Neglected tropical diseases are a group of protozoal, parasitic, bacterial, and viral diseases endemic in 149 countries causing substantial illness globally. Extreme poverty and warm tropical climates are the 2 most potent forces promoting the spread of neglected tropical diseases. These forces are prevalent in Central and South America, as well as the U.S. Gulf Coast. Advanced cases often require specialized medical imaging for diagnosis, disease staging, and follow-up. This article offers a review of epidemiology, pathophysiology, clinical manifestations, diagnosis (with special attention to medical imaging), and treatment of neglected tropical diseases specific to the Americas.

Medical Imaging of Neglected Tropical Diseases of the Americas

Patrick Jones, BS, R.T.(R)

After completing this article, the reader should be able to:
- Define the term neglected tropical disease and discuss international interest in these conditions.
- Discuss neglected tropical diseases affecting the Americas including their pathophysiology, clinical manifestations, radiologic signs, diagnosis, and treatment.
- Describe the need for the medical community to actively prevent continued spread of neglected tropical diseases within resource-limited communities.

Neglected tropical diseases (NTDs) are a group of protozoal, parasitic, bacterial, and viral diseases endemic in 149 countries and cause substantial illness for more than 1.4 billion people globally. They are called neglected diseases because they have been largely eradicated in more developed parts of the world and persist only in the poorest, most marginalized communities and conflict areas. These diseases are contrasted with HIV/AIDS, tuberculosis, and malaria, which generally receive greater treatment and research funding. One hundred percent of low-income countries are affected by at least 5 NTDs simultaneously.

In endemic countries, NTDs cause impaired physical and cognitive development as well as illness and death, killing an estimated 534,000 people worldwide every year. Furthermore, individuals often are afflicted with more than one parasite or infection at a time. Subsequently, related morbidity and mortality, as well as contamination of potential farmland, results in difficulty earning a living and limited productivity in the workplace. These conditions trap the poor in a cycle of poverty and disease and cost developing economies billions of dollars annually. It is especially difficult to rationalize neglecting these conditions considering that the treatment cost for most NTDs mass drug administration programs is estimated at less than $0.50 per person per year. The World Health Organization (WHO) has prioritized 17 NTDs, and in May 2013, the 66th World Health Assembly adopted resolution WHA66.12 that calls for intensified, integrated measures and planned investments to improve the health and social well-being of populations affected by NTDs. WHO also has been working with member states to ensure implementation of the resolution.

Impact in the Americas

The Americas comprise North America, Central America, and South America. The phrase “Latin American and Caribbean region” can be used to
identify all countries within the Western Hemisphere and south of the United States (see Figure 1).

The Latin American and Caribbean region has a population of almost 600 million people, of whom an estimated 99 million live on less than $2 per day. Approximately 10% of the region’s extremely poor live in Bolivia, Ecuador, Nicaragua, and Venezuela. NTDs are common wherever poverty is pervasive, and these 4 countries carry approximately 14% to 15% of regional cases of Chagas disease, cutaneous leishmaniasis, dengue, and intestinal helminth infections. Bolivia leads in the number of Chagas disease cases (620,000) and the number of children who require deworming for intestinal helminth infections (3.4 million), whereas Nicaragua leads in cutaneous leishmaniasis cases (9,000-14,800), and Venezuela has the largest number of dengue cases (3.5 million). Although Cuba is better off economically, it also has many cases of dengue and intestinal helminth infections. NTDs also are widespread along the Gulf Coast states of Mexico (Tamaulipas, Veracruz, Tabasco, Campeche, Yucatan, and Quintana Roo) and in Chiapas and Oaxaca on the southern Pacific coast. Mosquito-borne diseases are especially prominent with dengue incidence rates increasing approximately 8-fold from 2000 to 2011, with peaks occurring in 2002, 2007, and 2009 (see Table 1).

Extreme poverty and warm tropical climates are the 2 most potent forces promoting the endemcity of NTDs, and these same forces are widely prevalent in the 5 states bordering the U.S. Gulf Coast: Texas, Louisiana, Mississippi, Alabama, and Florida, with 10 million Gulf Coast residents living below the U.S. poverty line. Thus, today the Gulf Coast is considered North America’s most vulnerable and impoverished region with high rates of NTDs emerging there (see Table 2). In fact, the term emerging should be used with caution because many of these NTDs are not new to the region. Outbreaks of dengue were reported in Texas from 2003-2005, with a return of the disease in late 2013 affecting the poorest communities. In addition, dengue was reported in Florida in 2009 and 2010. The U.S. Gulf Coast also is considered vulnerable to the introduction of chikungunya, a virus transmitted by Aedes mosquitoes that clinically resembles dengue, with the possibility of year-round transmission in the warm Gulf climate. Chagas disease transmission also has been confirmed in Texas and Louisiana, and a recent economic analysis revealed that Chagas disease already incurs nearly $900 million in costs in the United States.

Some urgent needs in addressing NTDs include specific recommendations for greatly expanded disease surveillance and understanding of disease transmission. For many NTDs, diagnostic tests are cumbersome or not widely available. One example is the lack of access to radiology services. Many advanced cases require specialized medical imaging, and affected individuals must travel to specialty clinics situated in more affluent communities, a journey that often is not feasible. As of 1997, when data was last collected, more than half of rural hospitals in Latin America did not offer radiology services. For this reason, a lack of awareness exists among health professionals regarding tropical disease management including identification of radiologic manifestations.

Figure 1. Map of the Western Hemisphere with Latin American and Caribbean regions in blue. Image courtesy of Heraldry via Wikimedia Commons. Licensed under the Creative Commons Attribution-Share Alike 3.0 Unported license.
Chagas Disease

Epidemiology

Chagas disease is caused by the parasite Trypanosoma cruzi and is transmitted by triatomine bugs.\(^{28}\) Chagas disease is recognized as one of the major health problems in almost every Central and South American country. Once a rural disease, Chagas disease has become an urban phenomenon as a result of socioeconomic changes, rural exodus, deforestation, and urbanization. Increases in immigration have resulted in Chagas disease becoming a health care concern in Europe and the United States as well. Furthermore, 2015 estimates from WHO indicate that 6 million to 7 million people are infected worldwide.\(^{28}\)

Pathophysiology

The pathophysiology of Chagas disease is not entirely known. Sometimes referred to as the kissing bug, triatomine bugs transmit the T cruzi parasite when insect feces enter an individual through an insect bite or skin penetration.

