



Review Article

Gastrointestinal parasites of surgical importance and their management — A review

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ABSTRACT

Parasitic diseases continue to affect millions of people in the endemic areas causing morbidity as well as affecting the quality of life. The visitors to these endemic areas get infected with these parasites and present a confusing picture to the physicians in their native area. Prolonged exposure to some of these parasites like ‘*Opisthorchis viverrini*’ is a well-known risk factor for malignancy. However, most of these diseases are easily curable. This review article provides a brief description of a few parasitoses like amebiasis, ascariasis, clonorchiasis, opisthorchiasis, and fascioliasis. Their causative agents, disease-causing mechanisms in the host, clinical features, specific radiologic findings, and immunodiagnostic tests are discussed. The medical and surgical aspects of treating these parasitoses are also summarized.

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1. Introduction

A parasite (*Greek: parasitos*) is defined as an “organism that lives in a close association with another organism of a different species (host), derives nutrition from it and is pathogenic to it, although this potential may or may not be always expressed”.¹ Although any infectious agent is, in strict terms, a parasite, medical terminology uses this word in a narrower sense to mean eukaryotic pathogens, the protozoa, and helminthic organisms. Though the connection between humans and parasites seems to have existed since prehistoric times, it was only in the nineteenth century that the discoveries resulting from exploration and colonization were pieced together.² Many life cycles of parasites were elucidated and the role of parasites in human disease became evident. Parasitology thus gained a firm footing in medicine. This article reviews a few parasites, which infect the gastrointestinal tract and are of relevance to the surgeon. Important clinical features, laboratory tests,

imaging findings, and surgical aspects of management are highlighted.

2. Entamoeba Histolytica (Amebiasis)

Entamoeba histolytica causes invasive amebiasis. It ranks third in causing death due to parasite disease, after Schistosomiasis and Malaria. This parasite is endemic in areas, where overcrowding, poor sanitation, and low socioeconomic conditions are prevalent.³ In developed countries, travelers, immigrant populations, homosexual men, HIV-infected individuals, individuals in mental institutions, and prisons constitute high-risk groups. This disease spreads via the fecal-oral route. Humans get infected when they consume food and water contaminated with feces containing amebae.

3. Pathogenesis of Entamoeba Histolytica

The life cycle of this parasite is depicted in Figure 1. In the colon, trophozoites of *E. histolytica* adhere to the Gal/GalNAc adherence lectin on colonic epithelial

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cells.⁴ This stimulates the secretion of interleukin-8(IL-8) and tumor necrosis factor (TNF α) leading to polymorphonuclear cell infiltration.^{5,6} The resulting interaction leads to inflammatory cell death and necrosis of tissue, exposing the extracellular matrix. The amebae being highly pathogenic, release multiple enzymes like cysteine protease, collagenase, and several polypeptides that degrade host defense molecules such as IgA and complement proteins. This results in widespread destruction of submucosal tissue and the formation of flask-shaped ulcers in the colon (histo = tissue, lytica = death). In some cases, trophozoites gain access to a submucosal venule, and move through the portal circulation, to reach the liver. The right lobe of the liver receives venous blood from the right side of the colon, making it the most common site of abscess formation. The sluggish blood flow in the sinusoids creates a perfect environment for their lodgment and multiplication. Amebae damage the neutrophils, and the lysosomal enzymes so released, which promote hepatocyte death. This produces a cavity filled with blood and liquefied liver tissue, known as "Anchovy sauce pus," at aspiration.

4. Clinical Features of Amebiasis

After about 2-6 weeks of the incubation period, the patient presents with lower abdominal discomfort and loose stools.⁷ The stool frequency increases in severe disease. The content of the stool is mainly blood and mucus with very little fecal matter. Erosion of a blood vessel in the ulcer bed results in bleeding. Perforation of the ulcer leads to peritonitis. The cecum and rectosigmoid junction are favored sites for the formation of the 'amoebic granuloma' or 'ameboma' (Figure 2A), a lesion resulting from a florid inflammatory process surrounding a necrotic area, where the hypertrophic granulation tissue and fibrosis cause the formation of a pseudotumor. This occurs rarely, in about 1-2% of patients with repeated bouts of infection and noncompliance with the treatment. These mass lesions may be mistaken for malignancy and resected, only to reveal the benign nature of the disease.⁸

In the amebic liver abscess, there is sudden onset right-sided abdominal pain. Fever, accompanied by rigors and profuse sweating, may be present. The pain is constant, severe, and increases with deep inspiration or hiccough. The classic sign on physical examination is painful hepatomegaly with guarding in the right upper quadrant. Mild jaundice is common.

