

Amoebic Liver Abscess and Chronic Pancreatitis in Children: Coincidence or Correlation?

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Abstract

Recurrent abdominal pain is a common presentation in pediatric patients, with a wide range of possible underlying causes. Amoebic liver abscess (ALA) and chronic pancreatitis, though relatively rare in children, present significant diagnostic, and therapeutic challenges. In this case report, we present a 15-year-old girl who presented with abdominal pain and was subsequently diagnosed with amoebic liver abscess and chronic pancreatitis that occurred an unusual. The patient's condition resulted in a series of medical interventions and hospitalizations due to complications, including pleural effusion. Despite ongoing treatments and diagnostic procedures, the patient's condition worsened over time, leading to recurrent hospital admissions. This case highlights the importance of thorough diagnostic evaluation and timely intervention in managing recurrent abdominal pain in pediatric patients.

Keywords: Amoebic Liver Abscess, Chronic Pancreatitis, Abdominal Pain.

Introduction

Amoebiasis, a parasitic condition, caused by *Entamoeba histolytica*, affects at least 50 million people globally and killing 100,000 individuals every year. The most common extra intestinal manifestation of invasive amoebiasis is the Amoebic Liver Abscess (ALA), apart from its classical amoebic colitis. Amoebic liver abscess is uncommon in children and ten times more common in men than in women. Complications of ALA include pulmonary-pleural form of amoebiasis, secondary lesions in the peritoneal cavity and pericardium, hepato-intestinal fistula, bacterial coinfections, and biliary dysfunction. Hepatic vein thrombosis and portal vein thrombosis are rare complications of ALA. The incidence of pancreatitis has not been reported.¹

Pancreatitis as a complication of ALA has not been establish. Suppurative pancreatitis with associated liver abscess has been reported in 1945. Szanto et al. reported the first case of hepatic abscess after choledochal-duodenostomy, and their patient presented with cholangitis 1 year after operation. A prospective study of patients undergoing pancreatic jejunostomy reported spontaneous abscess formation in the pancreas and liver during follow-up for chronic pancreatitis, but pathogenesis liver abscess causes pancreatitis still unclear.^{2,3} This case report discusses a child with an amoebic liver abscess and chronic pancreatitis.

Case Report

A 15-year-old girl was admitted to our hospital complaining of abdominal pain. The patient had a history of recurrent abdominal pain that persisted for several months. Physical examination revealed pale conjunctiva, dyspnea, no jaundice, and no cyanosis. Abdominal examination revealed slight distension, epigastric tenderness, hepatomegaly, spleen schuffner 1. From the results of examinations for the first time showed a cystic mass in the epigastric region and left pleural effusion. Abdominal computed tomography revealed abscesses in the lesser curvature of the stomach and pancreatic abscess. Serial laboratory examinations were then performed. Amylase was increase and lipase still in normal limit (Table 1). An MRCP examination was performed for dilated ductus pancreas with multiple pseudocysts in the parenchyma and multiple calcifications, liver abscess in peripheral segments II and III of the liver lobes, and gall bladder hydrops [Figure 1a].

Table 1: Laboratory Examination during hospitalization (November 2022 to March 2023).

	Nov 2022	Dec 2022	Feb 2023	Mar 2023
Amylase (25-125)	351	106	37	85
Lipase (73-393)	362	233	119	48
Serology IgG <i>E. hystolitica</i>			Negative	
Hemoglobin (12-15 g/dL)	7.8			
Hematocrit (35-48%)	25.2			
Leukocyte (3.6-11 x 10³/mm³)	24260			
Thrombocyte (150-450 x10³/mm³)	79000			
MCV (86.7-102.3%)	84.8			
MCH (29.7-32.4%)	26.3			
MCHC (29.7-33.1%)	31			
Basophil (0.3-1.4%)	0.6			
Eosinophil (0.6-5.4%)	0.2			
Neutrophil (39.8-70.5%)	88.4			
Lymphocyte (23.1-49.9%)	4.8			
Monocyte (4.3-10%)	6			
APTT (9-12)	17.6			
PPT (23-33)	52.7			
ALT (0-50)	17			
AST (0-50)	47			
BUN (7-18)	3			
Creatinine (0.6-1.3)	0.5			
Procalcitonin	1.36			
CRP	9.85			

