



## Parasitic infestations requiring surgical interventions

Afua A.J. Hesse, MD,<sup>a</sup> Abdellatif Nouri, MD,<sup>b</sup> Hussam S. Hassan, MD,<sup>c</sup>  
 Amel A. Hashish, MD, CT, MHPE<sup>c</sup>

From the <sup>a</sup>Pediatric Surgery Unit, Department of Surgery, University of Ghana Medical School, Accra, Ghana;

<sup>b</sup>Department of Pediatric Surgery, Fattouma Bourguiba Hospital, Monastir, Tunisia; and

<sup>c</sup>Faculty of Medicine, Tanta University, Tanta University Hospital, Tanta, Egypt.

### KEYWORDS

Parasites;  
 Surgery;  
 Africa;  
 Children;  
 Amoebiasis;  
 Ascariasis;  
 Dracontiasis;  
 Schistosomiasis;  
 Hydatid;  
 Myiasis

Parasitic infestation is common in developing countries especially in Africa. Children are often more vulnerable to these infections. Many health problems result from these infestations, including malnutrition, iron-deficiency anemia, surgical morbidities, and even impaired cognitive function and educational achievement. Surgical intervention may be needed to treat serious complications caused by some of these parasites. Amoebic colitis and liver abscess caused by protozoan infections; intestinal obstruction, biliary infestation with cholangitis and liver abscess, and pancreatitis caused by *Ascaris lumbricoides*; biliary obstruction caused by Fasciola; hepatic and pulmonary hydatid cysts caused by *Echinococcus granulosus* and multilocularis are examples. Expenditure of medical care of affected children may cause a great burden on many African governments, which are already suffering from economic instability. The clinical presentation, investigation, and management of some parasitic infestations of surgical relevance in African children are discussed in this article.

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Parasitic infestations can occur in children of all ages. The relatively poorly developed immune system in children increases their susceptibility to the pathophysiological disturbances associated with these infestations. Several parasitic diseases occur more frequently in developing countries, but their prevalence has not been well studied. African children are more vulnerable because of many complicating socioeconomic, environmental, and sanitary-hygienic conditions. Parasitic infections may interfere with the nutritional status, growth, and development of the affected children. Anthelmintic prophylaxis to prevent morbidity from multiple helminthes infestations is recommended by the World Health Organization.<sup>1</sup>

There are three main classes of parasites that can cause disease in humans: Protozoa, such as amoeba (*Entamoeba*),

the flagellates (*Giardia* or *Leishmania*), the ciliates (*Balanitidium*), or sporozoa (*Plasmodium*); Helminthes, such as flatworms (flukes or tapeworms) or roundworms (*Ascaris lumbricoides* [*A lumbricoides*]); and Ectoparasites, such as ticks, fleas, lice, and mites.

This article highlights the prevalence, the etiology, pathophysiology, and current management options of the parasitic infestations of surgical interest in children, such as ascariasis, dracontiasis, amoebiasis, schistosomiasis, hydatid disease, and myiasis.

### Protozoa

#### Amoebiasis (*Entamoeba histolytica* infection)

Amoebiasis is a parasitic disease caused by the protozoan parasite *Entamoeba histolytica* (*E histolytica*) that is commonly transmitted via the fecal-oral route. Amoebiasis may

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**Address reprint requests and correspondence:** Amel A. Hashish, MD, CT, MHPE, Faculty of Medicine, Tanta University, Tanta University Hospital, Elgish Street, Tanta, 3111, Egypt.  
 E-mail: amelhashish@gmail.com.

affect any age group and has no gender preference. It is considered the third leading parasitic cause of death worldwide, surpassed only by malaria and Schistosomiasis. On a global basis, amoebiasis affects approximately 50 million persons each year, resulting in nearly 100,000 deaths mostly from liver abscesses or other complications.<sup>2</sup>

The parasite has 2 forms: a motile form, called the trophozoite, and a cyst form, which is responsible for person-to-person transmission of infection. Humans are the only reservoir of *E histolytica*. Cysts passed in the feces, can survive in moist environmental conditions for weeks to months. Upon ingestion of contaminated food or water, the cysts travel to the small intestine, where the trophozoites are released. In 90% of patients, the trophozoites reencyst and produce asymptomatic infection, which usually spontaneously resolves within 12 months. In the remaining 10% of patients who are infected, the parasite causes symptomatic amoebiasis.<sup>2</sup>

### Intestinal amoebiasis

In symptomatic cases, the trophozoite of *E histolytica* inhabits the large intestine to produce amoebic colitis and amoebic dysentery giving symptoms that can range from mild diarrhea to dysentery with mucus and blood, which comes from amoebae invading the lining of the intestine. The passage of large volumes of malodorous stools with slough from the mucosa in a child with preexisting malnutrition suggests amoebic colitis. Amoebic colitis is gradual in onset, with symptoms presenting over 1-2 weeks, distinguishing it from bacterial dysentery.<sup>3</sup> Invasion of the colonic mucosa leads to dissemination of the organism to extracolonic sites, predominantly the liver, leading to amoebic liver abscess, which is considered the most frequent extraintestinal manifestation of *E histolytica* infection.

