from endemic areas. Other tests like RT-PCR and antigen detection on pus are available but their role is not yet established.

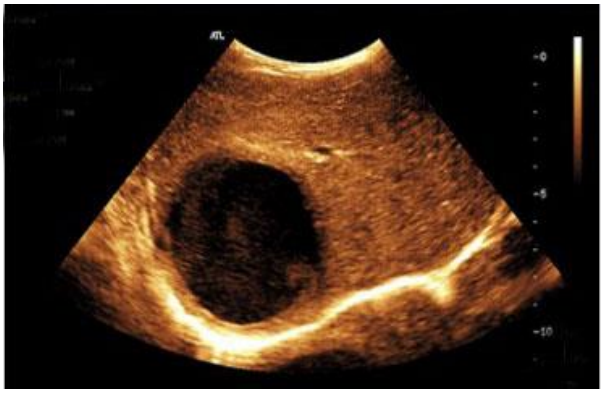
Ultrasound

Liver

Ultrasound is reported to be as sensitive as CT and MRI, but very early pre-colliquative stages cannot be detected. On ultrasound, ALA lesions are typically single (in over 60% of cases), located in the right hepatic lobe near the surface of the organ, round or oval in shape. They appear hypoechogenic, with initially irregular and ill-defined margins (first 4–5 days); occasionally they are hyperechoic. Later, with the progressive colliquation of necrotic material, the lesion assumes a homogeneous hypoechoic pattern, with regular, well-defined margins [Figure 1] (2, 3). This appearance typically occurs within 2 weeks. In immunocompromised patients, the amoebic abscess can assume a tumour-like or honeycomb appearance. In the healing phase, a slow progressive evolution can be observed with the lesion increasing in echogenicity and showing an irregular and ill-defined margin. Sometimes a sterile cystic cavity can persist for months or years (4-6).

Figure 1 Different sonographic appearance of ALA. Well-defined margins with almost echo-free content in a quasi-cystic ALA (a). Hypoechoic with centrally located necrotic areas (b). Large hypoechogenic lesion with almost solid content (c).

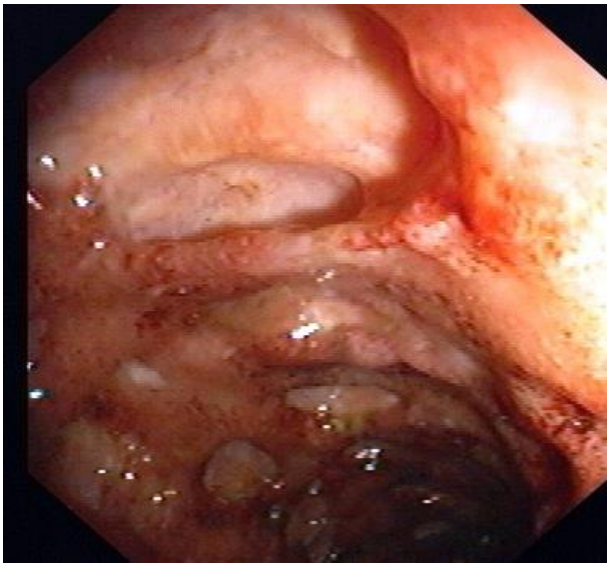
a



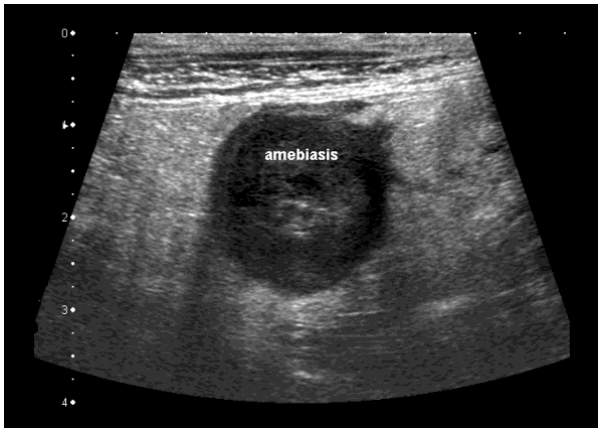
b

Figure 2 **Amoebic colitis. Endoscopy reveals typical ulcerations (a). Ultrasound shows the signs of severe ulcerative colitis including loss of layer structure, transmural inflammation and surrounding peri-intestinal inflammatory reaction (b).**

