Case Report

AMEBIC PERICARDITIS- A RARE DISEASE WITH A RARER PRESENTATION & MANAGEMENT

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ABSTRACT

Amebiasis is a relatively common disease in the tropical region of the world especially in the Indian subcontinent. Extra-intestinal dissemination happens through the portal route and hence the liver is the most common site for the same. Other sites can be also involved albeit less commonly. Pericardial involvement is mostly due to extension of the infection of the left lobe of liver due to anatomically proximity.

Keywords: Amebic Pericarditis, Amebic Liver Abscess, Rupture

INTRODUCTION

Amebiasis caused by *Entameba histolytica* can cause invasive disease affecting 40 to 50 million people worldwide annually. Amebic liver abscess is the most common extra intestinal manifestation of amebiasis. Ameba establishes hepatic infection by ascending the portal venous system. If not detected early and treated, it can erode and rupture into almost all of the adjacent organs. Depending on the site of rupture, the presentation can vary from mild to catastrophic. Rupture into pericardium is a well-known and well-documented complication of amebic liver abscess. We report a unique case of pericardial rupture of amebic liver abscess who presented with cardiac tamponade with no classical signs of liver abscess and who was managed by a pericardial sheath placement leading to drainage of both the pericardial and hepatic abscess solely through the pericardial route.

CASES

A 47 year old male came with complains of chest pain and breathlessness and generalized weakness since last 2 months. Breathlessness was insidious in onset and of NYHA CLASS II. Chest pain was more of a pleuritic type, aggravated on lying down and coughing and relieved in sitting position. The patient denied intake of alcohol. Examination revealed a malnourished man with tachycardia and a low volume in the pulse. His mean arterial pressure was 76 mm Hg. He was having raised JVP with feeble and muffled heart sounds. Chest roentenogenogram showed cardiomegaly and ECG was suggestive of low voltage complexes with poor R-wave progression. Routine blood work did not level any presence of leucocytosis or deranged liver functions. Serum TSH was also normal. HIV ELISA was also negative. Echocardiography was performed and it revealed massive pericardial effusion with diastolic collapse of the right ventricle. The pericardial fluid was tapped under guidance and sent for routine studies. Around 300 ml of thick muddy fluid could be drained out on that instance. Routine and microscopic studies of pericardial fluid were suggestive of pyogenic picture with Staphylococcus aureus grown on culture. It was sensitive to ceftriaxone and vancomycin. GeneXpert[™] for the tubercle bacillus was negative. In view of pyogenic nature of the fluid a search for the focus of dissemination was taken. Sonography of the abdomen revealed a liver abscess in the left lobe of liver in the superior surface around 5.1/3.5/1.4 cc and around 220 cc. Computed Tomography of thorax with abdominal cuts showed the extent of the abscess with breach in the capsule and subdiaphragmetic collection of size 3.5/1.6cm draining in to pericardium. Serology report IgG antibody for Entameba histolytica was positive with high titers (1.74; normal range less than 0.4). Pericardial aspirate did not show the presence of Entameba histolytica and Stool examination for trophozoites and cysts of the same were negative.

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Figure-1: The Arrow Shows the Connection Between the Subcapsular Liver Abscess and Pericardial Sac as Depicted in this CT Scan of the Thorax with Abdominal Cuts

Over the period of two weeks, the patient was tapped 3-4 times with around 700 ml of fluid being more tapped. He was started on intravenous metronidazole 1000 mg three times a day with oral diloxanidefuroate (AMICLINE) for intestinal carrier state. A repeat 2D Echo was done after two weeks revealed thin rim of pericardial effusion which could not be drained. Repeat sonography of the abdomen was done which showed collapse of the abscess. The patient being vitally stable is advised discharge. On follow-up after four weeks his echocardiography was done which didn't reveal any pericardial effusion. His repeat sonography of the abdomen also showed resolution of the abscess.

DISCUSSION

Amebiasis is a common parasitic infection in the tropics. Mostly asymptomatic, it can lead to an invasive disease, which if left untreated, can lead to multiple complications. Amebic liver abscess usually presents with chronic low grade fever, abdominal pain, and emaciation. If left untreated, it can be complicated by rupture into the thoracic structures like pleural or pericardial cavities and abdominal structures like stomach or peritoneal cavities. Amebic pericarditis is an infrequent complication of liver abscess and accounts for 4 percent of all extraintestinal amebiasis (Ganesan *et al.*, 1975).