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Table 1
Ranking of Neglected Tropical Diseases in Latin American Countries by Prevalence and Distribution\(^{12}\)

<table>
<thead>
<tr>
<th>Disease</th>
<th>Population Currently Infected in LAC</th>
<th>Population at Risk in LAC</th>
<th>Main Vulnerable Populations or Geographic Areas</th>
<th>No. of LAC Countries Infected</th>
<th>Percentage of LAC Population Infected (% Poor People Infected)</th>
<th>Percent Global Disease Burden in LAC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trichuriasis</td>
<td>100 million</td>
<td>523 million</td>
<td>Poor rural &amp; urban slums</td>
<td>27</td>
<td>17.8 (46.9)</td>
<td>16.6</td>
</tr>
<tr>
<td>Ascariasis</td>
<td>84 million</td>
<td>514 million</td>
<td>Poor rural &amp; urban slums</td>
<td>27</td>
<td>15.0 (39.4)</td>
<td>10.4</td>
</tr>
<tr>
<td>Hookworm</td>
<td>50 million</td>
<td>346 million</td>
<td>Poor rural</td>
<td>26</td>
<td>8.9 (23.5)</td>
<td>8.7</td>
</tr>
<tr>
<td>Chagas disease</td>
<td>8 million-9 million</td>
<td>25 million-90 million</td>
<td>Poor rural &amp; urban slums</td>
<td>13</td>
<td>1.6 (4.1)</td>
<td>99.8</td>
</tr>
<tr>
<td>Schistosomiasis</td>
<td>1.8 million</td>
<td>36 million</td>
<td>Poor rural</td>
<td>4 with &gt; 1000 cases</td>
<td>0.3 (0.8)</td>
<td>0.9</td>
</tr>
<tr>
<td>Blinding trachoma</td>
<td>1.1 million</td>
<td>ND</td>
<td>Poor rural</td>
<td>3</td>
<td>0.2 (0.5)</td>
<td>1.3</td>
</tr>
<tr>
<td>Lymphatic filariasis</td>
<td>720 000</td>
<td>8.9 million</td>
<td>Poor rural &amp; urban slums</td>
<td>7</td>
<td>0.1 (0.3)</td>
<td>0.6</td>
</tr>
<tr>
<td>Dengue</td>
<td>552 141 reported in 2006</td>
<td>ND</td>
<td>Urban slums</td>
<td>23</td>
<td>0.1 (0.2)</td>
<td>ND</td>
</tr>
<tr>
<td>Cysticercosis</td>
<td>400 000</td>
<td>75 million</td>
<td>Poor rural</td>
<td>15</td>
<td>&lt; 0.1 (0.2)</td>
<td>ND</td>
</tr>
<tr>
<td>Cutaneous (CL) and visceral (VL) leishmaniasis</td>
<td>62 000 CL and 5000 VL</td>
<td>ND</td>
<td>Poor rural &amp; urban slums</td>
<td>18</td>
<td>ND (ND)</td>
<td>ND (ND)</td>
</tr>
<tr>
<td>Leprosy</td>
<td>47 612 new cases</td>
<td>ND</td>
<td>Poor rural &amp; urban slums</td>
<td>22</td>
<td>&lt; 0.1 (&lt; 0.1)</td>
<td>11.4</td>
</tr>
<tr>
<td>Onchocerciasis</td>
<td>64 new cases in 2004</td>
<td>515 675</td>
<td>Poor rural</td>
<td>6</td>
<td>&lt; 0.1 (&lt; 0.1)</td>
<td>0.3</td>
</tr>
<tr>
<td>Jungle yellow fever</td>
<td>86 new cases in 2004</td>
<td>ND</td>
<td>Jungle &amp; urban slums</td>
<td>4</td>
<td>&lt; 0.1 (&lt; 0.1)</td>
<td>&lt; 0.1</td>
</tr>
</tbody>
</table>

Abbreviation: LAC, Latin American Countries; ND, not determined.
partial or absent relaxation of the lower esophageal sphincter occurring simultaneously. In the late stages of the disease, patients begin to experience megaesophagus and megacolon involving damage to submucosal layers and the mesenteric nerve plexus. As a result, megacolon patients can present with severe constipation for periods of 60 days or longer. Chagas disease often affects the heart, resulting in epicardial ventricular tachycardia, which can lead to sudden death.

Diagnosis

Gastrointestinal and cardiac symptoms accompanied with recent travel to Latin America would be suggestive of Chagas disease. Physical examination should identify one of the following disease phases:

- Acute, nonspecific symptoms.
- Asymptomatic and a significant chronic cardiac disease.
- Formation of digestive megaesophagus or megacolon.

To confirm diagnosis, laboratory tests might include enzyme-linked immunosorbent assay (ELISA) serology testing, a technique that uses the absorption of antibodies by insoluble preparations of antigens. Another option for infectious disease diagnosis is polymerase chain reaction assays that amplify a few copies of a piece of DNA across several orders of magnitude.

In addition, histologic studies show that when host cells within parasitized defect. Body parts particularly vulnerable to parasitic transmission are the mucous membranes of the eyes or mouth. Other routes of infection include consumption of contaminated water or food, blood transfusions, and organ transplantation.

Clinical Manifestations

Patients with acute Chagas disease usually have characteristic inflammatory lesions at the site of T. cruzi entry called *chagomas*. Other early signs of the disease include fever, headache, enlarged lymph glands, pallor, muscle pain, difficulty breathing, abdominal or chest pain, and purplish swelling of one eyelid called *Romaña sign* (see Figure 2). As the disease progresses, the 3 organs predominantly affected are the esophagus, colon, and heart. Dysphagia is the most common digestive symptom and results from abnormalities of esophageal motility with esophageal muscle contractions and relaxation. Body parts particularly vulnerable to parasitic transmission are the mucous membranes of the eyes or mouth. Other routes of infection include consumption of contaminated water or food, blood transfusions, and organ transplantation.

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tissue rupture, trypomastigotes (a developmental stage of *T cruzi*) are released and often can be detected by microscopic examination of anticoagulated blood.\(^{34}\)

Dysphagia studies using fluoroscopy can exhibit a measurable delay in digestive and swallowing movements as compared to normal esophageal motility. These delays can affect the upper esophageal sphincter and various stages of the swallowing process including oropharyngeal transit, pharyngeal transit, and pharyngeal clearance.\(^{34}\) Approximately 7% of Chagas disease patients with megaesophagus develop esophageal cancer (see Figure 3). Computed tomography (CT) can demonstrate the extent of such tumors, detecting mediastinal and nodal involvement, and it can be used to confirm cases of mediastinitis and esophageal perforation.\(^{35}\)

Chest radiography is an important and inexpensive tool used to identify patients with dilated cardiomyopathy secondary to Chagas disease. Calcifications in the apical vasculature often are a radiologic manifestation of this condition.\(^{33}\) CT, as well as ultrasonography, magnetic resonance (MR) imaging, scintigraphy, and angiography, enable evaluation of infarcts resulting from chronic Chagas heart disease.\(^{35}\) When advanced imaging capabilities are available, noncontrast multidetector electrocardiography-gated CT scanning for calcium scoring is the protocol of choice, followed by contrast-enhanced CT coronary angiography for examining Chagas disease–associated infectious myocarditis.\(^{36}\)