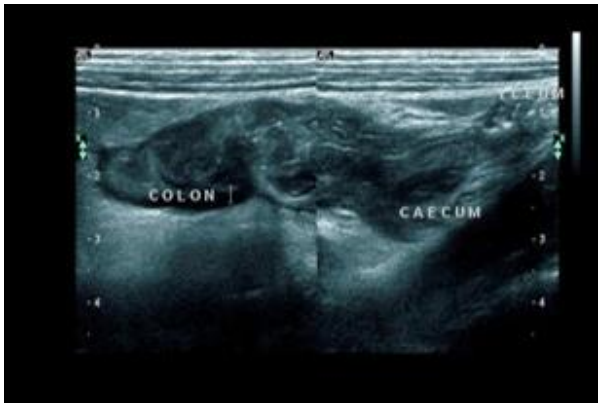
a

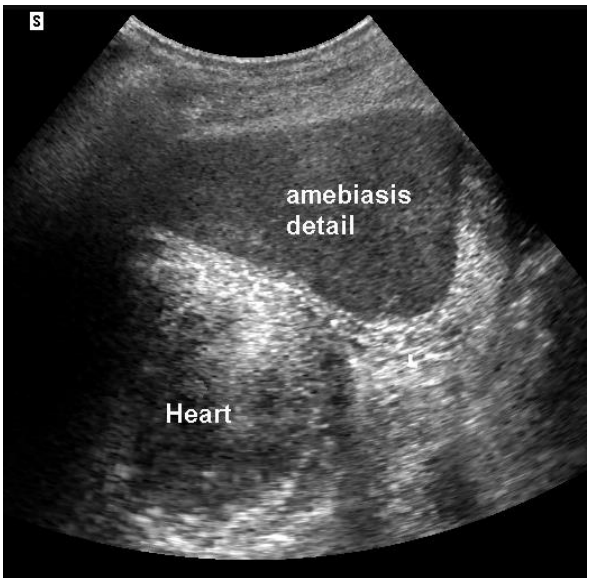


b

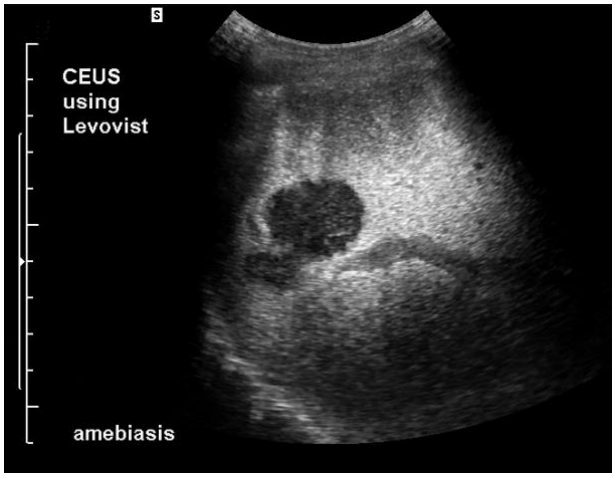


c





c



Primary treatment of uncomplicated amoebic liver abscess consists of a tissue agent and a luminal agent to eliminate intraluminal cysts.

Metronidazole

Patients with uncomplicated amoebic liver abscess are traditionally treated with metronidazole orally 3 x 500 mg for 7 to 10 days (17). The cure rate is >90%. Shorter duration of treatment is not recommended. Intravenous treatment generally offers no advantage since metronidazole is well absorbed from the gastrointestinal tract. In case of slow response to metronidazole or relapse following therapy, percutaneous catheter drainage, and/or a prolonged course of metronidazole may be warranted. In pregnancy, metronidazole

hyperechoic wall. The residual lesions have been found to persist for more than a decade and may pose differential diagnostic problems (24).

Ascariasis

Introduction

An estimated 1.2 billion people are infected by *Ascaris lumbricoides*, making ascariasis the most common human helminthic infection (25). Although most infections are asymptomatic, over 250 million people are estimated to suffer from associated morbidity, and more than 200,000 deaths are attributed to ascariasis every year. Ascariasis is a significant cause of biliary disease in areas where the rate of infection is high and *Ascaris*-infections account for up to 10-19% of all hospital admissions. Ascariasis is found throughout the world, but it is more common in warm climates and overcrowded rural communities with inadequate sewage systems (26). The infection is more common and severe among children, whereas biliary ascariasis is more common in adults (27, 28).