5. Diagnosis of Amebiasis

5.1. Blood tests

The laboratory tests may be normal in mild cases of colitis, with leukocytosis occurring in severe disease. The white blood cell count is raised above $10 \times 10^9/L$ in most patients with amebic liver abscesses. Alkaline phosphatase

(ALP) level rises in long-standing disease,⁷ Serum levels of transaminases are elevated in 50% of cases and signify acute disease or complications.

5.2. Stool microscopy

The classical finding on stool microscopy is the presence of hematophagous trophozoites (i.e., trophozoites which have ingested red blood cells). Microscopy has fallen out of favor as it requires skill and experience on the part of the microscopist and it has a low sensitivity (10-60%).

5.3. Serology

Antigen detection using enzyme-linked immunosorbent assay (ELISA) is the preferred investigation in the endemic areas. They show 55% to 100% sensitivity, and 93% to 100% specificity in comparison to the other established testing methods. The antibody detection in serum is better in non-endemic areas. Again, ELISAs are the most sensitive with very low false-negative results. The serological tests should be repeated in 5-7 days, as they may be initially negative in the acute phase of infection.⁹

6. Radiology

In the acutely ill patient, if a plain erect abdominal radiograph shows colonic dilatation or gas under the diaphragm, surgery must be considered (Toxic megacolon or hollow viscus perforation needs exploration regardless of the etiology). On ultrasound, amebic liver abscesses are often located near the liver capsule presenting as a unilocular, round-oval, hypoechoic lesion with low-level internal echoes and no discernible wall echoes. An adjoining disruption of the diaphragm is highly suggestive of the diagnosis.¹⁰ On CECT, they appear as peripherally enhancing, well-circumscribed, rounded lesions with fluid attenuation of 10-25HU suggesting complex fluid. The thick peripheral wall (3-15mm) may show a 'target' or 'double rim' appearance with a surrounding rim of edema (Figure 2B). The central cavity could show fluid-debris levels or multiple septae too.¹¹ On MRI, amebic abscesses show homogenous low signal on T1 and high signal on T2 weighted images respectively, simulating the other pyogenic abscesses with perilesional edema in about 50% of cases.

7. Management of Amebiasis

The amebic infections are treated with two classes of drugs. The luminal amebicides (such as diloxanide furoate and iodoquinol) are poorly absorbed from the intestine, hence acting best on luminal amebae. Tissue amebicides, such as metronidazole, tinidazole, dehydroemetine, and chloroquine reach a high concentration in blood and tissue after oral or parenteral administration. Hence, they are best used in the treatment of amebic abscesses. They are less

effective against luminal organisms.

For the liver abscess, combined treatment with metronidazole or tinidazole, followed by diloxanide furoate is recommended. More than 90% respond to this treatment, becoming afebrile in about 3–5 days. Dehydroemetine and emetine can cause nausea, vomiting, and cardiac arrhythmias and are best used as second-line agents.

8. Role of Surgery in Amebiasis

Routine aspiration or ultrasound/CT guided drainage is avoided, except¹² when the diagnosis is in doubt — serology is inconclusive & pyogenic liver abscess cannot be ruled out; when the antiamebic drugs are contraindicated (e.g. pregnancy); when the secondary infection of the abscess occurs (15% of cases);¹³ when fever and pain do not respond to 5 or more days of anti-amebic therapy and when rupture of the abscess into the thorax or pericardial cavity is imminent. The surgical intervention is indicated only for complications.¹⁴

8.1. Colitis with hollow viscus perforation

Laparotomy followed by a thorough peritoneal lavage is done. The decision regarding primary closure of the perforation, resection of the affected segment of bowel, or proximal diversion is made on a case-to-case basis. Postoperatively metronidazole is added to the antibiotic regimen.

8.2. Abscess rupture into thoracic cavity

Intercostal tube drainage is tried first, with the positioning of the drain as high as possible on the right lateral side of the chest. Aggressive procedures such as thoracotomy and drainage or decortications may be required in chronic cases or loculated collections.