The patient underwent serial laboratory and radiological evaluations. Amylase and lipase serum levels were normal results a month later. The MRCP evaluation was performed 3 months later, and the results still showed liver abscesses in segments V, VI, VII, and VIII and an edematous pancreas [Figure 1b]. However, the immunological test for IgG *E. hystolitica* was negative. After a month, the patient underwent core liver biopsy under ultrasound guidance, and trophozoites were identified [Figure 2]. The patient was diagnosed with amoebic liver abscess and chronic pancreatitis and received antibiotic treatment for 2 months, pain management, and parenteral nutrition. The MRCP evaluation performed after 1 month and showed improvement and patient discharged [Figure 1c].

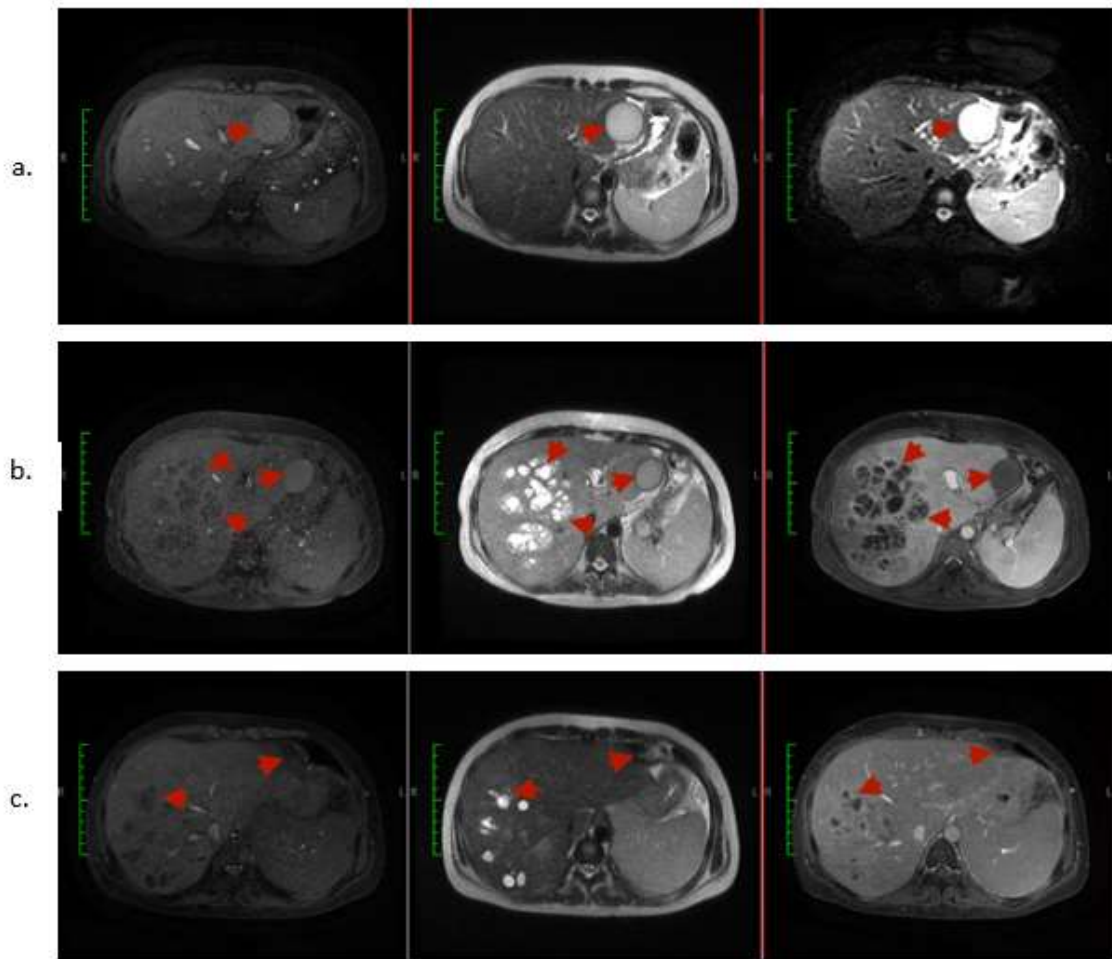


Figure 1: (a) Liver abscess in peripheral segment II, III of the liver lobe (MRCP November 2022); (b) Liver abscesses in segments V, VI, VII, and VIII of the liver and edematous pancreatitis (MRCP evaluation in February 2023); (c) Compared to the previous MRI, **the abscess impression was reduced**), simple cyst in segments II and III of the liver lobe (MRCP evaluation in March 2023).

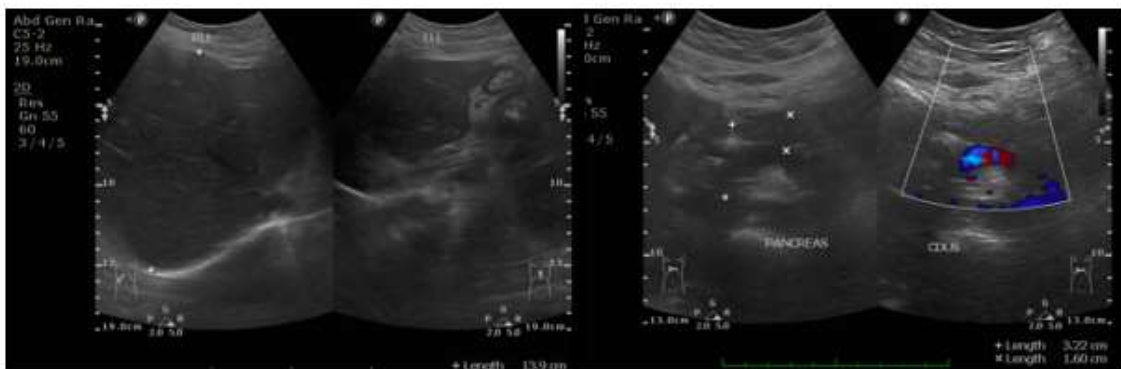


Figure 2: Liver in segments V, VI, VII, and VIII of the liver, Edematous pancreatitis (Abdomen ultrasonography, February 2023).

