Cardiac Tamponade Due to Liver Amebiasis Rupture

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ABSTRACT

Amebiasis is common cases in Asia, Africa, and South Africa. Liver amebiasis has become a serious problem worldwide especially in health and social aspect. The protozoa named Entamoeba histolytica was easily found in area with poor sanitation, low socioeconomic status, and poor nutrition status. The incidence of amebiasis in several hospitals in Indonesia is 5-15% per year. Epidemiological observation showed the comparison of incidence among male and female population and it was approximately 3 : 1 until 22 : 1 with male predominance. The potential age suffered from amebiasis is around 20 - 50 years old. The route of infection spread to oral-fecal and oral-anal-fecal.

The most common complication is abscess rupture (5-15.6%). Rupture may be located in pleural cavity, pericardial cavity, lung, bowel, intraperitoneal, and skin. Rupture of liver amebias spread to pleural and pericardial cavity is a rare case and frequently under reported.

This case report illustrates a 40-year-old male with cardiac tamponade due to rupture of liver amebiasis. Patient's was admitted with chief complaint of shortness of breath, positive Beck's triad, hepatomegaly, pleural effusion, liver abscess on sonography and swinging of heart on echocardiography. This patient was treated with metronidazole as a drug of choice, and pericardiocentesis for the cardiac tamponade.

Keywords: *cardiac tamponade, Entamoeba histolytica, oral-anal-fecal, metronidazole, pericardiocentesis*

INTRODUCTION

Amebiasis is common in Asia, Africa, and South America, and also endemic in many regions of the Philippines. Hepatic involvement is a frequent extra intestinal complication which is encountered in 3-9% of the amebiasis cases. In Africa, over a period of 20 years, the King Edward VIII hospital was reported 2,074 cases of hepatic abscess, while in New York City at the Mount Sinai hospital only 11 cases were seen in approximately 500,000 admissions. The Santo Tomas University hospital in Manila had an incidence of one hepatic amebic abscess per thousand admissions over a ten-year period. Despite the advances in diagnostic tests and management, amebic liver abscess remains a health problems in many parts of the world with poor hygiene.[1]

Approximately 10% populations in the world, especially in developing countries, were easily suffered from *Entamoeba histolytica (E. histolytica)* infection, but only 10% symptomatic. The Incidences of liver amebiasis is about 5-15% cases per year. Epidemiological observation stated that comparison incidence of this disease among male and female is 3 : 1 through 22 : 1. which is male 3.4-8.5 times more

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frequent than female. Potential ages infected by liver amebiasis is around 20-50 years of ages and seldom occured in children. The route of infection is through oral-fecal and oral-anal-fecal.[2]

The most common complications is abscess rupture (5-15.6%). The sites of rupture may occured in pleural cavity, pericardial cavity, lung, bowel, intraperitoneal, and skin. Liver amebiasis rupture spread to pleural and pericardial cavity is a rare case and frequently under reported. Prescribing appropriate drugs is able to reduce mortality rate significantly. Mortality rate in well hospital facility is 2%, and the mortality rate in poor hospital facility is around 10%. A high incidence of amebiasis death is due to poor performance status, malnutrition, septic shock, or hepatorenal syndrome.[1,2]

CASE ILLUSTRATION

A 40-year-old male patient, came to hospital with chief complaint pain in upper chest both side and back, which is accompanied by shortness of breath. He also complained of sweating and disable to sleep because of pain. The pain is felt in all kind of position, radiated progressively to both right and left shoulder. The pain was experienced since two weeks before admission, and it has been more severe than before. The shortness of breath was experienced since two days before admission. Others complained about pain in upper right abdominal quadrant, intermittent fever accompanied by chills, anorexia, nausea, vomiting, loss of appetite, history of dark stool, diarrhea two times a day and history of raw vegetables consumption. He never has either heart disease, diabetes mellitus, hypertension, or hospitalized from any other diseases.