Previous report has shown 501 cases of amebic liver abscess. There were 326 cases of rupture through the diaphragm with 5 cases (0.01%) rupturing into the pericardium and 1 case rupturing into both pleural and

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pericardial cavities (Ibarra-Perez, 1981). The second such study from China by *Meng et al.*, (1994) reported 503 cases of amebic liver abscess over 21 years. 22% of the cases in this series developed complications due to perforation. Perforation into pericardial cavity remained a very rare complication in both the studies. To the best of our knowledge, five cases of amebic abscess rupturing into pericardium have been reported from India (Gupta *et al.*, 1980; Suryanarayan *et al.*, 1974).

Amebic pericarditis, although rare, is the most dangerous complication of amebic liver abscess. In majority of the cases, this is a complication of the left lobe superior surface amebic liver abscess.

There are four types of amebic pericarditis (Kapoor, 1979).

1. *Suppurative or Purulent Type* - This can further be subdivided into acute or gradual onset. This term is a misnomer. It should ideally be termed as pericardial amebiasis with secondary infection. The latter follows pericardial tapping where the first aspirate is odourless chocolate coloured pus and the second aspiration shows change in colour either to yellowish white or green and it may also emit a foul smell. The importance of diagnosing such cases is to give them additional antibiotics for secondary infection.

2. Sympathetic or Non-Suppurative Type - An amebic liver abscess can produce sympathetic effusions in pleural, pericardial or peritoneal cavities depending on the site of the abscess. An abscess in the superior surface of the left lobe can produce sympathetic effusion in the pericardial cavity. These effusions are usually harmless. The importance of this clinical sign is that it serves as a warning of the proximity of the abscess to the pericardium and a possibility of a frank ruptures of the former into latter. Most of these effusions disappear with the management of the liver abscess.

3. Constrictive Amebic Pericarditis

4. Amebic Hydropneumopericardium

To establish the diagnosis of amebic pericarditis, one should demonstrate a communication between left lobe liver abscess and pericardial sac, a high serum titer of amebic hemagglutination antibodies, and the presence of *Entameba histolytica* trophozoites in the pericardium or pericardial aspirate and response to amebicidal therapy. In this case, anchovy sauce pus was aspirated from the pericardial cavity. A fistulous tract communicating between liver abscess and pericardial cavity was demonstrable by CT scan. Serology for amebiasis was positive, and patient showed dramatic response to the amebicidal drugs. The presence of all these factors confirms amebic aetiology of pericardial involvement in this case.

The patients of amebic pericarditis should be with amebicidal drugs such as metronidazole and dihydroemetine preferably in intensive care unit. Percutaneous needle aspiration of the liver abscess, followed by insertion of a percutaneous drain into the pericardial cavity for continuous drainage, should be done as soon as possible (Rajput *et al.*, 2003). Our case is unique where abscess was drained solely by pericardial route and did not require any separate drain for the liver abscess. Pericardiocentesis is useful both as a therapeutic, and as diagnostic tool. With this approach, most of the patients improve. Despite the best possible management, rarely few patients develop signs of constriction over next few weeks. There is evidence in literature that this stage may resolve with conservative management.

In spite of the patients being diagnosed and treated in time and pericardial aspiration also being undertaken, some patients still die for no obvious reason. It is postulated that ultra-microscopic changes in the myocardium results in sudden death from cardiac arrhythmia. In the case reported by *researcher*, varieties of tachy and bradyrhythmias were recorded in the intensive care unit before the patient died of ventricular fibrillation at a time when he seemed otherwise to be progressing well (Kapoor, 1979).

Our aim of reporting the case is due to the rarity of its occurrence. Also our patient did not have any symptoms related to liver abscess and presented solely with the symptomatology due to pericardial effusion. Recently a decline is noted in reporting of amebic pericarditis cases and this is probably due to early diagnosis and treatment of amebic liver abscess which prevents the development of this rare complication.

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