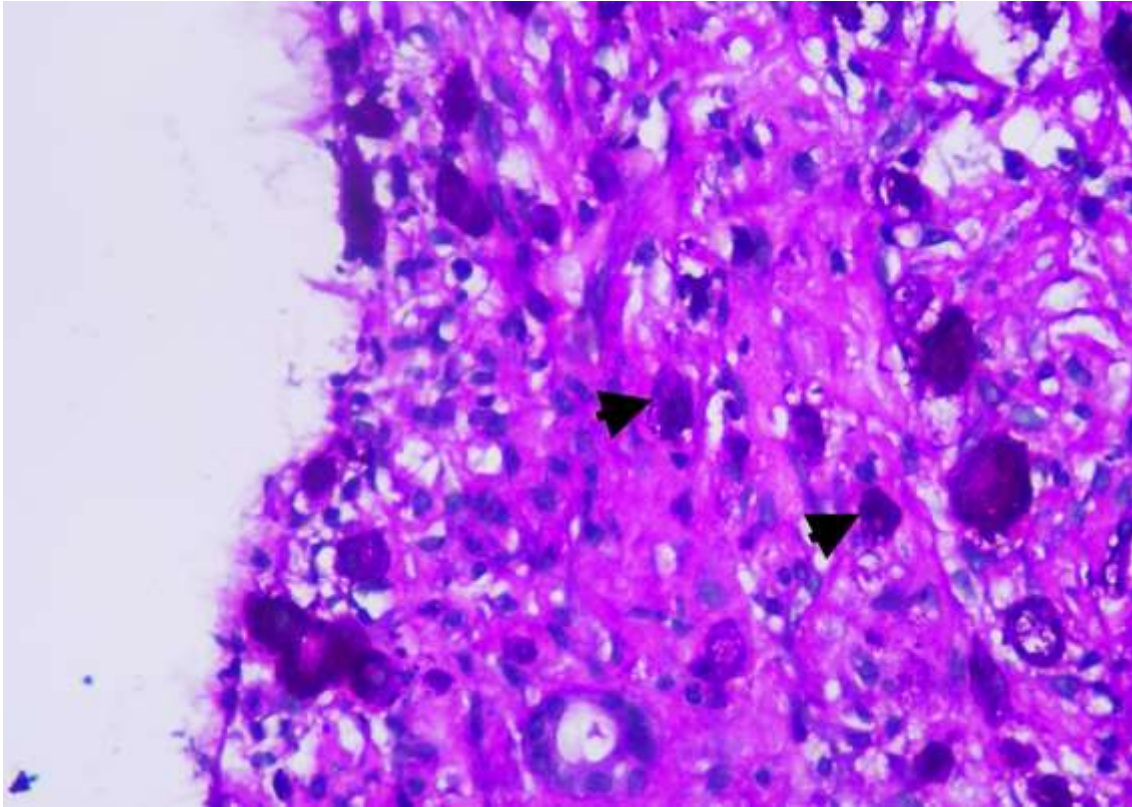


Figure 3: Liver Biopsy shown trophozoite amoeba (The biopsy tissue consists of fibrous connective tissue with proliferation of capillaries accompanied by inflammatory cells such as lymphocytes, neutrophils, monocytes and some eosinophils. Among them are several amoeba trophozoites (on PAS staining).

Discussions

Amoebic liver abscess is a pathological cavity arising in the liver tissue due to amoebic infection, characterized by suppuration with pus formation accumulating purulent necro-inflammatory debris consisting of necrotic liver tissue, inflammatory cells, and/or blood cells within the liver parenchyma. Amoebic liver abscess is the most common extraintestinal manifestation of amebiasis. Amoebic liver abscess is caused by *Entamoeba histolytica* infection.⁴⁻⁶ This parasite can cause invasive disease in the human host, including amoebiasis, which primarily affects the large intestine and can lead to the formation of liver abscesses when the parasites migrate to the liver via the bloodstream.⁷

This abdominal pain can also be a common symptom of amoebic liver abscess and pancreatitis, although in most cases, patients also have a fever.^{8,9} Right upper abdominal pain is also present in 75% - 90% of patients with amoebic liver abscess. This abdominal pain is more severe than in patients with pyogenic liver abscesses. The pain may be dull or pleuritic and may radiate to the shoulder. The pain may be accompanied by nausea, vomiting, anorexia, weight loss, and weakness. Pain in the right upper abdomen will be seen from the patient's typical movement, walking bent forward with both hands on the abdomen.^{8,9}

Diagnosis of amoebic liver abscess is challenging. The diagnostic methods used to evaluate the cause of liver abscess and chronic pancreatitis are based on clinical, biochemical, radiological, and histological findings. In this case, liver abscesses were reported on ultrasound and observed on MRCP. However, serological tests for *E. histolytica* (Eh) showed negative results, ALA was diagnosed via liver biopsy.

Serological tests detect antibodies to *Entamoeba* antigens with a sensitivity of 65-92% and a specificity of > 90%. Moreover, the antibody detection tests seem to be time- and cost-effective. Another difficulty also exists for the detection of antibodies to *E. histolytica*: serological methods cannot be performed in a

timely manner.¹⁰ Puncture of the abscess with aspirate collection is of diagnostic and therapeutic importance. The high density and chocolate color of the obtained aspirate are characteristic. Culture both of blood and aspirate for bacterial infections with aerobic and anaerobic flora and fungi are necessary, where indicated, by colonoscopy and biopsy of intestinal amebic lesions or by drainage of liver abscess.^{10,11}