### Hepatic amoebiasis

Amoebic liver abscess is caused by the parasite ascending the portal venous system. Signs and symptoms of amoebic liver abscess are initially nonspecific. Fever and abdominal pain are the most common element in the history and present in 90%-93% of patients. Pain is most frequently located in the right upper quadrant (54%-67%) and may radiate to the right shoulder or scapular area and increases with coughing, walking, and deep breathing. It is usually constant, dull, and aching. Elevation of the diaphragm and atelectasis or effusion, rigor, nausea and vomiting, and diarrhea may also occur.<sup>4,5</sup> Complications of hepatic amoebiasis includes subdiaphragmatic abscess, perforation of diaphragm to pericardium and pleural cavity, and perforation to abdominal cavity giving amoebic peritonitis.

### Pulmonary amoebiasis

Pulmonary amoebiasis can occur by hematogenous spread or by perforation of a hepatic abscess through the diaphragm into the pleural cavity and lung. It can cause lung abscess, pulmonopleural fistula, empyema, and bronchopleural fistula.

Amoebiasis can also cause amoebic brain abscess and amoebic meningoencephalitis. Cutaneous amoebiasis can occur in the skin of the perianal region or at the site of drainage of liver abscess.

### Diagnosis

The diagnosis of amoebiasis can be very difficult. Stool examination is insensitive unless a fresh "warm" stool specimen is examined when typically amoeba, which has ingested red blood cells, is seen. Leukocytosis without eosinophilia is observed in 80% of cases. Elevated transaminase levels, mild elevation of serum bilirubin level, and reduced serum albumin level may also be present. Elevated alkaline phosphatase is present in 80% of patients. The serologic reactions that reveal serum antibodies specific to the *E histolytica* are very useful in the diagnosis of invasive amoebiasis. The most frequently used tests are enzyme-linked immunosorbent assay, indirect hemagglutination, and indirect immunofluorescence. Serum antibodies against amoebae are present in 70%-90% of individuals. Antiamoebic antibodies are present in most of the patients who have symptomatic liver abscess for longer than a week. In cases of amoebic liver abscess, chest radiography may show an elevated right hemidiaphragm and right-sided pleural effusion. Ultrasonography is preferred for the evaluation of amoebic liver abscess. Rectosigmoidoscopic examination and barium examination have little value in establishing the diagnosis.

### Treatment

Metronidazole is considered the mainstay of therapy for invasive amoebiasis. Broad-spectrum antibiotics are added to treat cases of bacterial superinfection and amoebic colitis.

Most uncomplicated amoebic liver abscesses can be treated successfully with amoebicidal drug therapy alone. Metronidazole, 750 mg 3 times a day orally for 10 days, is first used to eradicate the invasive trophozoite forms in the liver. After completion of treatment with tissue amoebicides, luminal amoebicides are administered for eradication of the asymptomatic colonization state.<sup>6</sup>

Surgical intervention is required for an acute abdomen due to perforated amoebic colitis. In cases of liver abscess, surgery is suggested only on failure of medical treatment. Imaging-guided needle aspiration and catheter drainage are the procedures of choice. Open surgical drainage is considered when the abscess is inaccessible to needle drainage or a response to therapy has not occurred in 5-7 days.<sup>7,8</sup>

### Helminthes

The prevalence of helminthic infestation in African children varies depending on the age, geographical area, and ethnic group studied. Prevalence of infestations is as high as 98% in some villages in Cameroon.<sup>9</sup> In a survey of 1820 children in Sierra Leone, ascariasis was found in more than 33.3%.<sup>10</sup> A higher prevalence in children was reported in Nigeria

(88.5%),<sup>11</sup> South Africa (76%),<sup>12</sup> and Lake Langano, Ethiopia (60.2%).<sup>13</sup>

### Ascariasis

A study in South Africa confirmed that human infestation with *A lumbricoides* was responsible for 20% of acute admissions annually to the pediatric surgical wards of the Red Cross Children's Hospital in Cape Town in the 1980s.<sup>14</sup> Of these (66%) were intestinal, 30% were hepatobiliary, and 4% were pancreatic.