Cardiac MR imaging studies have shown that both myocardial fibrosis and segmental cardiac wall motion abnormalities were associated with ventricular arrhythmia in patients with chronic Chagas heart disease. Even in patients with this condition who have preserved or minimally impaired ventricular function, the arrhythmogenic substrate can be present. Myocardial fibrosis detected on cardiac MR is the most important variable associated with ventricular arrhythmia.\(^{37}\)

**Treatment**

Chagas disease is curable if treated in the acute phase.\(^{32}\) Surgery is a treatment option for megacolon secondary to the disease and can consist of either the Duhamel-Haddad or the Habr-Gama procedures, which result in similar final configurations involving

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**Figure 3.** Anterolateral (A) and lateral (B) chest projections showing megaesophagus as a result of Chagas disease in a man with development of cancer in the distal third of the esophagus with perforation forming a large abscess that extends into the right lower lobe and pleural space. Reprinted from Palmer P, Reeder M. The Imaging of Tropical Diseases [DVD]. The International Society of Radiology Web site. http://www.isradiology.org/tropical_diseases/tmr/chapter4/esophagus.htm. Accessed December 17, 2015.
rectalsigmoidectomies of all, or almost all, of the dyskinetic rectum.\textsuperscript{39} Most patients with Chagas disease have some form of heart disease, and this must be considered when determining the level of risk in surgical intervention.\textsuperscript{32}

To cure patients of parasitemia from Chagas disease, the prescribed medication is either benznidazole or nifurtimox. These 2 medications destroy the \textit{T cruzi} parasite.\textsuperscript{39} In the United States, these antiprotozoal agents are not U.S. Food and Drug Administration approved and are available only from the Centers for Disease Control and Prevention (CDC) under investigational protocols.\textsuperscript{40} The main contraindications to these treatment options are pregnancy, neurologic and psychiatric conditions, and existing kidney or liver failure.

Preventive measures involve spraying insecticides and improving housing to increase hygienic living conditions because no vaccination solutions are available for Chagas disease.\textsuperscript{41}

**Cysticercosis**

**Epidemiology**

Cysticercosis is an infection caused by a helminth (parasitic worm), specifically the tapeworm, \textit{Taenia solium}. It is found worldwide and is endemic in most Latin American countries, most often in rural areas where hygiene practices are poor. As a result of increasing immigration, numbers of patients with cysticercosis are rising in countries where local transmission typically is low. The prevalence of neurocysticercosis, which occurs when larvae reach the brain, is 0.2 to 0.6 per 100 000 inhabitants in some western states of the United States, and it is diagnosed in more than 2% of patients visiting emergency departments for seizures.\textsuperscript{42}

**Pathophysiology**

\textit{Taenia solium} has a 2-host life cycle between humans and pigs. Only humans can serve as a host for adult tapeworms, whereas both pigs and humans can serve as intermediate hosts for the larval form. Oncospheres, or tapeworm embryos, are passed to the environment in human feces, which in settings of poor sanitation and free-roaming animals can be ingested by pigs. After ingestion, the embryos hatch and actively cross the intestinal mucosa to the bloodstream, providing access to the liver, eyes, central nervous system, and striated muscle, where they develop into cysticerci, larval tapeworms enclosed within a sac. Pigs with cysticercosis become intermediate hosts, and the disease lifecycle is completed when undercooked pork infected with cysts is consumed by humans.\textsuperscript{43,44} After cysts are ingested by humans, the scolex, or tapeworm head, turns inside out, attaches to the intestinal wall, and in 2 months matures into a 2-m to 4-m ribbon-like tapeworm.

Cysticercosis infections usually cluster around tapeworm carriers, meaning person-to-person spreading of the disease is likely to be the predominant means of human contamination vs contamination through environmental sources.\textsuperscript{45-47} Individuals infected with tapeworms spread \textit{T solium} eggs indirectly through poor hygiene practices such as lack of hand washing and contaminated food, water, or surfaces. Affected individuals can be reinfected with larvae produced by tapeworms already in the body.

**Clinical Manifestations**

Acute cysticercosis outside of the central nervous system usually is not associated with clinical manifestations, with the exceptions of ocular involvement and rare cases of massive muscular involvement. It can be months or years after initial infection before symptoms of chronic infection manifest, and these symptoms are dependent on the location and number of cysts in the body. When the cysts die, the surrounding tissue swells, increasing pressure and inducing symptoms. Cysts in muscle tissue might develop into palpable, sometimes tender, subcutaneous masses. Cysts in the eyes can float in the vitreous humor, disturbing vision and potentially swelling, causing detachment of the retina. Recurrent seizures occur in approximately 80% of symptomatic cases of neurocysticercosis, making epilepsy the most common neurologic manifestation\textsuperscript{48} and neurocysticercosis one of the most important causes of seizures in the world.\textsuperscript{49} Other neurologic symptoms include focal deficits (16%), increased intracranial pressure (12%), and cognitive decline (5%).\textsuperscript{48} In fact, neurocysticercosis is regarded as “the great imitator” because it can mimic almost any neurological disorder including isolated headaches, stroke, or involuntary movement.\textsuperscript{45,50-52}
**Diagnosis**

Histological confirmation of the parasite is not possible in most neurocysticercosis cases because of complications from inflammatory response or effects of prior medications. Diagnosis usually is based on neuroimaging and confirmed by serological testing via enzyme-linked immunoelectrotransfer blot assays, which use lentil-lectin purified glycoprotein antigens to detect *T solium* antibodies. Enzyme-linked immunoelectrotransfer blot sensitivity is approximately 98%, meaning patients with more than one viable cyst will have a positive serology, and a negative serology result should lead to investigation of alternative diagnoses. Detection of anticysticercal antibodies in the cerebral spinal fluid by ELISA is 89% sensitive and 93% specific in patients with viable neurocysticercosis infections and is used when enzyme-linked immunoelectrotransfer blot is not available. Antibodies to *T solium* frequently are reported in the asymptomatic general population in endemic regions, suggesting prior or current exposure to the parasite.

Radiologic confirmation of cysticercosis can be completed with radiography or ultrasonography, although it typically is limited to late disease identification when cysticerci are more distinguishable. Soft tissue radiography commonly demonstrates multiple (often several hundred) calcifications. These calcifications appear either linear or oval and measure 4 mm to 10 mm or more in length and 2 mm to 5 mm in width. Moreover, calcified cysts have their long axes in the plane of the surrounding muscle bundle (see Figure 4).

On chest radiography, cysticerci might be seen in the lungs with measurements of about 3 mm to 6 mm in diameter. The outer shells of the lesions are calcified, with somewhat lighter and softer centers, and remain more rounded when compared with the oval calcified cysts found in muscle. Ultrasonography is particularly useful in examining cysticercosis of the eye and extraocular muscles. In examinations of the orbit, as well as other soft tissue and musculoskeletal structures, the primary diagnostic features are oval or round well-defined cystic lesions with an eccentric echogenic scolex within them. In cases of calcified cysticercosis, ultrasonography demonstrates multiple calcifications in soft tissue similar to the pathognomonic millet seed-shaped elliptical calcifications in soft tissue described on radiography.

