Short Report: Budd-Chiari Syndrome as a Vascular Complication of Amebic Liver Abscess

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Abstract. Amebiasis remains a major public health issue in most of the world. Amebic liver abscess is the most common extraintestinal manifestation. A complication such as venous obstruction associated with amebiasis is rare. We report a thrombosis in hepatic veins associated with amebic hepatic abscess in a traveler.

CASE REPORT

An 87-year-old man was initially hospitalized in the Franco-Vietnamese Hospital in Ho Chi Minh City in Vietnam for abdominal pain, fever, chills and anorexia. Laboratory examination showed a leukocyte count of 20,900 cells/mm³ 90% neutrophiles. The C-reactive protein level was 170 mg/L and results of hepatic tests were abnormal with elevated levels of liver enzymes (alkaline phosphatase and γ-glutamyltransferase) 15-fold higher than normal values. A computed tomography (CT) scan of the abdomen showed many focal hepatic lesions.

On initial aspiration of the abdominal lesions, 300 mL of brown fluid was obtained, followed by another 150 mL. Pus was sterile, but the amebic serologic results were positive (agglutination and enzyme-linked immunosorbent assay; Biotrin International, Dublin, Ireland). He was treated with metronidazole, 500 mg, three times a day, and ofloxacin, 200 mg twice a day, for five days and then only metronidazole for a week.

Because of persistent hepatic pain, he was referred to our infectious and tropical diseases unit in France. The patient was afebrile and had a tender right upper quadrant of the liver, subconjunctival jaundice, and bilateral lower limb pitting edema without any portal hypertension signs. He had a weight gain of 4 kg. A chest radiograph showed pleural effusion, and a contrast abdominal CT scan showed a hypodense filling defect, suggestive of thrombus in the median hepatic vein, after the hepatic and portal venous phase (Figure 1).

Congenital and acquired thrombophilia tests were conducted. These tests included autoimmune investigations and a screening for protein C or S deficiency, Factor II, Factor V Leiden gene mutations, myeloproliferative disorders, anti-phospholipid syndrome, and hyperhomocysteinemia. All test results were negative. In addition, there were no underlying comorbidities that could facilitate venous thrombosis.

Progressive recovery was seen after therapeutic anticoagulation by low molecular weight heparin (enoxaparine) given concomitantly with a vitamin K antagonist (fluindione) for three days and then fluindione alone for six months. The patient also received metronidazole, 500 mg three times a day for 3 days to complete the 14-day therapy initiated in Vietnam. Re-evaluation with an abdominal CT scan six months later showed partial regression of hepatic lesions and total regression of the thrombus.

The worldwide incidence of symptomatic amebiasis (colitis and liver abscess) is estimated to be approximately 50 million cases annually, with liver abscess in perhaps 1% of the cases.1 The causative protozoan parasite, Entamoeba histolytica, is a virulent pathogen. Trophozoites of E. histolytica invade the intestinal mucosa, causing amebic colitis, and can breach the mucosal barrier and travel through the portal circulation to the liver.1 Amebic liver abscess is the most common extra-intestinal manifestation of amebiasis, and is often characterized by a painful and enlarged liver associated with fever. This abscess consists of a few E. histolytica trophozoites surrounding dead and dying hepatocytes and liquefied cellular debris.3

Complications of amebic liver abscess include its rupture into pleural, pericardial, and peritoneal spaces but also superinfection, anemia, or compression of nearby anatomic structures such as the biliary duct. The early detection of complications is important in reducing morbidity and mortality.

The association of amebic hepatic abscesses with thrombotic vascular complications is seldomly reported. Budd-Chiari syndrome is a hepatic venous outflow obstruction that involves one or more draining hepatic veins, occasionally involving inferior vena cava. The vascular complications of pyogenic liver abscesses are better known but more prevalent in the portal venous system. Among a study of 81 patients with bacterial abscesses, three vascular complications were observed.2 This obstruction may be secondary to an endoluminal invasion by a malignant tumor (such as a primitive liver or kidney cancer, an adrenocortical carcinoma or a leiomyosarcoma) or a parasitic tumor like alveolar echinococcosis. Rarely, it could be caused by a compression by a benign tumor such as an amebic abscess or a hepatic polycystosis.3 In most cases, the obstruction is caused by a thrombosis.4 Thrombosis is associated in two of three cases to one or several underlying prothrombotic affections, among which the most common are the primitive myeloproliferative syndromes. Factor V Leiden or protein C deficiency, anti-phospholipid syndrome, and nocturnal paroxysmic hemoglobinuria or Behçet’s disease (approximately 50%, 25%, 20%, and 5% of cases, respectively).4

To our knowledge, there is no wide series in the literature assessing the prevalence of thrombotic vascular complications during amebic liver abscesses. However, a study based on autopsy cases reported that 27.5% of portal veins, 29.5% of hepatic veins, and 4% of inferior vena cava were thrombosed.5 These thromboses were caused by either compression of the inferior vena cava or the portal or hepatic veins. The diagnosis of Budd-Chiari syndrome should be considered in a clinical manifestation of prolonged fever unresponsive to treatment for amebiasis or ascites associated with bilateral lower limb edema and collateral abdominal venous circulation.8

Computed tomography is an ideal method for diagnosing hepatic abscess with a sensitivity as high as 97%. Complete resolution of the cavity occurs in 3–9 after treatment. Follow-up imaging studies are not necessary for patients who have
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In our case, we chose for our patient with secondary Budd-Chiari syndrome a simple six-month curative anticoagulation procedure. Evolution was favorable.

As for the other cases reported in literature, a patient was treated by intravenous anticoagulation and thrombolysis because of extension of the thrombus into the right atrium. This therapy resulted in partial regression of the thrombosis after a two-month anticoagulant treatment. In another case, the management of an inferior vena cava thrombosis complicated by pulmonary embolism consisted only of administration of anticoagulants. Computer tomography two months after initiation of the treatment confirmed dissolution of the blood clot from the inferior vena cava. The global duration of the anticoagulant therapy was not mentioned. Invasive treatment of amebic liver abscess–related Budd-Chiari syndrome has not been reported in the literature. Vascular complications of hepatic amebiasis should be routinely investigated in patients with fever, tender liver, or signs of portal hypertension after a 5–7-day anti-amebic treatment.

Received May 3, 2009. Accepted for publication July 14, 2009.

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REFERENCES


Figure 1. Contrast abdominal computed tomography scan showing A, amebic liver abscess (arrow) and B, hepatic vein thrombosis (arrow) in the patient.

clinical resolution of symptoms after an uncomplicated amebic abscess. Computed tomography is also useful for the diagnosis of hepatic veins obstruction although false-positive results may be found, especially in chronic cirrhosis, regardless of its origin. This technique enables visualizing intrahepatic collateral circulation, inferior vena cava obstruction, and ascitis. These elements were not found in our patient, probably because of early management of the thrombosis. In our patient, the pathophysiology of hepatic vein thrombosis could be explained by extension of the abscess and vein compression. Thus, the inflammatory process may have induced intra-luminal thrombosis.

Medical management includes the treatment of the thrombosis and its cause. During the past 20 years, many non-surgical therapies developed for the management of extra-hepatic vascular pathology. These therapies include thrombolysis, angioplasty, stent graft, intrahepatic portosystemic anastomosis, and transjugular intrahepatic portosystemic shunt. Simultaneously, anticoagulants were introduced in the treatment of liver vascular diseases for a variable time depending on the etiology. However, the therapeutic management is still not well codified.