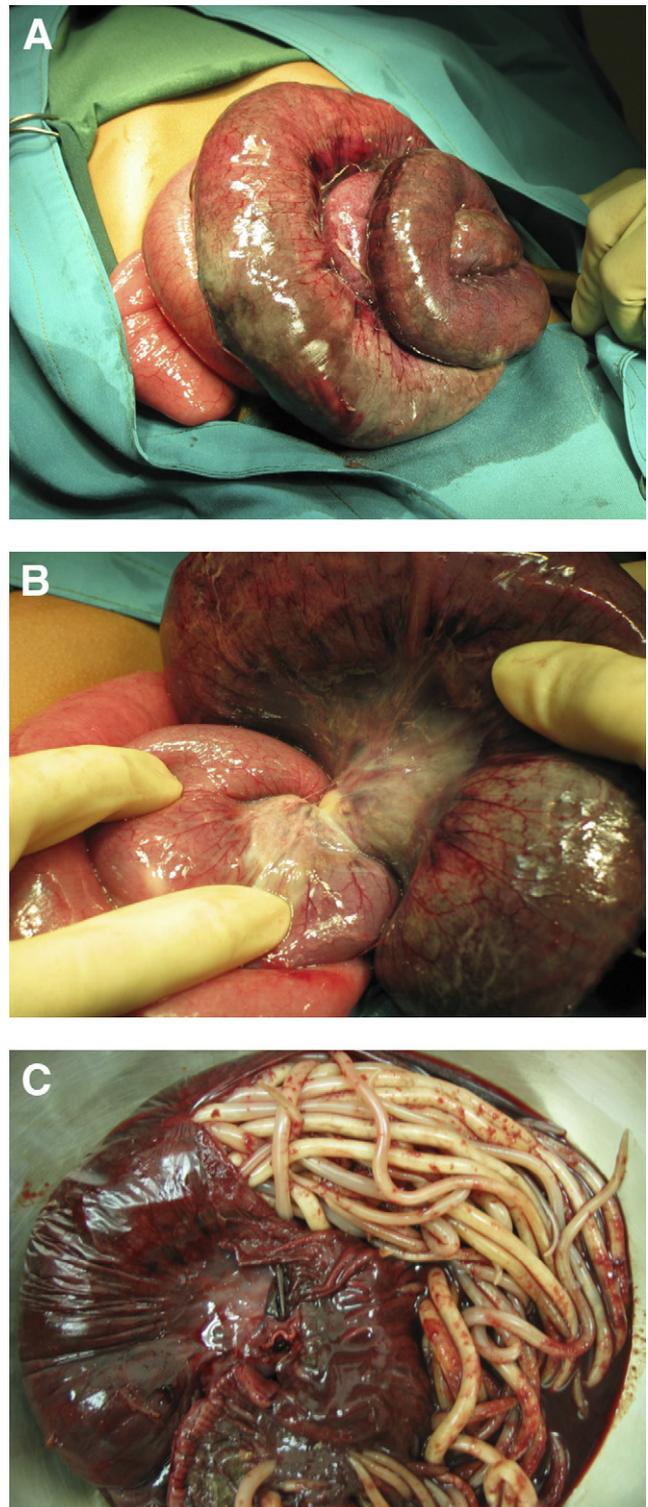
In another study of 145 cases of surgical complications due to ascariasis, intestinal obstruction occurred in 74%, appendiceal perforation in 7%, and extra intestinal migration to the biliary tree and the peritoneal cavity in 19% of cases.<sup>15</sup> Aydin<sup>16</sup> in Turkey reported a 3.5% incidence of incidental finding of *A lumbricoides* and *Enterobius vermicularis* (*E vermicularis*) infection in removed appendices.

Symptomatic cases may manifest as pyrexia of moderate degree, vomiting, malnourishment, growth retardation, pneumonitis (Loeffler syndrome), and abdominal pain. Severe or radiating abdominal pain may suggest biliary colic, cholangitis, pancreatitis, or appendicitis. Presence of ill-defined masses with intestinal obstruction may occur in heavy infections.

Identifying *Ascaris* eggs in a stool sample is diagnostic. Eosinophilia is present in the early phases of infestation, but is not diagnostic. In cases of heavy infestation, worms appear radiolucent in the plain abdominal x-ray. In cases of intestinal obstruction, diagnosis is based on history of passage of worms per mouth or rectum and on plain abdominal x-ray and ultrasonography findings.

Anthelmintic medications, such as mebendazole and albendazole, can kill the adult worms.<sup>17</sup> Conservative management of partial intestinal obstruction and biliary ascariasis is usually effective. The patient is maintained nil by mouth with intravenous fluids to hydrate the patient. Piperazine salt per nasogastric tube and glycerine plus liquid paraffin emulsion enemas can be also added.<sup>18</sup> Antispasmodics (hyoscine butyl bromide) are given for colic, and anthelmintic drugs are administered after the attack has subsided. Surgical exploration is required in cases of unsatisfactory response to conservative therapy, presence of multiple air fluid levels on abdominal radiographs, and abdominal guarding or rigidity. Milking of worms to the large bowel, resection of gangrenous bowel, ileostomy, and enterotomy with removal of a worm bolus are the most common surgical procedures used to manage bowel obstruction due to ascariasis. A worm bolus in the intestine may undergo volvulus with strangulation and necrosis of the loop of intestine requiring emergent laparotomy and bowel resection (Figure 1).