On physical examination, we found the patient with severe disease impression, compos mentis, and good nutrition status. Blood pressure is 100/60 mmHg, pulse rate is 120 beats/minute, respiration rate is 40 times/minute, axillary's temperature is 37.8ºC, and positive paradoxal pulse. Head revealed normal examination. On eye examination, conjunctival were anemic, and sclera were icteric. On neck examination, jugular venous pressure was elevated. On thoracic examination, the heart sound was far, and also positive for pericardial friction rub. On lungs examination, there were vesicular breath sound, positive rough rales at left and right lung, and negative wheezing at both sides. On abdominal examination, there was a pain on right upper quadrant, liver was palpated four fingers below costae arc and four fingers below xiphoid process, the palpable liver was smooth and tender. Extremities showed bilateral edema. Blood examination suggested hemoglobin 9.6 mg%, leukocyte 42,200 /uL, thrombocyte 533,000 u/L, aspartate aminotransferase (AST) 94 U/L, alanine transaminase (ALT) 67 U/L, blood glucose 238 mg/dL, ureum 206 mg/dL, creatinin 2.1 mg/dL, sodium 132 mEq/L, potassium 4.66 mEq/L, chloride 105 mEq/L, blood gas analysis showed respiratory acidosis. Chest radiography showed elevated right diaphragm, infiltrate, and right pleural effusion. Electrocardiograph (ECG) records showed sinus tachycardia with non specific ST-T wave changes in lead V3-V4. Echocardiography showed moderate pericardial effusion with ejection fraction 83% and decrement of right ventricle contractility. Sonography examination suggested liver abscess.

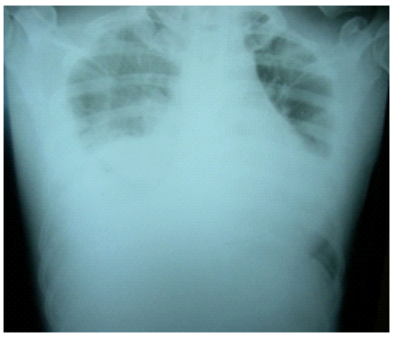

Figure 1. Chest radiography showed elevated right diaphragm, infiltrate, and right pleural effusion

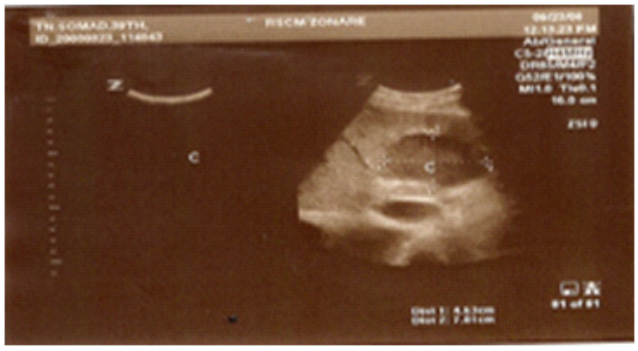

Figure 2. Sonography showed liver abscess

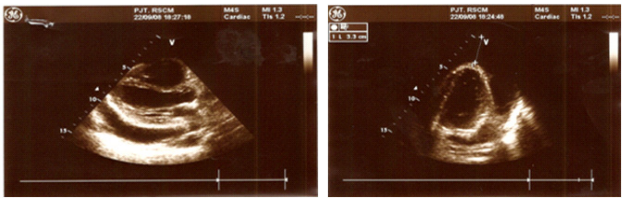

Figure 3. Echocardiography showed severe pericardial effusion suspicious to tamponade

Patient had working diagnosis of sepsis with disseminated intravascular coagulation (DIC), suspicious multiple organ dysfunction syndrome (MODS), liver abscess, severe pleural effusion, pneumonia with right pleural effusion, reactive hyperglycemia, history of melena, acute kidney injury, and anemia.