The basic pathogenesis of chronic pancreatitis is a persistent inflammation of the pancreas, resulting in tissue injury. Histopathologic findings include pancreatic parenchymal destruction, interstitial fibrosis, loss of acinuses, and varying degrees of inflammatory cell infiltration. This results in the central and branch pancreatic ducts being dilated or stenosed in an irregular pattern, as well as protein plugs and calcifications found in the pancreas.¹²

Laboratory examination showed that the patient had elevated amylase and lipase. The results of biochemical examinations during an acute exacerbation are like those of acute pancreatitis, including elevated serum amylase and lipase. However, these test results are often standard with non-specific findings during the symptom-free interval. Chronic pancreatitis patients with severe pancreatic insufficiency have decreased trypsinogen, lipase, elastase-1, and amylase serum levels. These decreased serum pancreatic enzyme levels are precise (92-98%) but highly insensitive (20-32%) in the diagnosis of chronic pancreatitis.¹³

CT scan, MRCP, and ERCP examination showed the patient had chronic pancreatitis and liver abscess. MRCP examination has been used in pediatric patients and helps determine the cause of chronic pancreatitis and in following its clinical course. ERCP is performed selectively in patients with a high index of suspicion for pancreatic duct anomalies not detected by MRCP, as well as for therapeutic purposes.¹³

The radiologic examination can confirm acute or chronic inflammatory changes in the pancreas. Abdominal plain photographs and computed tomography (CT) scans show calcifications throughout the pancreas, and ultrasonography and CT depict pancreatic atrophy, fat replacement, dilation of the central pancreatic duct, and stones. Contrast-enhanced CT scans of the abdomen are used primarily to assess smaller abscesses. CT abdomen has a similar sensitivity to ultrasound (95%). MRI examination does not have a higher sensitivity than CT but can be performed when CT imaging gives doubtful results.^{9,14}

In our case, it was an amoebic liver abscess and chronic pancreatitis. Anatomically, at least in this case it is difficult to explain that pancreas and liver might have involved from a common source. Most probably two diseases might have occurred simultaneously. The liver and pancreas are closely associated organs that share a common embryological origin. They display amphicrine properties and have similar exocrine organization with parenchymal cells, namely, hepatocytes and acinar cells, secreting bile and pancreatic juice into the duodenum via a converging network of bile ducts and pancreatic ducts.¹⁵ Pyogenic liver abscess associated with acute pancreatitis was first reported in 1945 by Shallow et al.,² which was detected perioperatively. Pyogenic liver abscess in chronic alcoholic pancreatitis that was reported by Gundling et al. in 2004 was due to a pancreaticohepatic fistula. However, in our case no fistula could be documented.

The mortality of amoebic liver abscess without complication does not exceed 1%. Amoebic liver abscess is a disease with a good prognosis and proper diagnosis and management. The prognosis of amoebic liver abscess in the left lobe of the liver is worse as there is a greater chance of rupture. The prognosis may worsen if the diagnosis of amoebic liver abscess is made late. The prognosis also worsens in amoebic abscesses with complications. The mortality rate of amoebic liver abscess increases with abscess rupture into either the peritoneum or pericardium.^{9,16}

Conclusion

Recurrent pain in the upper abdomen is the most common complaint experienced by patients with pancreatitis and liver abscess. In this patient was already have a case of chronic pancreatitis and the amoebic liver abscess is a new insult. Amoebic liver abscess and chronic pancreatitis occurring together might initially seem coincidental. Understanding these connections is crucial for developing comprehensive treatment strategies that address both the direct causes of these conditions and the underlying factors that may contribute to their severity and persistence.

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Statement of Ethics

This case report was published in accordance with the Declaration of Helsinki of the World Medical Association. Written informed consent was obtained from the patient for publication of this case report and any accompanying images. Ethical approval was not required for this study in accordance with national guidelines.

Conflict of Interest

The authors declare that there is no conflict of interest.

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