In cases of biliary obstruction, endoscopic retrograde pancreatography and endoscopic removal of common bile duct worms can be successful in 55% of cases. Open surgery is indicated in patients with worsening intestinal obstruction or persistent biliary impaction and complicated



**Figure 1** (A) An *Ascaris* worm bolus with impacted adult worms in a 9-year-old boy, which has undergone volvulus. (B) Visible is the base of the twisted loop of bowel. (C) The resected specimen demonstrates the necrotic loop of bowel with worm impaction.

hepatic disease with abscess formation.<sup>14</sup> All visible worms are extracted, abscesses are drained, and a T-tube left in situ. Oral anthelmintic are given to clear the bowel lumen of

worms. The T-tube can be removed after a cholangiogram shows complete clearance of worms from the bile ducts.

## Schistosomiasis

Schistosomiasis is a chronic parasitic disease caused by blood flukes (trematode worms) of the genus *Schistosoma*. The important species being *Schistosoma hematobium* (*S. hematobium*), *Schistosoma mansoni*, and *Schistosoma japonicum*. *S. hematobium* was first identified by Theodore Bilharz, a German pathologist working in Cairo, in 1851. Schistosomiasis is the third most devastating tropical disease in the world, being a major source of morbidity and mortality for developing countries.<sup>19</sup> It is endemic in Southern Africa, sub-Saharan Africa, Lake Malawi, and Nile River valley in Egypt.<sup>20,21</sup>

There are an estimated 207 million people infected with one of the major schistosomes with more than 90% of the cases occurring in sub-Saharan Africa.<sup>21,22</sup> People at Africa, are at risk of infection because of agricultural, domestic, and recreational activities, which expose them to infested water.

Fewer than 5% of the African patients receive treatment.<sup>23</sup> Almost 300,000 people die annually from schistosomiasis there.<sup>24</sup>

Infestation occurs when the larval forms of the parasite released by fresh water snails penetrate the skin during contact with infested water. Larvae develop into adult schistosomes in the human body. Adult worms live in blood vessels where the females release ova. Some ova pass out of the body in the feces or urine to continue the parasite life cycle. Other ova, trapped in body tissues, causes an immune reaction and progressive pathological changes.<sup>25</sup> Deposition of ova in the portal tracts of the liver causes progressive fibrosis with the development of portal hypertension, leading to two complications of surgical concern: splenomegaly and esophageal varices.<sup>26</sup>

Symptoms of schistosomiasis are caused by the body's reaction to the worms' eggs, not by the worms themselves. Approximately 60% of the affected population has symptoms, including organ-specific complaints and problems related to chronic anemia and malnutrition from the infection.<sup>27</sup> Intestinal schistosomiasis can result in abdominal pain, bleeding per rectum, hematemesis, and hepatosplenomegaly. Infertility, ascites and hepatic coma can occur in advanced cases. Urogenital schistosomiasis is characterized by hematuria. Bladder cancer is one of its complications. Other complications include gastrointestinal bleeding, severe anemia, malnutrition, portal hypertension, pulmonary hypertension with cor pulmonale, and central nervous system impairment. Urogenital schistosomiasis is considered to be a risk factor for HIV infection, especially in women.<sup>19</sup> Schistosomiasis is diagnosed through the detection of parasite eggs in stool or urine specimens. For people from nonendemic or low-transmission areas, serological and immunological techniques may be useful in the detection of infestation. Calcification of the bladder wall and the lower end of the ureters are seen on plain x-ray of the pelvis in

advanced cases. Abdominal ultrasonography scanning is useful for early identification of periportal hepatic fibrosis and assessment of hepatosplenomegaly. Cystoscopy can detect sandy patches, granulomatous ulcers, tubercles, or bilharzioma of the urinary bladder. Sigmoidoscopy, esophagogastroscope, and liver biopsy may be indicated in intestinal schistosomiasis.

Praziquantel is the only available treatment against all forms of schistosomiasis. It is effective, safe, and low cost. It may be given as a single oral dose of 50 or 20 mg/kg 3 times at 4-hour intervals.

Surgical treatment may be necessary for cases of fibrous contracture or carcinoma of the bladder, stenosis of the ureter, hydronephrosis, portal hypertension, and stenosis of the bowel. Patients with nonbleeding varices may require schedule of sclerotherapy and beta blockers. For bleeding varices, if repeated sclerotherapy and drug treatment fails to control bleeding attacks, direct surgical intervention becomes necessary. Surgical options are either portosystemic shunt operations or decongestion operations.<sup>28</sup>

## Echinococcosis (hydatid disease)

Hydatid disease is a parasitic infestation caused by the larval form of short tapeworms of the genus *Echinococcus*. *Echinococcus granulosus* and *Echinococcus multilocularis* are responsible for most of the overt cases of the disease, although other species are known to exist. It is a cosmopolitan zoonosis, and it is endemic in many African areas. Tunisia is the most endemic area among the countries of the Mediterranean,<sup>29-31</sup> where one in every 10,000 people under the age of 20 is affected. In the rest of the Maghreb countries, the annual incidence of hydatidosis is similar to Tunisia.<sup>30</sup> Eastern Africa is the second most endemic area with an incidence that can reach 2/1000 in Kenya (especially in the Turkana District) and in Ethiopia.<sup>32,33</sup> Screening of 3443 people in southern Sudan revealed a prevalence of human hydatid disease of about 0.5%, with a male:female ratio of 1:1.7.<sup>34</sup>