In regions where CT and MR are available, these scans can provide information on the morphology and localization of cysticercosis cysts, burden of infection, stage of the cysts, and surrounding inflammation. The appearance of parenchymal brain lesions on neuroimaging indicates their stage of shrinkage and can be used to monitor the effectiveness of treatment regimens. Live vesicular cysts are small, rounded lesions with little or no pericystic edema; they do not enhance with contrast. The cysts frequently show the tapeworm scolex as an internal asymmetric nodule (ie, a hole-with-dot), and several viable cysts showing scolecis confirm the diagnosis. Calcified cysticerci are clearly visible on CT as nonenhancing hyperdense nodules, also without peripheral edema. The degenerative process becomes apparent when colloid cysts with poorly defined borders are surrounded by edema and show a marked ring or nodular contrast enhancement. MR diffusion-weighted

imaging and apparent diffusion coefficient maps might allow visualization of the scolex in colloidal cysticerci, which are seldom visible on CT or conventional MR sequences. In rare cases in which a solitary degenerating cyst is present, misleading differential diagnoses can lead to unnecessary biopsies. For example, neurocysticercosis and tuberculosis often are encountered in low-income regions of the world and have some similar clinical and radiological features. Thus, diagnostic criteria have been validated to differentiate these entities in patients with one brain nodule on the basis of size, edema, and clinical presentation (see Figure 5).

**Treatment**

Therapy can include symptomatic therapy, antiparasitic treatment, or surgery (eg, lesion resection or shunt placement) and often requires a combination of these options. Advantages of medical therapy include treating surgically unreachable and multifocal cysticercosis. Therapy planning depends on proper characterization of the neurocysticercosis type, location, and level of brain involvement. Surgery is considered only when a diagnosis is in doubt or the infection is clinically progressive.

The International Task Force for Disease Eradication has targeted *T solium* infection for focal elimination and eventual eradication. Furthermore, field control efforts have been attempted since 1987 in Latin American countries including Ecuador, Mexico, Peru, Guatemala, and Honduras. Preventive measures recommended by the CDC include good hand washing hygiene using soap and warm water after bathroom use and changing diapers and before handling food. Proper food and water consumption practices involve washing and peeling all raw vegetables and fruits before eating, drinking only bottled or boiled water and bottled drinks, and filtering and dissolving iodine tablets into water when traveling within developing countries.

**Echinococcosis**

**Epidemiology**

One of the oldest known diseases is hydatid disease, also called human cystic echinococcosis. Today, this parasitic disease is the most serious and widespread human flatworm infection in the world, affecting more than 1 million people and costing $3 billion annually in clinical treatments and livestock compensation. The disease has 2 predominant forms: alveolar and cystic. Alveolar echinococcosis is not commonly found in the Americas, whereas cystic echinococcosis can be found in Central America, South America, and in rare cases, North America.

**Pathophysiology**

Cystic echinococcosis is zoonotic (ie, transmissible from vertebrate animals to humans), and the transmission cycle begins when dogs eat meat infected with cysts that grow into adult tapeworms (*Echinococcus granulosus*). The dogs eventually shed tapeworm eggs in their feces, contaminating the soil. Sheep subsequently consume the eggs and develop parasitic larva in their viscera. The cycle affects humans when they consume water or food that has been contaminated by dog fecal matter, thus sheep farmers are more likely to develop hydatidosis because their flock often acts as...
Ultrasonography can provide multiple imaging planes for optimized diagnosis of hydatid cysts. Used exclusively or in combination with laboratory tests, radiography and ultrasonography can allow for an almost 100% positive diagnosis of hydatid disease in the majority of cases.35

Using ultrasonography, cysts initially were categorized via the Gharbi classification in 1981, a system widely used but in modified forms. The Gharbi classification is as follows35,65,69:

- **Type I** – pure fluid collection.
- **Type II** – fluid collection with a split wall.
- **Type III** – fluid collection with septa.
- **Type IV** – heterogeneous echo patterns.
- **Type V** – reflecting thick walls.

The cyst classification process also was dependent on information related to the size, number, and localization of the cysts and any associated complications.66 In 2003, WHO proposed a standardized classification based on the active-transitional-inactive cyst status as indicated by ultrasonography (see Table 3).70

Contrast-enhanced CT (CECT) also is an effective modality for diagnosing cystic echinococcosis. The “air bubble” sign, caused by air trapped within a cyst, can provide significant statistical results (85.7%
sensitivity and 96.6% specificity). A hydatid cyst is viewed optimally in the mediastinal window as a single or multiple small, rounded radiolucent area with specific margins in the outer portion of a solid mass lesion. An infected cyst also will produce higher attenuation values (> 20 HU), appearing brighter when compared with unruptured cysts. The density of an infected cyst can be problematic when differentiating it from an abscess or neoplasm. However, follow-up CECT scans can be useful in determining the presence of an “empty cyst” sign, an indication that contents were completely expectorated.68 Unless infection produces gas, premature death of the cyst and infection have the same appearances on CT and ultrasonography. In both cases, the fluid component becomes echogenic on ultrasonography and denser on CT, and there is an overall loss of clarity of the lesion’s internal detail.35

**Treatment**

Cystic echinococcosis can be expensive and complicated to treat, sometimes requiring extensive surgery, prolonged drug therapy, or both.64 Today, the accepted form of treatment is an image-based and stage-specific approach.71 Options for management are64:

- Surgery.
- Anti-infective drug regimen.
- Expectant management, or watching and waiting with medical imaging surveillance.

Surgery remains the preferred treatment for liver cystic echinococcosis64 and often can lead to a cured status, whereas treatment with a drug therapy of albendazole or mebendazole decreases the risk of disease recurrence and intraperitoneal seeding of infection that might develop with cyst rupture.72 Some cysts can be asymptomatic and remain inactive. These often disappear without treatment; therefore, watching and waiting can be beneficial.66

**Foodborne Trematodiases**

**Epidemiology**

Foodborne trematodes (flatworms or “flukes”) in the larval stage enter a host when they are ingested as part of a contaminated meal. These infections are particularly prevalent in East and Southeast Asia, and in Central and South America. The WHO estimates that 40 million people have these types of infections.73 Most of these infections are mildly pathogenic, but several are severe including clonorchiasis, opisthorchiasis, paragonimiasis, and fascioliasis. Of these, only paragonimiasis and fascioliasis are known to affect people in the Americas.74

Paragonimiasis infections are transmitted by freshwater shellfish within the Western Hemisphere.74