Patient was underwent treatment in internal medicine ward and given oxygen 2-3 liters, ceftriaxone 3 g once daily, levoflovacin 500 mg once daily,

metronidazol 500 mg three times daily, and heparin 25,000 IU. On the 7th day care, patient underwent second echocardiography and suggested severe pericardial effusion suspicious to tamponade and decided to be punctured immediately. Patient was inserted pigtail catheter through pericardial cavity to evacuate pus daily, and the characteristic color of pericardial fluid were brown. On the 9th day care, patient underwent pleural punctured, and the color of pleural fluid were also brown. On the 11th day care

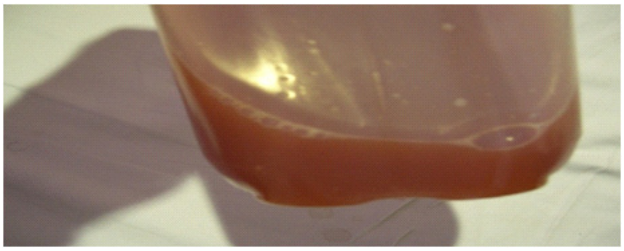

Figure 4. Pleural fluid were chocolate brown "anchovy-like" material

Figure 5. Pericardial fluid were chocolate brown "anchovy-like" material

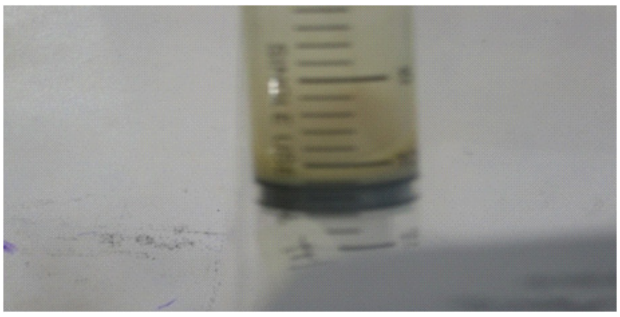

Figure 6. Liver abscess fluid were yellowish color (necrotic material)

patient's liver abscess was evacuated.

Patient was given rifampisin 450 mg once daily, isoniazid 300 mg once daily, ethambutol 250 mg three times daily, pirazinamide 500 mg twice daily, vitamin B6 1 mg three times daily, and prednisone 1 mg/kg BW/day converted to methylprednisolon.

The result of polymerase chain reaction (PCR) tuberculosis (TB) from pleural and pericardial fluid was positive, culture results from both cavities were no microorganism. Culture result from liver abscess was *Acinetobacter calcoaceticus bacteria* that sensitive to the antibiotic which had been given earlier. Amoeba serology test was positive suggested liver amebiasis.

On the day 20th patient condition was getting much better, the sign of infection was diminished, leukocyte 6,000 u/L. The pigtail catheter was removed because the fluid has minimal based on echocardiography. There was no advanced action to the abscess, because based on sonography reexamination suggested reduction in abscess size.

DISCUSSION

Amebiasis is an infection with the intestinal protozoa *E. histolytica*. About 90% of infections are asymptomatic, and the remaining 10% produce a spectrum of clinical syndrome ranging from dysentery to abscess of the liver or other organs.[3] The clinical syndrome from this case show an abscess of the liver and complications of pericardial rupture and pleuropulmonary involvement.

Diagnosis of liver amebiasis recommended using some criteria; Sherlock criteria, Ramachandran criteria, or Lamont and Pooler criteria. In this case fit to the criteria, there are tenderness hepatomegaly, well respond to amebisid medications, leukocytosis, elevated right diaphragm, pus aspiration, sonography suggested abscess, dysentry history, hematology disorder, and positive serology test.[2]