The worm lives attached to the villi of the small intestine of canines like dogs. When it is evacuated, the gravid proglottis bursts releasing the eggs, which are ingested by the intermediate hosts that can be sheep or man. After ingestion, the embryo is liberated in the small intestine and can penetrate the intestinal mucosa to reach the portal circulation. It may invade the liver and form a hydatid cyst in about 75% of cases. Embryo can pass the portal circulation to enter the general circulation, where it can form a hydatid cyst in any other organ of the body, such as the lung (5%-15%), bones, brain, heart, and kidneys (10%-20%).

The age of presentation ranges from 18 months to 15 years (average 8 years). Tantawy<sup>35</sup> reported a predominance in boys over girls (62.5% vs 37.5%). This is thought to be mostly because of behavioral differences between the sexes, with more exposure in boys. In a Tunisian series of 408 patients, 51% had pulmonary hydatid cyst, 31% had hepatic hydatid cyst, and 16% had synchronous hepatic and pulmo-

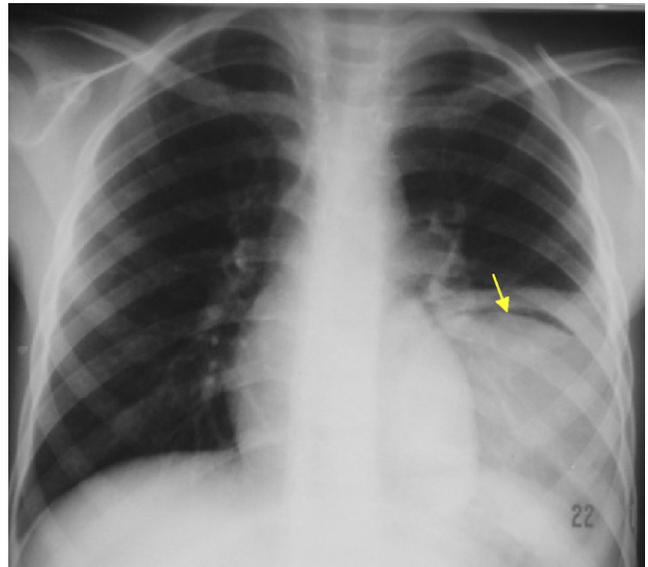
nary hydatid cysts. Other locations include the peritoneum and spleen, and also occurred in kidney, muscle, brain, heart, and pancreas in 2% of patients.<sup>30</sup>

Hepatic hydatid cysts are often asymptomatic until they reach a large size. The patient may then remain asymptomatic for years depending upon the location, the size, and the number of the developing cyst/s. Pulmonary hydatid cyst is often symptomatic, and patients presented with cough (85%), chest pain (40%), hemoptysis (15%), dyspnea (13%), hydatid vomits, (10%) and fever (8%). In 10% of the patients, right upper abdominal pain, jaundice, fever, hepatomegaly, abdominal mass, anaphylactic reaction, and itching may occur. Skin rashes may develop, as well as a cough and chest pain. Brain cysts may produce seizures or loss of strength or sensation.

For cystic echinococcosis, imaging is the main method for diagnosis. Chest x-ray can confirm the pulmonary hydatid cyst by the presence of one or more of the pathognomonic signs; it can also determine whether the cyst is intact (Figure 2), fissured, or ruptured as shown in some characteristic radiological signs, such as the meniscus sign (Figure 3), which can be thin or large and corresponds to a fissured hydatid cyst, the floating membrane or water lily sign (Figure 4) or collapsed membrane (Figure 5) corresponds to a ruptured hydatid cyst, and the “snake sign” (Figure 6) corresponds to a dry retention of the membrane when all the fluid is expelled. Ultrasonography is considered the imaging technique of choice, especially for hepatic cases (Figure 7). False-positives results occur in up to 10% of cases because of the presence of nonechinococcal serous cysts or abscesses.<sup>36</sup> Ultrasonography is also helpful in the follow-up of treated patients. In addition to ultrasonography, both magnetic resonance imaging and computed tomography (CT) (Figure 8) scans are often used. Magnetic resonance imaging is preferred to CT scans because it gives better visualization of liquid areas within the tissue. CT is the best investigation for detecting extrahepatic disease and volumetric follow-up as-



**Figure 2** Bilateral lower lobe homogenous, round, well-delineated opacity due to intact hydatid cysts in an 8-year-old child (arrows). (Color version of figure is available online.)



