surgery resulting in anaphylaxis and/or secondary echinococcus (2-25% of cases). Recently,

Fig. 1 : Axial non-contrast image of abdomen at the level of liver showing multiple cystic lesions in subhepatic space and retroperitoneum with classical appearance of multicystic hydatid cyst having daughter cysts.

Fig. 2 Coronal reformatted image of CT scan of abdomen showing multiple cystic lesions in abdomen and pelvis with right renal hydronephrosis caused by cysts in pelvis.
Pleural Effusion of a Dual Etiology

Sir,

The synchronous existence of lung cancer along with parenchymal pulmonary tuberculosis – active and quiescent has been often reported. The coexistence of lung cancer with a malignant and tuberculous pleural effusion on the other hand is rare with on such previously reported instance. We report one such interesting case.

A 61 year old non smoking male came to us with a history of cough, mucoid sputum, loss of appetite and a significant weight loss since 2 months. He had no fever, night sweats, dyspnoea or chest pains. Physical examination revealed decreased thoracic movement, a stony dull note, absent breath sounds and decreased tactile vocal fremitus and resonance on the right side consistent with a pleural effusion. Chest radiograph (PA view) showed a massive right pleural effusion with multiple poorly defined circular shadows in the opposite lung. Aspirated pleural fluid was hemorrhagic, an exudate (Protein: 4.7 gm/dL) and having a lymphocyte differential of 54 %. Pleural fluid Gram stain and ZN stain were negative. Cytological examination revealed cell clusters with atypical nuclei. Pleural fluid carcino-embryonic antigen was also elevated (103.7 iu/ml, normal < 3.4iu/ml). Sputum smear examination for AFB was negative for 3 consecutive samples. Sputum examination for malignancy was repeatedly positive for poorly differentiated carcinoma. Bronchoscopy revealed external compression of RUL bronchus and bronchus intermedius and bronchial lavage smears were positive for malignant cell and negative for AFB. USG abdomen showed grade II prostatomegaly but serum PSA was normal. A diagnosis of bronchogenic carcinoma with malignant pleural effusion was kept. The patient refused any further palliative treatment for the same.

At 6 weeks pleural fluid culture for AFB (on L.J media) was found to grow colonies of mycobacteria. Patient was readmitted for further evaluation. Sputum smears were sent for Z.N stain which was positive for AFB. Subsequent sputum culture also grew mycobacteria species.

Our diagnosis was revised to sputum positive pulmonary tuberculosis with bronchogenic carcinoma (Stage IV) with malignant and tuberculous pleural effusion. Patient was started on 4 drug anti-tuberculous treatment (HERZ). After clinical stabilization he was discharged from the hospital.

The coexistence of bronchogenic carcinoma with pulmonary tuberculosis has been explained by the following mechanisms: 1. The chance coincidence without any relation – more relevant in countries like India with a high prevalence of tuberculosis, 2. “Scar carcinomas” i.e. tuberculosis giving rise to epithelial metaplasia with subsequent progression to carcinoma, 3. Re-activation of tuberculosis due to release of local tumor peptides and antigens from the tumor cells upsetting the milieu of the silent granuloma and allowing the mycobacteria to proliferate and, 4. Increased susceptibility to exogenous infection by mycobacteria due to malnutrition and weight loss related to an advanced neoplastic disease.

Immunosenesence and a decline in cell mediated immunity associated with ageing, poor nutrition, chronic alcoholism, lung neoplasm may all have contributed to

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