8.3. Abscess rupture into peritoneum

Thorough lavage of the peritoneal cavity is given, and the liver is handled as gently as possible. The temptation to break all “loculi” should be resisted — these loculi are blood vessels and ducts in the abscess cavity. Endoscopic stenting is a feasible option for postoperative bile leaks in case they occur.

9. Ascaris Lumbricoides (Ascariasis)

The roundworm *Ascaris lumbricoides* causes Ascariasis. It is the most common human parasitic infection in east and south Asia and sub-Saharan Africa. In endemic areas, about a quarter of the population may be infected.¹⁵

9.1. Life cycle of *ascaris lumbricoides* and pathogenesis (Figure 3)

The *Ascaris lumbricoides* larvae cannot move through the pulmonary capillaries. Hence, they burrow through the alveoli moving retrogradely to the trachea. A strong eosinophilic inflammatory response occurs, causing ‘Charcot Leyden crystals’, along with segments of dead larvae to appear in the sputum.

Adult worms migrate when they sense a disturbance in the environment. Stressful situations like fever, diarrhea, anesthesia, etc. are well-known triggers for worm migration.¹⁶ The migration can occur into the common bile duct (most common) or the gallbladder or the intrahepatic ducts.¹⁷ Sphincterotomy, sphincteroplasty, or bilio-enteric anastomosis reduces the resistance, making migration easier. In addition, the hormone progesterone has a relaxing effect on the sphincter, thereby explaining increased symptoms during pregnancy. Impacted worms cause spasms of the sphincter of Oddi, resulting in obstructive jaundice, suppurative cholangitis, and acute pancreatitis.¹⁸ The worm sometimes dies and disintegrates, eliciting chronic inflammation that leads to the formation of strictures in bile ducts. Fragments of the dead worm or ova can also act as a nidus for the stone formation. This has been demonstrated by some authors on microscopic examination of stones.¹⁹

10. Clinical Features of Ascariasis

Light infections do not usually cause symptoms. Acute manifestations occur with an increasing worm burden i.e., more than 100 worms in the GI tract. A pneumonitis occurs 4–16 days after the infection, as the larva migrates through the lungs. Fever, nonproductive cough, and dyspnea may be present (Löffler’s syndrome).²⁰ Moderate to severe breathlessness, sometimes even leading to status asthmaticus has been reported. The pneumonitis resolves in about 3 weeks. Children below the age of 10 years are prone to develop intestinal obstruction. This is easily understood as the small bowel is narrow in caliber and gets blocked easily due to the high worm burden.²¹ Acute onset abdominal distension, vomiting, and constipation occur, and the child becomes sick rapidly. Masses of worms sometimes cause volvulus or intussusception, resulting in bowel gangrene and perforation.

Worms in the biliary tract cause right upper quadrant pain with tenderness and guarding. Jaundice is usually absent. If the worm returns to the duodenum, symptoms subside. However, dead worms and eggs block the duct — leading to stone formation and cholangitis with pain, jaundice, fever with chills, and leukocytosis.²² Acute pancreatitis may be caused by a worm blocking the pancreatic duct but severe pancreatitis with progression to necrosis is uncommon.¹⁹

11. Diagnosis of Ascariasis

Passage of worms in the stool or a history of vomiting the worms usually gives the diagnosis. Stool microscopy shows the ova or worm remnants in the feces. Eosinophilia >5% is rare and if it occurs, the association with *Toxocara* or *Strongyloides* spp. infection must be suspected.

12. Radiological Features of Ascariasis

On ultrasound, adult worms are seen in the GB or CBD as non-shadowing tubular echogenic structures with an anechoic central longitudinal alimentary tract of the worm giving a ‘tube within a tube or inner tube’ appearance. Occasionally they present only as curved echogenic linear strips without shadowing (strip sign). Multiple worm infestation may form a bag or cluster in the GB, while in the CBD, the overlapping worm interfaces cause a spaghetti-like appearance. In long-standing infestations, calcified linear structures may be seen suggesting dead worms. The CBD and pancreatic duct dilatation, pneumobilia, and occasionally intrahepatic abscesses may be associated.^{23,24}