**Figure 3** Meniscus sign due to a fissured hydatid cyst (arrow).

essment. Serology can be helpful, but is not sensitive or specific for diagnosis. Serology tests such as indirect hemagglutination, enzyme-linked immunosorbent assay, or latex agglutination are used to verify the imaging results. Aspiration cytology is helpful in the detection of pulmonary, renal, and other nonhepatic lesions for which imaging techniques and serology do not provide appropriate diagnostic support.

Surgery remains the mainstay in the treatment of hepatic hydatid disease. Cystectomy and pericystectomy offer a good chance for cure and should be undertaken wherever possible. Surgical removal of the cysts should be combined

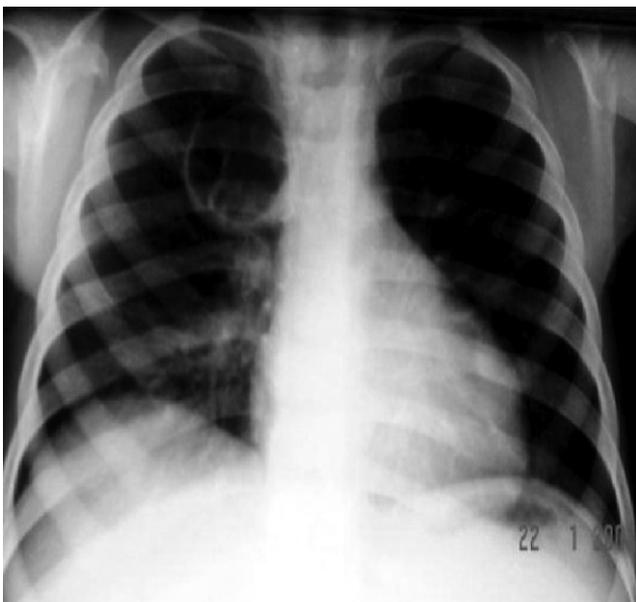


**Figure 4** Water lily sign; the endocyst membrane is seen floating on top of the remaining fluid.

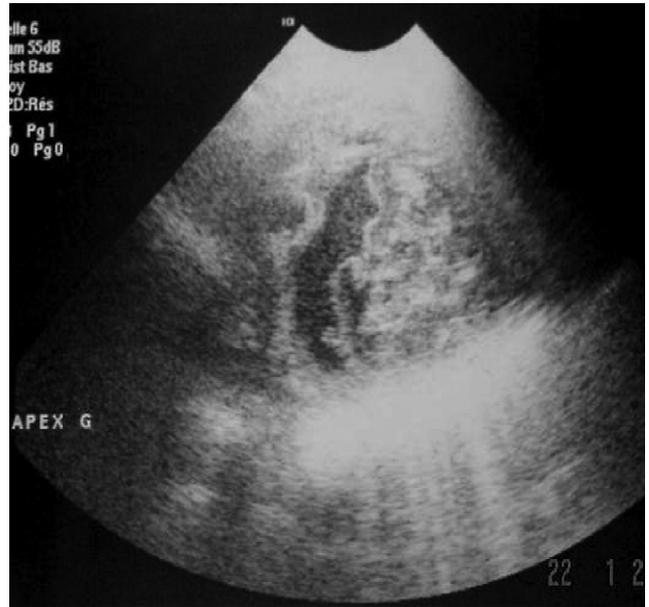


**Figure 5** Air containing hydatid cyst with an opacity at the bottom of the cyst corresponding to the collapsed membrane.

with chemotherapy using albendazole (12-15 mg/kg/day for 28 days)<sup>37</sup> or mebendazole (200 mg/kg/day in 3 divided doses for 16 weeks). Preoperative chemotherapy with albendazole or mebendazole is indicated for reducing the risk of secondary echinococcosis after operation, and it should begin at least 4 days before surgery and be continued for at least 1-3 months. Treatment with albendazole or mebendazole results in cyst disappearance in 30% of cases; cyst degeneration in 30%-50% results in significant reduction in cyst size in 20%-40% of patients and no morphologic change in 10% of cases. Ben Brahim et al<sup>38</sup> presented a success rate of 96% with multiple peritoneal cysts and



**Figure 6** The snake or serpent sign of detached membranes within the endocyst.



**Figure 7** Double-line sign.

58% of multiple pulmonary cysts with albendazole therapy alone.

Several procedures have been described for the treatment of hepatic echinococcal cysts, ranging from simple puncture of the cyst to liver resection and transplantation. The most commonly used technique is total or partial cystopericystectomy. Open endocystectomy with or without omentoplasty or simple tube drainage of infected or communicating cysts are other options. For pulmonary cysts, the Barrett technique, which entails thoracotomy and assisting intact endocystectomy without preliminary aspiration by hand ventilation, could be done. Pericystectomy and lobectomy are other options.

Surgery is not indicated for cysts in multiple organs or tissues or in risky locations. In such situations, chemotherapy and/or PAIR (puncture-aspiration-injection-reaspiration) have become alternative options of treatment.