Adult worms live in the small intestine, usually the jejunum, in which the females produce eggs that are passed into the faeces. In the environment, the larva develops within eggs in approximately 3 weeks. Infection occurs through ingestion of material (soil, food or water) contaminated with larva containing (i.e. fertile) eggs. Once swallowed, the larvae hatch and invade the intestinal mucosa. They then migrate through the portal and then systemic circulation to the lungs. Here the larvae penetrate the alveolar walls, ascend the bronchial tree to the throat and are swallowed again. On reaching the small intestine, they develop into adult worms [Figure 5] within 2–4 weeks.

Diagnosis

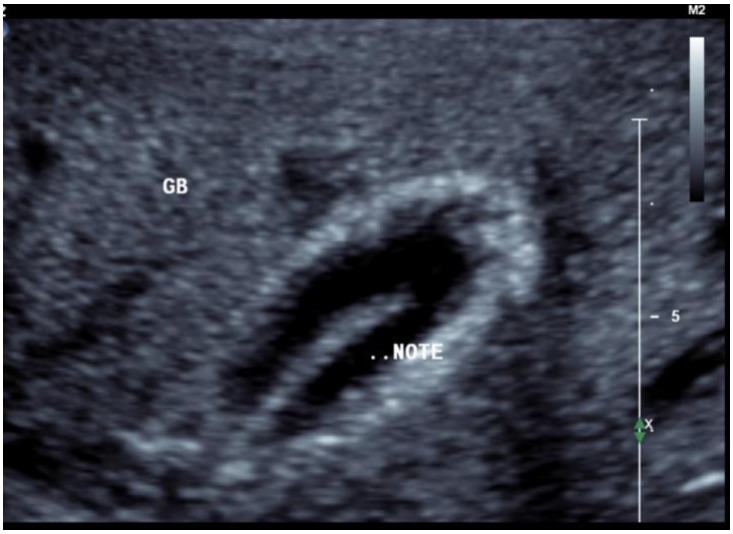
Diagnosis of intestinal ascariasis is usually achieved by parasitological stool examinations with visualization of the eggs. In patients with high worm burden the worms can be also excreted as a whole.

Ultrasound

Ascaris lumbricoides worms in the intestinal tract may be missed by ultrasound because of bowel gas. Ultrasound is a highly sensitive and specific non-invasive method for the detection of worms in the biliary tract [Figure 6], although the diagnosis of biliary ascariasis requires a high index of suspicion because the worms move in and out of the biliary tract and can be missed on biliary imaging (27, 28).

Figure 6 Ultrasound images of *Ascaris lumbricoides* in the gallbladder (a) in the common bile duct (b) and in the intrahepatic bile ducts (c) as well in the appendix (d) and the gut (e, f).

a



b

structure with a hypo- or anechoic centre, and can be seen moving with a slow-waving pattern. Multiple worms in the bile duct produce a spaghetti-like image, with alternating echogenic and anechoic strips or, if densely packed in the bile duct, can appear as an hyperechoic pseudotumour. On transverse sections, a “bull’s eye” echo can be seen owing to the presence of a worm in the dilated bile duct.

Treatment

The management strategy for patients with biliary ascariasis depends on the clinical situation; it can include conservative management, endoscopic extraction or surgical intervention. In most cases, pathology resolves with pharmacological treatment and response to treatment can be monitored by ultrasound (30). Conservative treatment includes the use of analgesics, antibiotics for pyogenic cholangitis and oral administration of albendazole, which paralyses the worms so that they can be expelled. Symptoms resolve within 3 days in 60–80% of patients, accompanied by the disappearance of worms on ultrasound. Endoscopic intervention is indicated in cases of acute severe pyogenic cholangitis, recurrent biliary colic non-responsive to analgesics, high amylasaemia, and when the worms persist in the bile duct for longer than 3 weeks (probably because they are dead). Endoscopic extraction of worms across the papilla leads to rapid resolution of symptoms and can be performed using grasping forceps or a Dormia basket (31). Surgical intervention is required when endoscopic treatment fails, or if the worms are located in the intrahepatic bile ducts or in the gallbladder.