On CT (Figure 4A and 4B), depending on their orientation within the biliary tree or intestine, worms are seen as round or elongated filling defects.²⁵ On T2W MRI and MRCP, the worm appears as a hypointense filling defect within the hyperintense bile and is a valuable noninvasive method of detecting ascariasis if ultrasound is equivocal.²⁶

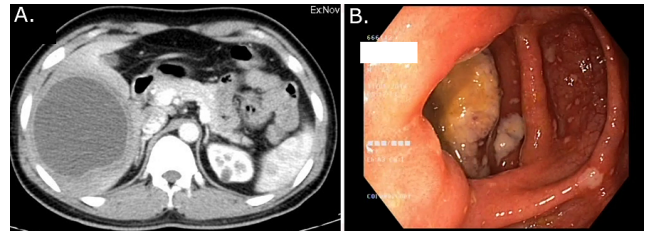


Fig. 2: A. Colonoscopy picture showing the cecal ameboma; B. Typical appearance of a liver abscess on contrast CT (water density mass with a low attenuation rim, a pigtail cat).

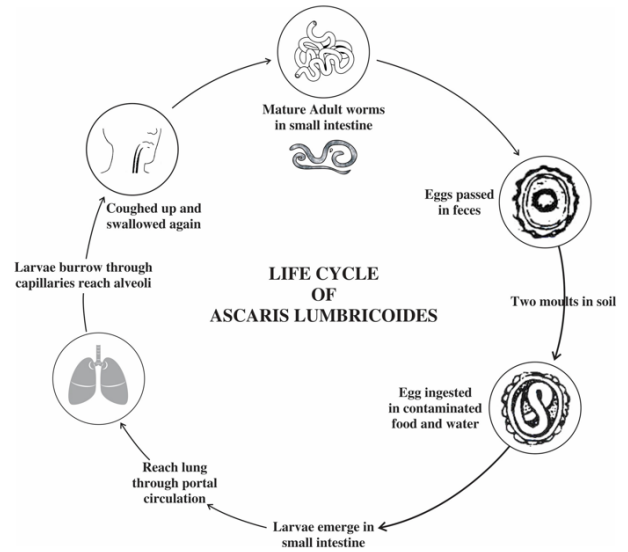


Fig. 3: The life cycle of ascaris lumbricoides

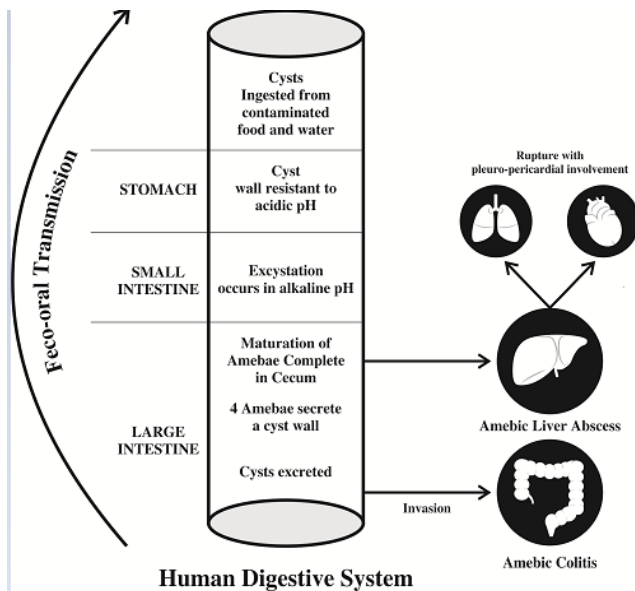


Fig. 1: The life cycle of Entamoeba histolytica.