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**Table 3**

**World Health Organization Classification of Cystic Echinococcosis Cysts and Imaging Features**

<table>
<thead>
<tr>
<th>Classification</th>
<th>Clinical Group</th>
<th>Ultrasonography Imaging Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cystic lesion</td>
<td>Group 1: Active group: cysts developing and are</td>
<td>Unilocular, with uniform content, cyst wall not visible</td>
</tr>
<tr>
<td></td>
<td>usually fertile</td>
<td></td>
</tr>
<tr>
<td>CE1</td>
<td>Unilocular, simple cyst with visible cyst wall</td>
<td></td>
</tr>
<tr>
<td>CE2</td>
<td>Multivesicular, multiseptated cysts; cyst septations</td>
<td>“wheel-like” structures, and the presence of daughter cysts is</td>
</tr>
<tr>
<td></td>
<td>produces</td>
<td>indicated by “rosette-like” or “honeycomb-like” structures</td>
</tr>
<tr>
<td>CE3</td>
<td>Group 2: Transition group: cysts starting to</td>
<td>Unilocular cyst that might contain daughter cysts; detachment of</td>
</tr>
<tr>
<td></td>
<td>degenerate, but usually contain viable protoscoleces</td>
<td>laminated membrane; “water-lily” sign</td>
</tr>
<tr>
<td>CE4</td>
<td>Group 3: Inactive group: degenerated or partially</td>
<td>Heterogeneous content, “ball of wool” sign, which is indicative of</td>
</tr>
<tr>
<td></td>
<td>or totally calcified cysts; very unlikely to be</td>
<td>degenerating membranes</td>
</tr>
<tr>
<td></td>
<td>fertile</td>
<td></td>
</tr>
<tr>
<td>CE5</td>
<td>Thick calcified wall</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviation: CE, cystic echinococcosis.
Various species, including *Paragonimus mexicanus*, *Paragonimus ecuadoriensis*, and *Paragonimus kelicotti*, have affected humans in Canada and Central and South America, especially Colombia, Ecuador, Peru, Venezuela, and parts of Brazil, as well as Costa Rica, Honduras, and Mexico. In contrast, fascioliasis, which is caused by the parasite *Fasciola hepatica*, is prevalent in sheep- and cattle-raising areas of South America, with worldwide infection estimated to exceed 2 million.

**Pathophysiology**

The life cycle of *Paragonimus* strains begins when the unembryonated eggs are released into fresh water by infected humans via unsanitary practices regarding their mucus or stool. While in the fresh water, the eggs become embryonated and hatch into miracidia (larvae) that infect snails, their primary host. Within the snail, the miracidia undergo several changes and leave the snail as cercariae that then infect a secondary host of crustacean crabs and crayfish, enclosing themselves within metacercariae (soft tissue cysts). Humans become infected following consumption of raw, undercooked, or pickled freshwater shellfish containing the metacercariae. The parasite then exits its cystic stage to penetrate the human host’s peritoneum and lungs. Once settled, the trematodes develop into adult flukes in 2 months (see Figure 7).

In contrast, fascioliasis involves development of *F. hepatica* following consumption by sheep and cattle where they grow into adult flukes, specifically in the bile ducts of the infected mammals. These animals then introduce immature *F. hepatica* eggs in their feces to a freshwater environment. Similar to *Paragonimus* strains, the eggs embryonate and hatch in the water after several weeks as the miracidium that infects a snail host. After several more weeks, the parasites emerge from the snail as cercariae; however, they select water plants to encyst as metacercariae. Humans become infected following consumption of contaminated plants, especially watercress. The maturation of the parasite in the human host can take approximately 3 to 4 months. During this time, the metacercariae exist in the duodenum and travel through the intestines, peritoneal cavity, and liver to reach the biliary ducts where they become adult flukes.

**Clinical Manifestations**

Clinical signs of paragonimiasis are from mechanical damage caused by the migration of the worm from the gut to the lungs. In some instances, the flukes can migrate in the host ectopically to the brain or subcutaneous sites of the extremities. When the parasite reaches the lung, the most common site of infection, it causes hemorrhaging, inflammatory response, necrosis of lung parenchyma, and fibrotic encapsulation. Acute paragonimiasis is demonstrated by a cough, abdominal pain, discomfort, and low-grade fever that can occur 2 to 15 days after infection. A person with chronic pulmonary paragonimiasis has a cough producing brown and blood-streaked pneumonia-like sputum. The hemoptysis typically is induced by strenuous work, and the coughing often can be confused with chronic bronchitis or bronchial asthma.
the intestine to and through the liver. Symptoms can include nausea, vomiting, and abdominal pain or tenderness. In addition, fever, rash, and difficulty breathing can occur in acute cases.\textsuperscript{1} When the fluke reaches the liver, hepatomegaly occurs as the migratory flukes destroy liver parenchyma. Chronic fascioliasis is associated with the presence of adult worms in the bile ducts of the host. At this stage, symptoms can be difficult to distinguish from other hepatic diseases such as cholangitis, cholecystitis, and cholelithiasis.\textsuperscript{75}

**Diagnosis**

Diagnosing paragonimiasis involves analysis of patient sputum, stool, and biopsies, as well as medical imaging.\textsuperscript{74} To overcome the low sensitivity of egg detection tests and low specificity of intradermal tests, serological testing, such as ELISA, is used to measure the serum levels of antibodies and has been found by the CDC to have a sensitivity and specificity greater than 95%.\textsuperscript{74}

In people suspected of having a paragonimiasis infection, chest radiographs show abnormalities of the lungs or pleura similar to tuberculosis including infiltrates, nodules, cavities, and fibrosis. Evaluation of the radiographs is based on the migration of the flukes. Once the flukes penetrate the lung, hemorrhagic and exudative pneumonia occurs around them, and 2-mm to 4-mm thick and 2-cm to 7-cm long band-like opacities adjacent to the pleura representing worm migration tracts or peripheral atelectasis are commonly seen.\textsuperscript{35}

Better-defined nodules or thin-walled cysts appear on medical imaging if flukes remain in the same position because surrounding airspace consolidation can occur. In patients with airspace consolidation without visible cysts on radiographs, a CT scan with intravenous contrast can highlight cysts within the consolidated lung because of the difference of the darker (lower attenuation) fluid-filled cysts compared with brighter adjacent consolidated lung. Once lytic cysts extrude intracystic fluid, a CT scan might show them as air-filled cysts within the consolidated lung.\textsuperscript{35} The most characteristic radiographic feature of the mature stage of paragonimiasis is the “solar eclipse” sign, which has been seen in up to 82% of patients in chest radiography and chest CT scans. These ring shadows represent the thin-walled borders of cystic cavities, with a crescent-shaped opacity along one side of the border appearing like the corona of a solar eclipse. This feature represents worms attached to the wall of a cyst (see Figure 8).\textsuperscript{35}

Similarly, in fascioliasis-endemic areas, examining stool samples for evidence of parasitic eggs can be performed; however, serologic diagnosis using *F. hepatica* excretion-secretion antigens and ELISA testing has been more effective. The indirect hemaglutination test, a suspension that binds red blood cells, has a 100% diagnostic sensitivity and 97% specificity, if the metabolic antigen is used.\textsuperscript{35} In addition, testing the blood for high eosinophil levels has been effective in up to 68% of individuals with severe fascioliasis infections. Although laboratory tests help with diagnosis, serologic assays are limited because they cannot distinguish between past and current infection.\textsuperscript{74}

CT and ultrasonography have become widely available in endemic areas to assist with diagnosing fascioliasis.\textsuperscript{35} When using CT to assess an infected liver, imaging shows hypodense migratory lesions.\textsuperscript{74} These nodular intrahepatic lesions present with diminished attenuation, ranging in size from 4 mm to 10 mm but sometimes as large as 2 cm. Intravenous contrast medium might allow definition of these lesions on dynamic and delayed scans (see Figure 9). These
nodosal lesions, which correspond to microabscess formation and can occur in clusters, are difficult to differentiate from necrotic neoplasms or other abscesses. If peripheral, convoluted lesions or channels are present in a patient from an endemic region, hepatic fascioliasis becomes the primary consideration. These convoluted channels can be seen during surgery and laparoscopy and in many instances are identified as migratory tracts left by the flukes.  

