Supra-Infected Hepatic and Renal Amebic Abscesses-A Case Report

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Abstract

We describe the case of a 69-year-old man referred from a nursing home facility for acute severe anemia. On presentation, the patient complained of fever, chills, and abdominal pain. Physical exam was significant for a fever of 103.8 F, pale conjunctivae, rales over the right lung base and direct tenderness to palpation overlying the right upper quadrant. Laboratory analysis revealed a normocytic normochromic anemia of 6.7 g/dL, leukocytosis of 17.2 K/µL, elevated creatinine and hepatic function panel with a urinalysis remarkable for turbid cloudy appearing urine, positive for leucocyte esterase and white blood cells. Abdominal Ultrasonography (US) and computed tomography (CT) of the abdomen and pelvis with contrast revealed a 9.2 cm in maximal diameter low-density collection in the posterior aspect of the right lobe of the liver contiguous with a 7.2 cm maximal diameter multi-loculated collection of the superior pole of the right kidney. CT guided drainage of the liver revealed anchovy paste like material and positive cultures for proteus mirabilis species were obtained from both the hepatic and renal foci. Serum entamoeba antibody testing for IgG was positive and intravenous metronidazole with oral iodoquinol was started. Ultimately the patient’s clinical status improved and he was subsequently discharged (Figure 1).

Keywords: Hepatic abscess; Renal abscess; Ameboesis; Proteus mirabilis

Introduction

Entamoeba histolytica is the causal agent for invasive amebiasis [1]. Predominantly infecting humans and other primates, it is a known anaerobic parasitic protozoan, of the genus entamoeba. First described by Dedor Losch in 1875 in St Petersburg, Russia as amebic dysentery, E. histolytica is estimated to infect about 40-50 million people worldwide. Although most cases of amebiasis are asymptomatic, amoebic dysentery and extra intestinal abscesses may occur, most commonly in the liver. It is most prevalent in developing countries and tropical areas with poor sanitary conditions due to its fecal oral spread. Risk factors also include recent travel to endemic areas, poor hygiene, low socioeconomic status and institutionalization [2].

Infection occurs by ingestion of mature cysts from fecal matter contaminated food, water or hands. These cysts later invade the gastrointestinal tract by a process called extracystation, releasing trophozoites. Trophozoites invade the intestinal mucosa causing amoebic dysentery. Dissemination through the blood stream may occur and known cases of liver, brain and lung invasion have been reported. Renal amebic cysts have been previously described in literature and are exceedingly rare [3,4].

Case Report

We describe the case of a 69-year-old man with a past medical history significant for diabetes mellitus, cerebrovascular accident (CVA) with residual R sided hemiplegia, hypertension, congestive heart failure, vascular dementia and gastritis that was referred to our facility from a nursing home for acute severe anemia. On questioning, the patient complained of fever, chills, and abdominal pain. He denied nausea, vomiting, jaundice, hematemeses, melena, hemathecza or other signs of bleeding. Physical exam was significant for a fever of 103.8 F, pale conjunctivae, rales over the right lung base and direct tenderness to palpation overlying the right upper quadrant with a negative fecal occult blood test. Laboratory analysis revealed a normocytic normochromic anemia with hemoglobin of 6.7 g/dL, leukocytosis of 17.4 K/µL, elevated creatinine and hepatic function panel with a urinalysis remarkable for turbid cloudy appearing urine with leucocyte esterase and white blood cells. Blood cultures were obtained, transfusion of packed red blood cells performed, and broad spectrum antibiotics initiated. Abdominal US revealed a space occupying lesion in the right lobe of the liver.

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measuring 5.7×3.9 cm and a solid mass in the upper right pole of the kidney measuring 6.3×4.1×4.0 cm. Endoscopic surveillance showed multiple ulcers in the gastric antrum and a positive H. pylori CLO test. Intravenous fluid hydration improved renal function and an abdominal CT with oral and intravenous contrast revealed a 7.2 cm maximal diameter multi-loculated collection in the superior pole of the right kidney seemingly contiguous with a 9.2 cm maximal diameter liver collection suspicious for infective versus neoplastic etiology. Urine culture was positive for proteus mirabilis, sensitive to ciprofloxacin and amoxicillin-sulbactam. CT guided drainage of the liver revealed anchovy paste like material and positive cultures for proteus mirabilis species were obtained from both the hepatic and renal foci. Serum IgG Entamoeba antibody testing was positive while blood cultures were negative.