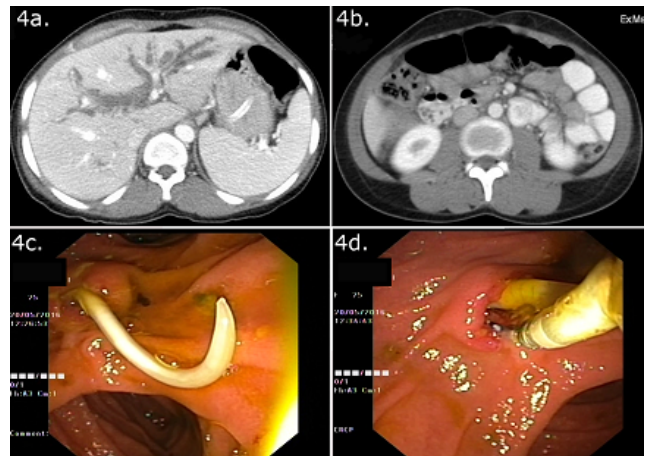


Fig. 4: Biliary ascariasis in a 24-year-old female patient. 4a. Contrast CT abdomen showing the dilatation of intrahepatic biliary ducts. 4b. Worms in the duodenum as viewed end on. c) A worm protruding out of the ampulla at ERCP. d) Removal of worms with balloon sweep and grasping forceps.

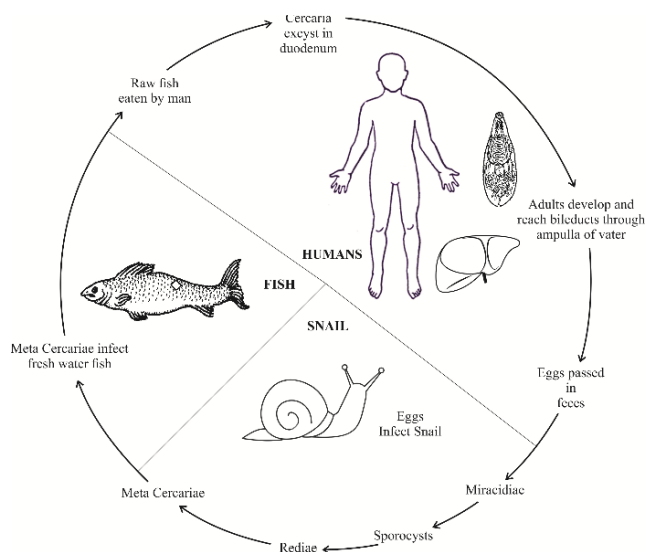


Fig. 5: The life cycle of clonorchis and opisthorchis species

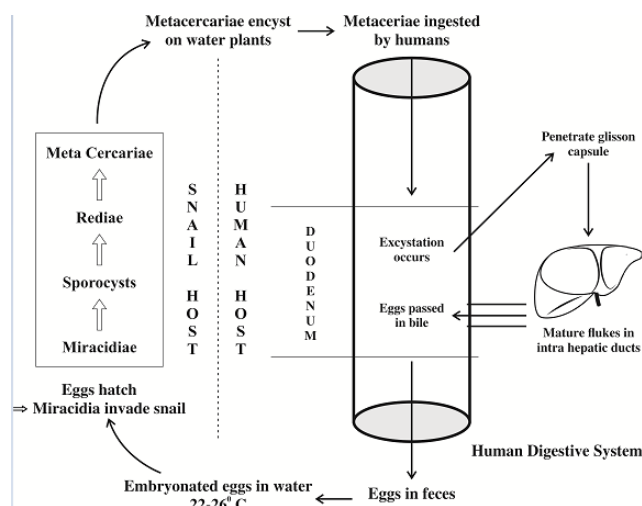


Fig. 6: The life cycle of Fasciola species.

13. Management of Ascariasis

Albendazole and mebendazole are the drugs of choice.²⁷ Their cure rate approaches 100%. Administering these drugs with a fatty meal enhances bioavailability. Levamisole and pyrantel pamoate can be used as second-line agents.

14. Role of Surgery in Ascariasis

Mohta et al.²⁸ elegantly described the management of intestinal obstruction due to ascariasis. Conservative management with analgesics, decompression via nasogastric tube, intravenous fluids can be tried in subacute cases. This is usually successful in most patients. Once the intestinal function has recovered, anthelmintic therapy is initiated. Failure of obstruction to resolve after

48 h or deterioration in the patients' condition mandates surgery.

At laparotomy, the small bowel is milked gently to displace the bolus of worms caudally into the colon; if unsuccessful, enterotomy is done and the worms are manually removed. The enterotomy is best closed using nonabsorbable sutures to prevent penetration by the remaining ascarids. Resection and primary anastomosis can be safely performed in a stable patient with bowel gangrene or perforation, whereas resection and ileostomy is an appropriate strategy in a patient with a poor general condition. All the family members of patients admitted with *Ascaris*-induced obstruction are treated with anthelmintics. This is a sound strategy in an endemic area against reinfection.