Often, the small cystic lesions calcify after treatment. Performing CT scans to follow up with fascioliasis patients helps document response to treatment. Biliary duct dilatation and irregular wall thickening are thought to be better demonstrated on ultrasonography. The nonspecific round nodular lesions of hepatic fascioliasis can be viewed with ultrasonography as lesions of variable echogenicity. However, the characteristic convoluted channels of fascioliasis are less identifiable on ultrasonography, and therefore CT is the preferred cross-sectional imaging modality for the hepatic form of this disease.  

**Treatment**

Treatment for paragonimiasis entails a short course of oral praziquantel 3 times a day for 3 days, which has an efficacy rate of at least 90%. Praziquantel is an anthelmintic, meaning it is used to treat worm infections, and works by inducing severe spasms and paralysis of the worm’s musculature. The worm is then either destroyed in the intestines or passed whole in the stool. When treating cerebral paragonimiasis, praziquantel should be administered in combination with corticosteroids.

Fascioliasis differs from other fluke infections in that it does not respond well to treatment with praziquantel. The most effective treatment is triclabendazole, another anthelmintic. This medication can be difficult to access in the United States; it is available only through the CDC under a special (investigational) protocol. The cure rate for the disease is approximately 80% after taking 1 or 2 oral doses of the drug. Repeat dosing is required if abnormal radiologic findings or eosinophilia do not resolve.

**Onchocerciasis**

**Epidemiology**

Onchocerciasis, often referred to as river blindness, is a parasitic disease caused by the filarial worm, *Onchocerca volvulus*. Onchocerciasis is an eye and skin disease transmitted to humans through the bites of infected black flies in the family Simuliidae, which breed in environments that have fast-flowing rivers and streams. Onchocerciasis is the world’s fourth leading cause of preventable blindness, with the parasitic disease threatening the health of approximately

25 million to 125 million people worldwide. Although 99% of those affected by onchocerciasis live in Africa, approximately 500,000 people in the Americas also are at risk. It is believed that onchocerciasis was brought to the Western Hemisphere through slave trade in the Americas and spread through migration. The greatest risks within the Americas region have been isolated to 13 foci of infection throughout Brazil, Colombia, Ecuador, Guatemala, Mexico, and Venezuela.

Pathophysiology
People who live in remote villages near rivers have the greatest susceptibility for black fly bites. Once the *O. volvulus* parasite is transmitted to the human host, the infective-stage larvae molt twice and mature into adult worms within nodules under the skin. These nodules are referred to as *onchocercomas*. At this point, the adult worms begin to reproduce, creating millions of larvae (microfilariae).

Clinical Manifestations
The infective activity of onchocerciasis can be differentiated by that of microfilariae and of adult worms. The most significant activity pertains to the wandering microfilariae, which can result in groups as high as 2000 larvae/mg of skin. In the infected host, microfilariae are found in all layers of skin, but are most concentrated within the dermal papillae. Histopathology of onchocercomas demonstrate that the nodules are firm and have 3 separate layers:
- An outer fibrous layer of granulation and scar tissue.
- A middle layer of inflammatory cells.
- A central soft core that contains adult nematodes surrounded by an amorphous, eosinophilic, hyaline material named Splendore-Hoepli material.

Symptoms of an onchoceriasis infection are caused by the migration of the microfilariae and include intense itching, rashes, disfiguring skin lesions (referred to as *leopard* or *lizard skin*), and eye disease that can result in blindness. Although the most severe infections occur in the skin and eyes, lymph nodes also are commonly affected. Onchocercal dermatitis is inflammatory and progressive, leading to fibrosis and replacement of healthy skin. Changes in skin thickness, pigmentation, edema, and scarring of the epidermis are key indicators of this disease.

Dermal elastic fibers are lost gradually, resulting in abnormally wrinkled skin. Evaluation of visually impaired patients involves assessing the amount of microfilaria in the cornea and the anterior chamber of each eye. Assessment also is performed for other conditions that might result from onchocerciasis such as limbitis, iridocyclitis, sclerosing keratitis, chorioretinitis, and papillitis.

Diagnosis
Physical examination and diagnosis begins with skin biopsies. Biopsy sites in people from South America suspected of having the onchocerciasis infection are more commonly collected from the upper body because the black fly endemic to the Americas is the *Simulium ochraceum*, a high-biting species. This differs from *Simulium damnosum*, which is endemic to Africa and a low-biting species.

Radiography demonstrates filamentous calcification within nodules, particularly on soft tissue extremity images or mammograms, although the pattern in many cases is nonspecific. Even with the anatomic regions of interest biopsied, the diagnosis still might be difficult to confirm because of microcalcification occurring late in the inflammatory process, long after the worms are dead and fragmented.

Ultrasonography increasingly is used in the developing world to diagnose onchocerciasis and identify associated skin nodules. With improvements in 2-D grayscale imaging and use of higher frequency transducers, onchocercomas can be categorized and differentiated from other nodules and lymph nodes. A typical pattern demonstrates a lateral acoustic (refractive) shadow, a hypoechoic rim or layer, and a central zone of intermediate echogenicity in which numerous tiny, highly echogenic foci are seen. These foci are referred to as a *worm center*. Single and conglomerate nodules also can be distinguished (see Figure 10). The conglomerate can be evaluated based on the presence of multiple worm centers. After images are obtained, nodules can be removed surgically. Comparisons then can be made between sonograms and pathological sections (see Figure 11).

Advances are being made with the use of MR imaging in regard to associations between onchocerciasis and epilepsy. T1-weighted and T2-weighted,
fluid-attenuated inversion recovery imaging and 3-D spoiled gradient recall imaging sequences to examine hippocampus anatomy have shown an association of intraparenchymal brain pathologies and *O volvulus* infection that might indicate a cause of nodding syndrome in young people in areas endemic for onchocerciasis. Symptoms of nodding syndrome are repetitive dropping forward of the head and other seizure-like activity.

