

Atrial Flutter and Pericarditis – A Rare Complication of Right Lobe Amoebic Liver Abscess

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SUMMARY

A 38 year old gentleman presented with fever and right hypochondrial pain. On further evaluation he was detected to have an amoebic liver abscess (ALA) in the right lobe of the liver. The abscess yielded anchovy sauce pus on percutaneous drainage. Following the percutaneous drainage the patient developed tachycardia. Electrocardiogram revealed atrial flutter with rapid ventricular rate and ST elevation in all leads suggestive of pericarditis. The atrial flutter was reverted to sinus rhythm by cardioversion. The patient then had an uncomplicated convalescence. Amoebic pericarditis, though rare, is a serious complication of amoebic liver abscess. Pericardial complications are usually seen with left lobe liver abscess due to its proximity. Both pericarditis and cardiac arrhythmias due to amoebic liver abscess especially from right lobe are very rare.

KEY WORDS:

Amoebic pericarditis, sympathetic pericardial effusion, E histolytica.

INTRODUCTION

Liver abscess is the most frequent extra intestinal complication due to *Entamoeba histolytica* infection. A rare and devastating complication of amoebic liver abscess (ALA) is its rupture into the pericardial cavity. Most of the pericardial complications due to the disease originate from the left lobe of the liver, which is in direct anatomical relationship with the heart and its membranes. We report a case of right lobe ALA causing pericarditis and atrial flutter and its successful management.

CASE REPORT

A 38 year old previously healthy gentleman presented to us with history of high grade fever and right hypochondrial pain of ten days duration. General physical examination revealed icterus, pulse rate -110/min, and blood pressure -110/70mm Hg. Abdomen examination revealed tender hepatomegaly. Investigations showed neutrophilic leucocytosis (22.3×10^3 ; 95% neutrophils), direct hyperbilirubinemia (Total 4.5 mg/dl, direct-3.6 mg/dl) and elevated liver enzymes (serum glutamic-oxaloacetic transaminase (SGOT)/ serum glutamic pyruvic transaminase (SGPT) -110/108 U/L; alkaline phosphatase -203U/L). Serum electrolyte panel and lipid profile were within normal limits. His HbA1C was 5.87%. Chest X ray showed minimal blunting of right costo-phrenic angle. Viral

markers for Hepatitis B, Hepatitis C and Human Immunodeficiency Virus were negative. Enzyme Linked Immunosorbant Assay for *Entamoeba histolytica* (both IgM and IgG) was strongly positive. Stool examination for *Entamoeba histolytica* cysts was negative. Electrocardiogram (ECG) showed sinus tachycardia without any evidence of ischemia or infarction. Contrast enhanced Computerised Tomogram of abdomen showed a hypodense lesion of size 8.7×9.4×10.3 cm with peripheral rim enhancement in the right lobe of liver (Figure 1) consistent with amoebic liver abscess. In view of the impending rupture he underwent ultrasound guided percutaneous drainage of the abscess, which drained 'anchovy -sauce' colour pus. Shortly after the percutaneous drainage the patient developed sudden tachycardia (pulse rate 180/min). Complication during the percutaneous drainage was suspected and a review ultrasonogram showed minimal peri-hepatic fluid and no evidence of pleural rupture. The patient was started on intravenous Meropenam and Metronidazole; Serial haemoglobin level monitoring did not show any drop but the tachycardia was persisting. After a 12 hour observation, it was decided to perform an exploratory laparotomy. The laparotomy did not show any evidence of peritoneal contamination, local collection or bleed. The abscess was evacuated and a drain tube kept in the cavity. Post-operatively also the pulse rate remained at about 180- 200/min and he started having hypotension. At this stage extra abdominal reason for tachycardia was suspected and a cardiologist opinion was sought. ECG at that time showed atrial flutter with rapid ventricular rate (Figure 2). Echocardiogram showed the presence of moderate pericardial effusion with no regional wall motion abnormality. As the patient was hemodynamically unstable, he underwent synchronised cardioversion and the cardiac rhythm was successfully reverted back to sinus mode. ECG taken on the next day was in sinus rhythm and ST elevation in all leads except aVR suggestive of pericarditis (Figure 2). Troponin "T" was mildly positive and there was no typical rise and fall of Creatine Phosphokinase – MB. The analysis of the pericardial fluid revealed a non purulent exudate (Lactate Dehydrogenase - 356U/dl, Protein - 5.9 mg/dl, Glucose - 68mg/dl, Adenosine Deaminase <5 U/L, cell count of 48 cells and predominantly lymphocytes) and was sterile on gram staining and culture. Subsequently the patient had a smooth convalescence and was discharged on post-operative day 12. The follow up ECG and echocardiograms done at discharge showed normal rate and rhythm and resolution of the pericardial effusion respectively.