Spontaneous return of the worm occurs in more than 90% of cases of biliary ascariasis.²⁸ This is beneficial for the patient and may save the need for an interventional procedure. therefore, a trial of conservative treatment with nasogastric aspiration, antispasmodic drugs, and intravenous fluids is instituted. This is discontinued when pain and tenderness settle, and a repeat ultrasound exam shows no worm in the bile duct. If conservative treatment fails, ERCP (Figure 4C and 4D) is attempted.²⁹ The sphincter is often found to be dilated due to the passage of worms, facilitating easy cannulation. Reddy et al.³⁰ prefer grasping forceps or dormia basket to polypectomy snares as these enable extraction of worms in toto. Complete extraction is important because remnants of worms left behind can lead to calculi formation or obstruction. Anthelmintic therapy paralyzes or kills the worm and interferes with its spontaneous return. The dead worms can fragment when handled, necessitating repeat procedures to clear the duct. Anthelmintic therapy is, therefore, given after the extraction.

Surgery is required for worms in the intrahepatic ducts, with stone and stricture formation. Worms are removed through a longitudinal incision on the CBD. Cholangioscopy is helpful and can ensure the completeness of the procedure. This is followed by saline irrigation and the incision is closed over a T-tube. A T-tube cholangiogram done on day 6 or 7 can identify the residual worms. A worm stuck in the T-tube may be removed by application of the suction to the tube, while withdrawing it. Residual worms can be removed by endoscopy. If these methods fail, repeat surgery may be required.^{14,30}

14.1. *Clonorchis sinensis*, *opisthorchis viverrini* & *opisthorchis felineus*

The World Health Organization (WHO) has classified them as biological carcinogens.³¹ These parasites are responsible for conditions ranging from cholecystitis, choledocholithiasis, recurrent pyogenic cholangitis to bile duct carcinoma. They are a major public health problem

in Southeast Asia and Eastern Europe.³² Interestingly, the incidence of cholangiocarcinoma is very high in these regions (e.g. Khon Kaen province in Thailand and Qidong in China).^{33–35} These areas have certain traditional eating practices, where people consume raw/fermented fish. The uncooked/fermented fish contain encysted metacercariae, which causes infection in humans. The life cycle of cercariasis is detailed in figure 5.

14.2. Pathogenesis of cercariasis

Multiple mechanisms play a part in the pathogenesis of fluke-mediated liver injury.³⁶ The mechanical factor is that the suckers during feeding can cause biliary ulceration. The metabolic and excretory substances released from tegument and excretory openings are released into the bile. These are highly antigenic as well as toxic to the ductal epithelial cells. There are marked inflammatory infiltrations have been observed around the intrahepatic bile ducts in pathologic specimens in animals experimentally infected with *Opisthorchis*. E.g. *O. viverrini* Granulin-like substance (Ov-GRN-1) is mitogenic to fibroblast and biliary cell lines in vitro.^{37–39} The liver fluke infection triggers several inflammatory cells (macrophages, mast cells, ductal epithelial cells) to produce various cytokines. One such is nitric oxide (NO) synthesized through the inducible pathway (iNOS).³⁸ NO reacts with free radicals forming peroxynitrite (ONOO-), which in turn binds to DNA forming 8-OXOdG and 8-nitroguanine adducts inducing DNA damage.³⁹ Secondly, the antigen of *O. viverrini* induces NF- κ B expression through the TLR2 pathway. NF- κ B translocates to the nucleus and turns on several genes, leading to increased production of iNOS and COX-2. COX-2 has an excitatory effect on inflammatory cells with the production of more cytokines. Chronic inflammation with sustained DNA damage may trigger errors in DNA repair and Microsatellite Instability (MSI) setting the stage for carcinogenesis.⁴⁰

14.3. Clinical features of cercariasis

Patients present with right upper quadrant pain, fever, rashes, and loss of appetite. *O. felinus* is commonly associated with arthralgias and lymphadenopathy. Stones in the gallbladder, extrahepatic and intrahepatic ducts can cause cholecystitis, obstructive jaundice, and life-threatening cholangitis.^{41,42} In an endemic area, a patient presenting with cholangitis should be evaluated for cholangiocarcinoma.⁴³