Hepatic involvement is a frequent extra intestinal complication which is encountered in 3-9% of the cases of amebiasis. Amebic liver abscess is a protozoal infestation that starts in the colon. The etiologic agent is *E. histolytica*. The protozoa was discovered by Losch in Saint Petersburg Russia, but named later on by Shaudinn. Its transmission is through contaminated water or vegetables. Amebic infestation is initiated by swallowing the cysts. The cysts are spherical, 10-20 µm in diameter which rupture in the intestinal tract lumen into four metacystic trophozoites. It is through the trophozoites that the disease evolves, often in the cecum and the ascending colon. The amebic protozoa may exist in its commensally state for years before becoming invasive. The invasiveness of the infestation is possibly related to the following factors, there are: diet, host resistance, virulence of the protozoa, humoral and cell mediated immunity. It is the protective cell mediated immunity that may be vital in restricting tissue invasiveness, as well as in aiding tissue resolution. The typical amebic abscess is due to necrotic lysis of the liver tissue, which varies in size from a few centimeters to a large lesion. It is often single, usually in the posterior superior aspect of the right lobe. The abscess is well-demarcated that consists of the chocolate brown "anchovy-like" material of necrotic liver tissue, bile, fat and other products. It is usually sterile but may be secondarily infected. Seen externally, the hepatic surface is smooth, swollen, and

tender to touch. If untreated, it may rapidly progress to form fibrous reactions on the hepatic surface and the surrounding tissues. Its superior extension can lead to rupture into the pleural cavity. Its rupture inferiorly can lead to fatal peritonitis. The left-sided lesions, though infrequent, can lead to cardiac tamponade. The present study showed complete resolution with almost little residual scarring with successful nonsurgical management.[2]

Infection is normally initiated by the ingestion of fecal contaminated water or food containing *E. Histolytica* cysts. The infective cyst form of the parasite survives passage through the stomach and small intestine. Excystation occurs in the bowel lumen, where motile and potentially invasive trophozoites are formed. In most infections the trophozoites aggregate in the intestinal mucin layer and form new cysts, resulting in a self-limited and asymptomatic infection. In some cases, however, adherence to and lysis of the colonic epithelium, mediated by the galactose and N-acetyl-D–galactosamine (Gal/GalNAc)–specific lectin, initiates invasion of the colon by trophozoites. Neutrophils responding to the invasion contribute to cellular damage at the site of invasion. Once the intestinal epithelium is invaded, extraintestinal spread to the peritoneum, liver, and other sites may follow. Factors controlling invasion, as opposed to encystations, most likely include parasite "quorum sensing" signaled by the Gal/GalNAc-specific lectin, interactions of amoeba with the bacterial flora of the intestine, and innate and acquired immune responses of the host.[3,4] In this case, patients' habits consumed raw or uncooked vagetables and history of diarrhea.

Extraintestinal infection by *E. Histolytica* most often involves the liver. Travelers who develop an amebic liver abscess after leaving an endemic area, 95% do so within 5 months. Young patients with an amebic liver abscess are more likely than older patients to present the acute phase with prominent symptoms of less than 10 days duration. Most patients are febrile and have right-upper-quadrant pain, which may be dull or pleuritic pain in nature and may radiate to the shoulder. Point tenderness over the liver and right sided pleural effusion are common. Jaundice is rare. Although the initial site of infection is the colon, fewer than one-third of patients with an amebic abscess have active diarrhea. The clinical diagnosis of an amebic liver abscess may be difficult to establish because the symptoms and signs are often nonspecific. Since 10-15% of patients present only with fever, amebic liver abscess must be considered in the differential diagnosis of fever of unknown origin. Complications of amebic liver abscess may involve pleuropulmonary involvement, which is reported in 20-30% patients and rupture into pericardium, usually from abscesses of the left lobe of the liver. Hepatobronchial fistula, peritoneal rupture, cerebral involvement, and genitourinary tract infections are also the complications.[4,5]

As the laboratory imaging, it is important to collect a data from stool examinations, serologic test, and non invasive imaging of the liver as the most important procedures in the diagnosis of amebiasis. Fecal findings suggestive of amebic colitis include a positive test for heme, a paucity of neutrophils, and amebic cysts or trophozoites. The definitive diagnosis of amebic colitis is made by the demonstration of hematogenous trophozoites of *E. Histolytica*. Serology is an important addition to the methods used for parasitological diagnosis of invasive amebiasis. Enzyme-linked immunosorbent assays (ELISAs) and agar gel diffusion assays are positive in more than 90% of patients with colitis, amebomas, or liver abscess. Radiographic techniques such as ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI) are all useful for detection of the round or oval hypoechoic cyst. More than 10% of patients who have had symptoms for more than 10 days have a single abscess of the right lobe of the liver.[4,5] In this case, positive serologic result and ultrasonography confirmed the diagnosis.