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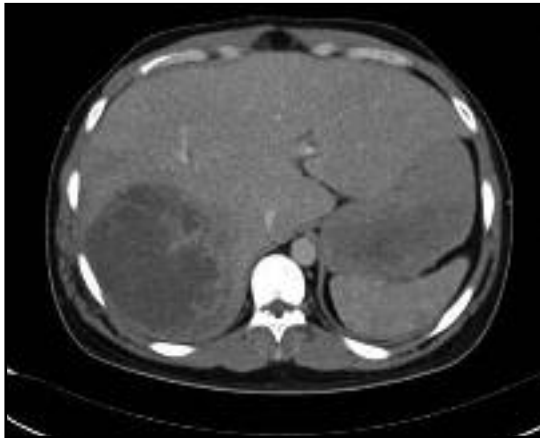


Fig. 1: Computerised tomograph of abdomen showing right lobe liver abscess.

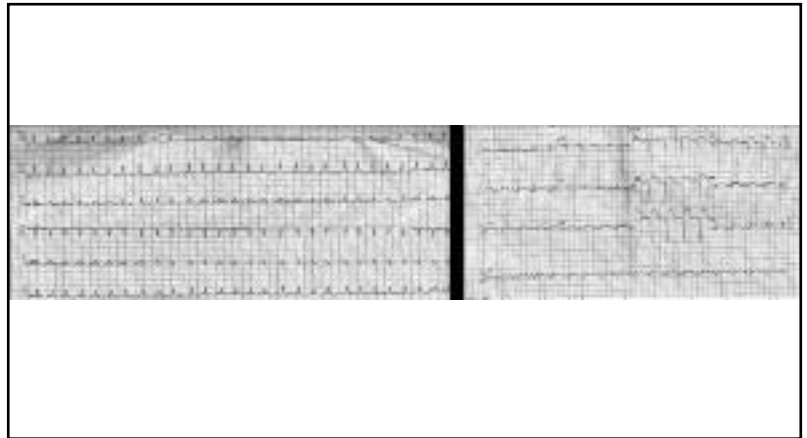


Fig. 2: ECGs showing atrial flutter with rapid ventricular rate and ST elevation in all leads.

DISCUSSION

Pericardial complications are not uncommon in patients with left lobe ALA but there are only nine reported cases of pericardial complications due to right lobe ALA¹. Amoebic pericarditis has a fatality rate ranging from 29.6% - 67% in earlier days with cardiac tamponade being the commonest cause of death^{2,3}. Our patient had a right lobe ALA developed pericarditis, atrial flutter and non suppurative pericardial effusion without any direct anatomical relationship with the pericardium, a unique combination that has not been reported till now.

Lamont and Pooler have described three stages in amoebic pericarditis⁴. First stage is that of sympathetic pericardial effusion, which is due inflammation caused by an abscess in the superior surface of the left lobe. This stage might be a warning sign of the impending rupture into the pericardial space. At this stage minimal pericardial effusion might be demonstrated and ECG may show evidence of pericarditis. The pericardial aspirate is usually exudative and will resolve once the ALA is managed successfully. Our patient was also identified at this stage, with the difference that our patient had the location of abscess in the right lobe of liver. He developed atrial flutter with rapid ventricular rate due to pericarditis which was not thought of by the treating surgeon hence he underwent a laparotomy. Second stage is suppurative pericardial effusion due to rupture of left lobe abscess into the pericardial cavity and would have the characteristic anchovy sauce pus. The third stage is that of constrictive pericarditis developing over a period of weeks to months. This is the rarest stage but has a high mortality⁴.

The clinical findings of pericardial amoebiasis are fever, chills, right hypochondriac pain, dysnea, jugular venous distension and distant heart sounds. Circulatory shock may be present in cases with cardiac tamponade. The diagnosis is by the echocardiographic evidence of pericardial effusion and aspiration of anchovy sauce pus from pericardial cavity along with the presence of an abscess in the liver and a positive serology for to *E.hystolitica*. *E.hystolitica* is only rarely demonstrated in the pericardial fluid⁵.

Treatment of amoebic pericardial effusion is drainage of pericardial fluid and pus from the liver abscess in cases seen at the stage of suppurative pericardial effusion. Sympathetic pericardial effusion usually don't need any specific treatment unless symptomatic. In patients with cardiac tamponade, emergency pericardiectomy is the treatment of choice. Pericardiectomy may be needed in the constrictive pericarditis stage.

Our patient had atrial flutter which was managed with cardioversion and the amoebic liver abscess was treated initially by percutaneous drainage then by complete evacuation during laparotomy.

We concluded that this was an instance of pericardial complications (atrial flutter and pericarditis with effusion) due to right lobe amoebic liver abscess because these complications developed during the course of hospitalisation and promptly resolved with successful treatment of liver abscess.

This case has been presented for the following reasons. First, pericardial and cardiac rhythm complications due to ALA are rare and more so are from those arising from right lobe. Second, though the mortality in patients developing amoebic pericarditis is very high, prompt diagnosis and treatment of the underlying liver abscess can lead to complete resolution of these complications. Third, unless the entity of liver abscess causing pericarditis and flutter are considered, the tachycardia may be mistaken for a catastrophic intra-abdominal complication. The awareness of this entity may prevent an un-necessary laparotomy as happened in our patient.

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