14.4. Diagnosis of cercariasis

Microscopy of stool, bile, or duodenal aspirates can readily demonstrate the operculated eggs of *Opisthorchis* or *Clonorchis*. Any of these methods — The Kato thick smear, Stoll's dilution, or the quantitative formalin ethyl acetate

concentration technique, can be used in moderate and heavy infections.⁴⁴

15. Serology

The antigen detection in feces by ELISA technique is more sensitive than routine stool examination when parasite load is low (< 500 eggs per gram of stool).^{45,46} Antibody detection techniques typically have low specificity, but the Ov-CP-1-based ELISA with a specificity of 96%, shows some promise.⁴⁷ These tests do not distinguish between active and past infection, as antibodies can be detected up to a few months following curative treatment.

16. Polymerase Chain Reaction (PCR)

The PCR-based tests can be done on fluke DNA in feces, particularly in light infections. The Loop-mediated Isothermal Amplification assay (LAMP) technique is particularly helpful as it allows visual detection of DNA amplification by turbidity, eliminating the need for the thermocycler.^{48,49}

16.1. Radiological features of cercariasis

On ultrasound, flukes in the GB may be seen as floating or dependent non-shadowing discrete echogenic foci. In the liver, the flukes cause diffuse dilatation of the peripheral small intrahepatic ducts with characteristically no or very minimal dilatation of large /central ducts and no obvious obstructing lesion.^{50–53} The CT and MRI show similar findings as ultrasound with bile duct thickening and periductal enhancement. In very heavy infestations, MRCP may demonstrate the very thin flukes as low-intensity intraluminal filling defects.^{50–52}

16.2. Management of cercariasis

Praziquantel is the drug of choice and has a 71% cure rate. Rim et al.³⁷ report an 83–85% cure rate in Clonorchiasis when a dose of 25mg/kg is given thrice a day 5 hours apart. There may be mild side effects, such as giddiness, emesis, and abdominal pain. Inflammatory changes and debris within the gallbladder also resolve with the elimination of the parasite. For biliary lithiasis and cholangitis, the choice of procedure depends on the location of stones and/or stricture. Endoscopic approaches are tried first, with surgery reserved for failed cases. Common bile duct exploration and stone retrieval with or without T-tube insertion or hepaticojejunostomy may be done on an individual basis, followed by antiparasitic therapy. Cholangiocarcinoma is managed according to the standard oncologic guidelines.

16.3. *Fasciola hepatica* & *fasciola gigantica* (Fascioliasis)

These two large liver flukes cause fascioliasis, a chronic inflammatory disease of the liver and biliary system. This disease covers Europe, Africa, the Americas, and Oceania.⁵³ These flukes primarily infect sheep and cattle, causing considerable mortality. Humans accidentally become final hosts when they eat plants (alfalfa or watercress) or drink impure water contaminated with feces from animals harboring *Fasciola*.

16.4. The life cycle and pathogenesis of fascioliasis (Figure 6)

After excystation in the duodenum, the larvae migrate through the bowel wall transmurally into the peritoneal cavity. They penetrate the Glisson's capsule and burrow through the substance of the liver, to reach the bile ducts, where maturation takes place. Symptoms occur during this phase. A strong eosinophilic response results in parenchymal cell death, parasite death, and hemorrhage. Repair mechanisms lead to extensive nodule formation which is often mistaken for malignancy. In chronic infection, mature flukes feed on the hepatocytes and ductal cells and live for many years in the biliary system. They release eggs into the bile that is excreted in feces. Mechanical obstruction of the ducts, secretion of proline by the fluke, and its spinous processes damaging the biliary epithelium all contribute to causing bile duct proliferation, dilatation, and fibrosis.⁵⁴

16.5. Clinical features of fascioliasis

Acute infection is the result of the movement of the larva through the liver and its accompanying hemorrhage. Patients present with fever, constitutional symptoms, itching, urticaria, night sweats, and upper abdominal pain. This occurs may occur 12 -16 weeks after the ingestion of metacercaria. Anemia, hepatomegaly, and splenomegaly may be present. Of note, blood investigations show leukocytosis with eosinophilia in almost all patients.