A diagnostic test reserved for cases in which all other tests prove negative is the Mazzotti test. This test involves 5 mg diethylcarbamazine administered orally to the patient to inhibit neuromuscular transmission in nematodes. The test is considered positive for onchocerciasis if an intense skin rash and itching results within 2 hours; these symptoms are caused by dying microfilariae. Corticosteroids can be administered postexamination to relieve pruritus for a few days, but severe systemic reactions and ocular complications are risks with this diagnostic method.

**Treatment**

The treatment of choice for onchocerciasis is ivermectin, which has been shown to reduce the occurrence of blindness and to reduce the occurrence and severity of nodding syndrome. It is also effective in reducing the severity and frequency of skin manifestations. Other modes of treatment include mebendazole and albendazole.

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of skin symptoms. Another preventive measure involves using environmentally safe insecticides to spray areas where black flies lay their eggs.  

Pan American Health Organization resolution CD48.12 was established in 2008 to interrupt transmission of onchocerciasis in Latin America. This public health program has been effective in eliminating the disease in Colombia and Ecuador. Such programmatic success is attributed to robust public-private partnerships involving national governments, local communities, donor organizations, intergovernmental bodies, academic institutions, nonprofit organizations, and the pharmaceutical industry. The accomplishment of disease control in these Latin American countries is informing the program about ways to control and eliminate onchocerciasis in Africa.

**Schistosomiasis**

**Epidemiology**

Schistosomiasis, also known as *bilharzia*, ranks second only to malaria in prevalence, affecting approximately 240 million people worldwide. It is considered the most lethal of all NTDs, leaving many chronically ill and causing 100 000 deaths annually. More than 76 countries are affected, with Brazil, Suriname, Venezuela, the Dominican Republic, Guadeloupe, Martinique, and Saint Lucia experiencing the greatest impact within South America and the Caribbean. These regions have a tropical and sub-tropical climate and consist of limited-resource communities without potable water and adequate sanitation.

**Pathophysiology**

Disease transmission occurs when infectious forms of the parasite (cercariae) living in certain types of freshwater snails emerge and contaminate the surrounding environment. Human hosts become infected when they come into contact with fresh water infested with blood trematodes, known as *schistosomes*. Most human infections are caused by the larval forms of parasitic blood fluke strains including *Schistosoma mansoni*, *Schistosoma haematobium*, or *Schistosoma japonicum*; however, the most common variety within South America and the Caribbean is *S. mansoni*. The various strains of schistosomes have different egg-laying capacities, which have been estimated to be as high as 3500 eggs per day in the case of *S. japonicum* and 300 eggs per day in the case of *S. mansoni*. Schistosomes can survive in the water without a host for 48 hours. People become infected when their skin comes in contact with the contaminated water and the cercariae penetrate the human host’s skin. While the egg matures into an adult worm, it can travel to and invade the intestines, liver, or bladder. *Schistosoma mansoni* causes intestinal schistosomiasis, dwelling within the blood vessels surrounding the intestines, in contrast to other strains that can cause urogenital schistosomiasis. The next generation of schistosome eggs undergoes asexual multiplication and is deposited within the rectum or large intestine. From there, the eggs are reintroduced to the environment via poor sanitation practices and contamination of fresh water with infected feces (see Figure 12).

**Clinical Manifestations**

In the first days of infection, a rash or urticaria might develop. Fever, chills, cough, and muscle aches can
plaques beneath the normal mucosa of the rectum. In some cases, the bowel wall might be limited to glistening granulation tissue, whereas in others, evidence of immune reaction might be related to polyp formation, with polyps potentially large enough to be mistaken for neoplasms. A biopsy of the lesions will determine whether they contain parasitic eggs, but some patients could have a normal examination, despite presence of an active infection.

**Diagnosis**

Identifying the parasite in stool and urine samples often is the first step when schistosomiasis is among the differential diagnoses. Evidence of infection also can be established through blood samples. However, the accuracy of blood tests requires deferring sample collection until 6 to 8 weeks following the most recent exposure to contaminated water. The most reliable serological tests are indirect immunofluorescence, using 2 antibodies to label a specific target antigen with a fluorescent dye, and ELISA.

Chest radiography typically displays normal lung markings or shows increased vascular and interstitial markings and minimal enlargement of the hilar lymph nodes. Radiography of the abdomen is of little assistance unless bladder or ureteric calcifications are present, in which cases they can be useful for investigation. Rectal and colonic calcification might be seen, most commonly in the right side of the colon. The radiologic manifestation varies with bowel distention and can therefore change frequently. Calcification might appear laminar (formed in a lumen), amorphous (formed in a solid organ), or corrugated (grooved) (see Figure 13). Nonspecific hepatosplenomegaly also might be identified, with splenomegaly being particularly recognized in patients from Brazil. Myelography can be useful to diagnose spinal schistosomiasis but is not as accurate as CT or MR imaging.

*Schistosoma mansoni* and *S japonicum* infections are investigated with barium-assisted examinations of the bowel, as well as colonoscopy. These studies can exhibit clusters of small, yellow, nodular pseudotubercles or plaques beneath the normal mucosa of the rectum. In some cases, the bowel wall might be limited to glistening granulation tissue, whereas in others, evidence of immune reaction might be related to polyp formation, with polyps potentially large enough to be mistaken for neoplasms. A biopsy of the lesions will determine whether they contain parasitic eggs, but some patients could have a normal examination, despite presence of an active infection.

Sonographic examinations can show liver enlargement, more prevalent in the left lobe than in the right. Multiple small nodules throughout the liver parenchyma also might be identified as having a hypoechoic character. These nodules can be 4 mm to 5 mm in
diameter and are typically well demonstrated on CT imaging as hypodense lesions with delayed contrast enhancement. Diagnosis is reliable only when imaging findings are correlated with the clinical findings, with marked eosinophilia, and with eggs found in feces or on biopsy.35

MR imaging previously was limited to evaluation of the central nervous system, particularly for imaging the brain or spinal cord when infected with S. mansoni. In addition, it was thought that MR imaging of intra-abdominal schistosomiasis had little advantage over ultrasonography or CT.35 However, abdominal MR imaging reveals related focal or diffuse liver disease and vascular territories. MR can show heterogeneity of hepatic parenchyma, the presence of peripheral periporal fibrosis, splenomegaly, siderotic nodules (pigmented by iron), and the presence of venous collateral pathways. Furthermore, when compared with ultrasonography and CT, MR imaging also might be more sensitive, showing disease progression, stage, and response to therapy.37

Treatment

The WHO strategy on using anthelmintic drugs makes it possible to control schistosomiasis in poor and marginalized communities.35 Efforts to control the disease have involved education on the appropriate disposal of feces and urine and treatment with praziquantel.36 Although treatment with praziquantel is fairly effective in reducing or eliminating active infection, it is not a cure for everyone. Reinfection continues to be a problem in high-risk communities. A repeat dose of praziquantel, given 2 to 8 weeks after the first dose, can improve cure rates and reduce the intensity of remaining infections in population-based programs. Repeated dosing has demonstrated particular advantages in the treatment of S. mansoni, but less consistent improvement was seen after double-dosing for S. haematobium, the cause of urogenital schistosomiasis.37

Soil-transmitted Helminths

Epidemiology

Soil-transmitted helminth infections are some of the most common infections worldwide and affect the poorest and most deprived communities. Among these communities, children are most often infected. Furthermore, because these infections are linked to populations with poor hygiene and a lack of sanitation, they occur wherever poverty exists. More than 4 billion people are at high risk throughout the world, with more than 1 billion already infected.39

Transmitted by contact with contaminated soil, the most infectious of these parasitic worms are the roundworm ascaris (Ascaris lumbricoides), affecting approximately 807 million to 1.12 billion people; the whipworm causing trichuriasis (Trichuris trichiura), which affects approximately 604 million to 795 million people; and hookworms (Anclostoma duodenale and Necator americanus), affecting approximately 576 million to 740 million people.34 These diseases are distributed widely in tropical and subtropical areas where they proliferate but also can be found in temperate zones during warmer months.