In acute pericarditis, effusion is usually associated with pain and/or ECG changes, as well as with an enlargement of cardiac silhouette. The chest radiography may show a ''water bottle'' configuration of the cardiac silhouette, but may also be normal. Echocardiography is the most effective imaging technique available since it is sensitive, specific, simple, non invasive, may be performed at the bedside, and can identify accompanying cardiac tamponade. The accumulation of fluid in the pericardial space in a quantity sufficient to cause serious obstruction to the inflow of blood to the ventricles results in cardiac tamponade. The three principal features of tamponade (Beck's triad) are hypotension, soft or absent heart sounds, and jugular venous distention.[6] In this case, patient present with shortness of breath and chest pain, positive Beck's triad, and also echocardiography imaging had confirmed the diagnosis.

Nitroimidazoles, particularly metronidazole, are the mainstay of therapy for invasive amebiasis. Nitroimidazoles with longer half-lives e.g. tinidazole, secnidazole, and ornidazole are better tolerated and allow shorter periods of treatment but are not available in the United States. Nitroimidazoles is able to kill trophozoit in all phase of intestinal, extra intestinal, and cyst. Approximately 90% of patients who present with mild-to-moderate amebic dysentery have a response to nitroimidazole therapy. It reduces pain and

febrile symptoms quickly. In the rare case of fulminant amebic colitis, it is prudent to add broad-spectrum antibiotics to treat intestinal bacteria that may spill into the peritoneum; surgical intervention is occasionally required for acute abdomen, gastrointestinal bleeding, or toxic megacolon. Parasites persist in the intestine in as many as 40-60% of patients who receive nitroimidazole. Therefore, nitroimidazole treatment should be followed with paromomycin or the second-line agent diloxanide furoate to cure luminal infection. Metronidazole and paromomycin should not be given at the same time, since the diarrhea a common side effect of paromomycin, may make it difficult to assess the patient's response to therapy.[4,5,7,8]

Therapeutic aspiration of an amebic liver abscess is occasionally required as an adjunct to antiparasitic therapy. Drainage of the abscess should be considered in patients who have no clinical response to drug therapy within five to seven days or those with a high risk of abscess rupture, as defined by a cavity with a diameter of more than 5 cm or by the presence of lesions in the left lobe. Bacterial coinfection of amebic liver abscess has occasionally been observed (both before and as a complication of drainage), and it is reasonable to add antibiotics, drainage, or both to the treatment regimen in the absence of a prompt response to nitroimidazole therapy. Imaging-guided percutaneous treatment (needle aspiration or catheter drainage) has replaced surgical intervention as the procedure of choice for reducing the size of an abscess.[4,5] This case had successfully evacuated pus from liver abscess as many as 2 cc, and according to microbiology examinations, the result was *Acinobacter colcoaceticus*.

Prognostic factor is determined by severity level of diseases, diagnostic approach, and early treatment, and also susceptibility of amoeba to medications.[9] This case showed complications in pericardial cavity and pleural cavity. Aspiration in both cavities was conducted to treat the patient beside drugs treatment.

From the case above we can conclude that ruptured liver amebiasis spread to the pericardial and pleural cavity with mixed infection between TB and non specific microorganism. This patients was also treated with anti tuberculosis drugs because there were positive PCR TB test on pleural and pericardial cavity. TB PCR is a rapid and reliable test in the diagnosis and management of tuberculosis.[10]

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