The patient was treated with CT guided drainage of the collection with pig tail placement followed by originally intravenous converted to oral metronidazole, oral ciprofloxacin and oral iodoquinol. Follow up visit revealed failure of resolution of his abscess due to dislodgement of his catheter with subsequent re admission and repeat drainage of his collection. He was eventually discharged and completed a course of intravenous therapy with metronidazole and ampicillin-sulbactam and oral iodoquinol and was subsequently lost to follow up.

Discussion

Amebiasis is a disease most prevalent in developing countries and immune compromised or institutionalized populations. It is most commonly an asymptomatic disease and symptomatic only in 4-10% of patients presenting most commonly as amebic colitis [5]. Less than 1% of infections lead to extra-intestinal manifestations, of which amebic liver abscess is the most common. Case reports exist of Central nervous system (CNS) extensions and very rarely of renal amebic abscesses but to our knowledge there are no reports of renal amebic abscesses with bacterial supra-infection. The most common way of transmission is the fecal oral route or less commonly via direct anal-oral contact with an infected individual. In non-endemic areas amebiasis is detected in travelers to endemic areas, immunocompromised or institutionalized individuals [5]. Of note our patient was a nursing home resident due to debilitation from an old CVA and vascular dementia. Patients usually present with fever, malaise, fatigue, anorexia and intestinal manifestations in 10-35% of the cases. Patients with liver abscesses often additionally have abdominal pain. Diagnosis is usually made after identifying a space occupying lesion in a CT scan. Serum antibodies are positive in 70-80% of the population at presentation. The diagnosis may be confirmed with real time PCR assay. Differential diagnosis includes pyogenic abscesses, echinococcal cysts and malignancy. Gold standard of treatment is metronidazole and aspiration or drainage is advised to rule out supra-infection or differentiate from pyogenic abscesses.

Pyogenic liver abscesses are usually due to diseases of the biliary tract spreading from the gastrointestinal tract via the portal vein, hematogenous spreading via the hepatic artery or trauma. The culprit is most commonly Escherichia coli, Klebsiella, Proteus, Staphylococcus and Streptococcus [6]. Diagnosis is usually made with US or CT scans. The mainstay of treatment is imaging guided drainage or surgical drainage and antibiotic therapy based on the aspirate culture.

Case reports of supra-infection of amebic abscesses exist but such cases are uncommon. One study conducted by Rani et al. [7] revealed that there is a higher incidence of peptostreptococcus (71.4%) and Bacteroides (14.2%) in the pus of these samples [7]. Moreover pyonephrosis leading to liver abscesses are also reported [8].

It is unclear whether this was a renal abscess with intra hepatic extension versus, a liver amebic abscess with concurrent renal bacterial super-infection of a renal cyst, versus liver amebic abscess with renal extension. It is our impression that a highly virulent invasive E. Histolytica affecting the liver may have extended to produce a renal abscess by contiguity. This process may have opened a pathway for colonization with proteus mirabilis species in the liver, originating from a super infected renal amebic abscess via a complicated urinary tract infection. Abdominal US and CT scan are both excellent imaging modalities for detecting hepatic abscesses but both fail to differentiate pyogenic versus amebic causes [9,10]. Though microscopic aspirate analysis was negative for E. Hystolitica, studies have shown that microscopy seldom proves diagnostic if from the fluid aspirate [11]. Confirmation of amebic abscess is made by Entamoeba antibody testing [12]. The available assays to detect antibodies to E hystolitica include IHA and ELISA which are widely used because of their high sensitivity of 99% and specificities of 99.8% [13]. The immunoassay results remain positive for 6-18 months following invasive disease with E. histolytica. As our patient had underlying bilateral renal cysts, colonization from a complicated urinary tract infection with proteus species may be possible while the concern for malignancy was also raised. Renal abscess may occasionally masquerade as a malignancy presenting with hematuria, weight loss and fever. Of note renal abscesses may also complicate renal malignancy [14]. Although pharmacotherapy alone is adequate in 85% of cases, indications for percutaneous drainage include larger abscess (>5 cm) that are at risk of rupture, left hepatic lobe abscess, and treatment failure [11]. Complications of amebic abscess are secondary to bacterial supra-infection, rupture and pleural effusion/empyema. Amebic renal abscesses are extremely rare with only a few cases reported [3,4,15]. Renal abscesses usually arise from ascending urinary tract infections or bacteremia with hematogenous seeding. Kidney stones and diabetes mellitus are the most common risk factors. Gram negative bacteria are the most common etiology. Whether, entamoeba Histolytica invaded the renal abscess remains to be proven. Nevertheless, anti-amebic pharmacotherapy regimens and drainage procedures remain an effective strategy against invasive amebiasis.

References