Chronic infection is usually asymptomatic. Mild jaundice may be present. Serum levels of Alkaline phosphatase and Gamma-glutamyl transpeptidase are raised indicating cholestasis. Ulcers in the bile duct due to adult flukes can cause haemobilia. Choledocholithiasis, cholangitis, pancreatitis, biliary cirrhosis, and hepatic fibrosis have all been reported.⁵⁵

16.6. Diagnosis of fascioliasis

Fascioliasis is usually diagnosed by observation of the eggs during microscopy of stool, bile, and duodenal contents. Egg detection has often been unsatisfactory. This may be because they cannot produce eggs (*Fasciola* larva may or

may not attain maturity in the human tissues as humans are not their natural hosts) or the eggs get encapsulated in granulomas. This results in infection with low egg shedding and necessitates sedimentation techniques to increase egg detection. Also, differentiation of eggs between various *Fasciola* species can be difficult.⁵⁶

17. Serology

Immunodiagnosis seems to be superior to microscopy — the antigen and the antibody can be detected much before egg shedding begins.⁵⁷ The more purified the antigen, the better is the antibody detection rate. The cathepsin CL1 (a cysteine protease) and the *Fasciola* 27kDa antigen have been used in several studies, with the latter showing excellent results (98.2% sensitivity and 100% specificity)⁵⁸ The antigen detection uses monoclonal or polyclonal antibodies against specific antigens (Circulating Antigen, Excretory Secretory antigen, or ES) in urine or stool samples using a sandwich ELISA. The study by Demerdash et al. for detection of ES antigen resulted in a diagnostic efficacy of 94% and 97% in serum and stool samples respectively.⁵⁹ These tests do not differentiate between various *Fasciola* species and do not quantitatively measure the parasite burden.⁶⁰

17.1. Radiological features of fascioliasis

Hepatic fascioliasis may present on ultrasound as multiple confluent, ill-defined hypoechoic nodules which are usually subcapsular. Minimal gallbladder wall thickening and dilatation of intrahepatic biliary radicles are often seen due to associated cholangitis. If seen, the parasite may resemble curvilinear or leaf-like echoes ranging from 5-25mm.⁶¹ On CECT, the ill-defined subcapsular nodules are seen to follow the portal triads and converge towards the hilum of the liver with hypodense linear inflammatory tracts seen in the subcapsular regions. MRI may confirm the segmental geographic areas of hepatic inflammation, subcapsular-periportal hypointense tracts, and micro abscesses with rim enhancement which on diffusion-weighted images may show restricted diffusion. Post-inflammatory fibrosis may be seen as hypointensity on T1 and T2 weighted images.

17.2. Management of fascioliasis

A single dose of Triclabendazole 10 mg/kg eliminates both immature and adult parasites with 90% cure rates and is recommended as the treatment of choice. Bithionol has been used as an alternative, with good results.^{62,63}

17.3. Role of surgery in fascioliasis

Most patients present or are referred to the surgeon with cholecystitis or obstructive jaundice and choledocholithiasis. Alban et al, even in an endemic area, have recorded only a 1.2% incidence of adult worm in

the gall bladder in their series of 162 cholecystectomies.⁶⁴ The anti-parasitic medications may kill the flukes and this, in turn, may obstruct the ampulla, causing biliary obstruction and cholangitis. Urgent Endoscopic retrograde Cholangiopancreatography (ERCP) is necessary to remove the dead worms and ensure good drainage of CBD. If ERCP fails, Percutaneous transhepatic cholangiography (PTC) may be necessary. When the pancreatic duct is involved, Parsak et al. recommend drainage followed by Triclabendazole with an antispasmodic to prevent sphincter of Oddi spasm.⁶⁵ It was reported that life-threatening pancreatitis seems to be uncommon.⁶⁶

18. Conclusion

Given the increasing incidence of international travel, parasitic diseases have assumed an even greater significance. These diseases may present a confusing picture to the unwary physician in a non-endemic area, leading to a departure from the optimal line of management, which in turn leads to increased health care costs and patient dissatisfaction. The physician's suspicion of a parasitic disease may be aroused only if he possesses a good working knowledge of these conditions. Early diagnosis and treatment at the individual level with health education and awareness at the community level plays a vital role in the control of parasitic diseases.

19. Source of Funding

None.

20. Conflict of Interest


None.

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