**Figure 8** Multiple hydatid cysts in the liver.

PAIR is a minimally invasive procedure that involves 3 steps: puncture and needle aspiration of the cyst, injection of a scolicedal solution for 20-30 minutes, and cyst reaspiration and final irrigation. Effective protoscolicedals with a relatively low risk of toxicity are 70%-95% ethanol and 15%-20% hypertonic saline solution. A direct communication between the hydatid cyst and the biliary tree may contraindicate the use of protoscolicedal solutions, which can cause chemical cholangitis leading to sclerosing cholangitis. Formalin should not be used for this reason. Patients who undergo PAIR typically take albendazole or mebendazole for 7 days before the procedure and 28 days after the procedure. PAIR is indicated in patients refusing surgery, infected cysts not communicating with the biliary system; inoperable patients, multiple cysts, relapse after surgery, and failure to respond to chemotherapy.

It has been reported that PAIR with chemotherapy is more effective than surgery in terms of disease recurrence, morbidity, and mortality.<sup>39</sup> The laparoscopic approach for hydatid liver cyst is also practiced with success. Its efficacy is still controversial even though studies have suggested that it has clear benefits. Recently, laparoscopic-assisted drainage of hydatid cysts has been performed with good results, but its advantage over PAIR and other procedures needs to be evaluated by long-term studies.<sup>39</sup>

For pulmonary cysts, thoracotomy has been used safely for long time. It allows resection of the cyst and closing the bronchocystic fistulas. Thoracoscopy is less invasive but is more difficult in dealing with fistulas. Capitonage is recommended to prevent a persistent fistula. A pulmonary cyst can rarely be recovered by rupture. This happens when all the membrane is expelled and the bronchocystic fistula is closed.

### Enterobius vermicularis

Humans are the only hosts of *E. vermicularis*, which is underestimated because it seldom produces any symptoms or complications. Infection with *E. vermicularis* has a prevalence that can reach 50% among general population; most of them are children under 18 years. In Nepal, Sah et al<sup>40</sup> identified *E. vermicularis* in 1.62% of 634 surgically removed appendices; none of them were found to have invaded the mucosa or caused mucosal inflammation. In Egypt, Helmy et al<sup>41</sup> found parasitic infections to be greatly implicated in the pathophysiology of acute appendicitis. They found *E. vermicularis* in 10% of removed appendices. A similar finding was reported by Yildirim et al<sup>42</sup> who identified *E. vermicularis* in 3.8% of 104 surgically removed appendices. They concluded that the presence of parasites in the appendix may produce symptoms resembling acute appendicitis, but are not the cause of mucosal invasion in these patients.

### Dracontiasis (guinea worm disease)

Dracontiasis is caused by a nematode called *Dracunculus medinensis*. It is a cause of disability in many rural parts of Africa. The disease is considered the oldest human parasite in Africa. It has been mentioned in the Egyptian medical Ebers Papyrus dating from 1550 BC. The disease is rare in children <3 years of age, and both sexes are equally affected. In Nigeria, Edungbola<sup>43</sup> reported an infection rate of 38% among 190 children <10 years of age. In 1986, an estimated 3.5 million cases of guinea worm, in addition to another 120 million persons at risk for the disease in 20 endemic nations in Asia and Africa, were reported.<sup>44,45</sup> In 2006, approximately 98% of dracontiasis worldwide were reported from Ghana and Sudan.<sup>46</sup> In 2007, many African countries, such as Benin, Faso, Chad, Cote d'Ivoire, Kenya, Togo, Uganda and Senegal, were certified as guinea worm free by the World Health Organization. However, Sudan, Mali, Ghana, and Ethiopia still had endemic transmission.<sup>47</sup>

*Dracunculus medinensis* is a long, thin nematode (male, 1-3 cm and female, 60-90 cm). The parasite enters a host only by ingesting stagnant water of ponds or wells contaminated with copepods infested with guinea worm larvae. Once inside the body, the female, which contains larvae, burrows into the deeper connective tissue of adjacent long bones or joints of the extremities. Approximately 1 year after the infestation, the worm creates a blister in the human host's skin, usually in the lower part of the leg around the ankles. Within 72 hours, the blister ruptures, exposing one end of the emergent worm. This blister causes a very painful burning sensation as the worm emerges. A few hours before the development of the local lesion, the symptoms are exacerbated and may include erythema, urticarial rash, severe pruritus, nausea and vomiting, diarrhea, dyspnea, giddiness, and syncope. Patients often immerse the affected limb in water to relieve the burning sensation. Once the blister is submerged in water, the adult female worm releases hundreds of thousands of guinea worm larvae, contaminating the water supply and repeating the life cycle of the worm. Secondary bacterial superinfection, septicemia, tetanus, severe arthritis, and ankylosis may be additional clinical manifestations of dracontiasis.

The main treatment is extraction of the worm by cautious winding around a piece of gauze or a stick and gentle traction applied daily until it is removed. Worm extraction can take hours to months. Worm extraction should be preceded by submersion of the affected area in a bucket of water; this causes the worm to discharge many of its larvae, making it less infectious. The water should be discarded far away from any water source. Submersion helps in relief of the burning sensation and makes extraction of the worm easier. Wet compresses are applied to the ulcer daily until the discharge from the worm ceases. Application of a topical antibiotic to the lesion prevents secondary bacterial infection and complications. The use of niridazole (25 mg/kg in 2 divided doses given orally daily for 10 days),

thiabendazole (50 mg/kg daily for 3 days), or metronidazole (10 mg/kg per dose at 8-h doses daily for 10-20 days) can help to lessen the intense tissue reaction and make extraction of the worm easier.

## Others

Other zoonotic parasites have been implicated in biliary disease, such as *Clonorchis sinensis*, *Opisthorchis viverrini*, *Opisthorchis felinus*, *Dicrocoelium dendriticum*, *Fasciola hepatica*, and *Fasciola gigantica*. The diagnoses can be made through direct microscopic examination of the eggs in the duodenum, bile, and stool. In some cases, radiologic imaging may show intrahepatic ductal dilatation. In most cases, oral treatment is inexpensive and has been found to be effective against most of these parasites.<sup>48</sup>

## Ectoparasites

### Myiasis

Myiasis is an infestation of the skin by fly larvae of a variety of fly species. Worldwide, the most common flies that cause the human infestation are *Dermatobia hominis* (human bot-fly) and *Cordylobia anthropophaga* (tumbu fly). Flies can transmit infection to people either by deposition of their larvae on or near patient's wound. Some fly larvae can enter skin through bare feet when children walk through soil containing fly eggs. Other flies attach their eggs to mosquitoes and larvae then enter through bites. Beside the cutaneous myiasis, there is nasopharyngeal myiasis, which affects the nose, sinuses, and pharynx; ophthalmomyiasis affects the orbits, periorbital, and the eyes giving severe eye irritation, redness, foreign body sensation, pain, lacrimation, and swelling of the eyelids, tissue, and urogenital and intestinal myiasis. Patients usually complain of painful, pruritic, and tender boil-like lesions usually on exposed areas of the body. Sometimes patients have the sense of something moving under the skin. Patients also complain of fever, swollen glands, or extremities. Ultrasonography or CT scan is very useful in establishing the diagnosis and in determining the size of the larvae.

Surgical removal with local anesthesia is usually the preferred approach. The skin lesion is excised followed by primary wound closure. Another surgical option is to perform a 4- to 5-mm punch excision of the overlying punctum and surrounding skin and tissue. Larvae can then be extracted carefully with toothed forceps. All precaution should be taken to avoid lacerating the larva because retained larval parts may precipitate foreign body reaction. Alternatively, local anesthetic can be injected forcibly into the base of the lesion in an attempt to create enough fluid pressure to extrude the larvae out of the punctum. Another line of treatment is by the occlusion/suffocation approach, which entails closure of the central punctum by liquid paraffin,

petroleum jelly, or beeswax. This approach helps in depriving the larva oxygen and encouraging it to exit on its own. Oral ivermectin (200 µg/kg) or topical ivermectin (1% solution) have also proven to be helpful.

## Prevention and control

Continued intensification of interventions against transmission of parasitic infestation is necessary to eradicate these endemic diseases from African countries. National prevention and control programs should be implemented in countries with high prevalence of infestation. Control strategies should be directed to the patterns of people behavior associated with the phases of transmission of the disease. For example, in 1922, the government of Egypt began a major control effort for schistosomiasis control, soon after the discovery that snails played an essential role in disease. In the 1940s, the country passed a number of ordinances and decrees to control snails and to require examinations for at-risk populations. The many years of efforts in Egypt have had an impact on schistosomiasis prevalence. The Egyptian Ministry of Health reported that from 1982 to 1992, the prevalence of *S hematobium* declined from about 15% to 1% in the Nile Delta and from 13% to 3% in Upper Egypt, and the prevalence of *Schistosoma mansoni* declined from about 40%-20% in the Nile Delta.

Good sanitary practice, as well as responsible sewage disposal or treatment, are necessary for the prevention of *E histolytica* infection on an endemic level. The provision of clean water sources and the treatment of contaminated drinking water with larvicides are very important in eradication of dracontiasis. The control of hydatid disease involves ensuring that dogs are not infected with the tapeworm, either by preventing the dog from eating tapeworm heads or by deworming dogs, preferably before they have had a chance to mature. Health education programs focused on cystic echinococcosis and its agents, improved hygiene, and deworming dogs are complementary methods for controlling infection with *Echinococcus*.

National control programs should be planned to encourage action at all levels to start serious study of disease prevalence, the use of anthelmintic drugs, health education programs, and community involvement. The control of parasitic infestation in Africa deserves more and renewed attention and commitment, particularly in sub-Saharan Africa. Simple but sustained control measures can relieve the burden of this underestimated problem, especially in areas of high transmission.

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