Pathophysiology

Soil-transmitted helminths live in the human intestine, and their eggs are passed to the environment through the host’s feces when defecation occurs near bushes, in a garden or field, or when used as fertilizer. After the eggs mature in the soil, they become infectious and infect new human hosts when the eggs are ingested, often when vegetables and fruits have not been carefully cooked, washed, or peeled before consumption.

Ascariasis develops when larvae hatch in the small intestine, penetrate the bloodstream, travel to the lungs, and return to the intestines via the airway. Once Ascaris is transmitted to a host, the adult form can live in the body for 1 to 2 years, and grow as long as 40 cm with the thickness of a pencil (see Figure 14).34 As with ascariasis, humans are infected with whipworm when eggs are ingested. Whipworm larvae hatch in the small intestine, but rather than penetrating tissue and traveling throughout the host, they mature and remain in the large intestine. The adult whipworm grows to 4 cm and can live in a person’s bowel for 1 to 3 years.34

Hookworm eggs are not infectious in the egg form. They hatch while still in the soil and larvae mature into a form that can penetrate the skin of humans. One exception is the Anclostoma duodenale hookworm,
vomited, or emerge through the nose or anus. When the worms migrate to the common bile duct, pancreatic duct, appendix, and other sites, secondary conditions might develop including cholangitis, cholecystitis, pyogenic liver abscess, pancreatitis, obstructive jaundice, or appendicitis. With heavy infestations of Ascaris, masses of worms can cause intestinal obstruction, volvulus, intussusception, or death.

Heavy infections of whipworm can be accompanied by abdominal cramps, tenesmus (recurrent feeling of needing to evacuate the bowels), diarrhea, distention, nausea, and vomiting. Trichuris dysentery syndrome might develop, particularly in malnourished younger children, with findings resembling inflammatory bowel disease including bloody diarrhea and rectal prolapse. Children who have become chronically ill might present with iron deficiency anemia, growth retardation, and clubbing of the fingers. Furthermore, a patient with heavy infestation can lose substantial amounts of blood. For example, a patient with 800 worms can lose approximately 4 cc of blood per day.

Symptoms unique to hookworm disease include:
- Transient pruritic skin rash.
- Pulmonary symptoms.
- Anorexia.
- Diarrhea.
- Abdominal discomfort.
- Iron deficiency.

Many of these symptoms result from chronic infection with large worm burdens and severe effects on the circulatory system. Patients can experience pallor, weakness, dyspnea, and heart failure caused by anemia. In addition, protein loss during hookworm infections can lead to hypoalbuminemia, edema, and ascites. This disease also can impair growth and cognitive development in children.

**Clinical Manifestations**

People with ascariasis typically are asymptomatic, although some patients present with fever, nonproductive cough, chest pain, dyspnea, eosinophilia, and eosinophilic pneumonia when worm migration involves the lungs. During migration, adult worms can be identified as they migrate and are coughed up, which can be transmitted by ingesting its larvae. Humans are at risk for hookworm infections when walking barefoot on contaminated soil. The worm travels through the bloodstream to the lungs where it spreads to the bronchi, trachea, and mouth. Once in the mouth, hookworms travel the alimentary canal and attach to the mucosa of the upper small bowel and mature into adult worms. Hookworms attached to the mucosa begin to suck blood from the host; the amount of blood loss is dependent on the number of adult worms involved in the infection.

**Diagnosis**

Analyzing stool samples and examining patients for adult worms emerging from the mouth, nose, or
Identifying whipworm in the colon using an air-contrast barium enema examination enables visualization of wavy radiolucent outlines of numerous small worms against the air-barium background of the colon and rectum. Furthermore, characteristic uncurled curvilinear patterns or S-shaped configurations of the female worms and the tightly coiled “pinwheel” or “target” pattern of the male worm might be recognizable. The posterior portions of the worms become outlined and are approximately 1 cm long, with the longer, slender anterior two-thirds of the worms lying uncoated by barium within the colonic mucosa (see Figure 16).

In patients with chronic and severe cases of hookworm disease, chest radiographs show a mild to moderate generalized cardiac enlargement caused by profound anemia and hypoproteinemia. General radiography also can be used to identify initial migration patterns of hookworms, with radiographs of the feet and ankles demonstrating evidence of hookworm infection (see Figure 17). Most patients with hookworm infection do not demonstrate radiographic abnormality on barium examination of the upper gastrointestinal tract; however, small bowel abnormalities can be found in 60% of hosts, with changes being proportional to the disease burden. For example, irregularities have been observed in the mucosal folds of the jejunum resulting in 2 to 3 times the thickness of healthy tissue. More advanced infections show increased tone in several loops, narrowing of the lumen, and vigorous peristalsis appearing to be in constant motion.

Treatment

Soil-transmitted helminths can be controlled and eliminated by drugs called benzimidazoles, which interfere with the worm’s cellular energy metabolism. The 2 main drugs used to treat ascariasis, whipworm, and hookworm infections are mebendazole and albendazole. Using these drugs is known as deworming and is not limited to treating symptomatic infections; it also is part of large-scale prevention effort in children in endemic areas, often paired with immunization campaigns for added effectiveness.

Dengue

Epidemiology

Dengue is the second most common vector-borne disease in humans after malaria. A member of the genus **Aedes**, dengue is transmitted primarily by Aedes mosquitoes. Dengue occurs year-round in tropical and subtropical areas, and is spread by the bite of infected mosquitoes. Symptoms include fever, headache, muscle and joint pain, and gastrointestinal symptoms. In severe cases, dengue can cause dengue hemorrhagic fever, which can be fatal if not treated promptly. Prevention involves avoiding mosquito bites and treating symptomatic cases. Treatment typically involves supportive care, as there is no specific antiviral treatment for dengue.
Dengue hemorrhagic fever occurs each year. The disease prevalence is attributed to climatic factors, travel, and urbanization. In the past 20 years, severe epidemics of dengue hemorrhagic fever have occurred in East Africa, Sri Lanka, and Latin America. Despite having this knowledge, it has been difficult to pinpoint specific locations at greatest risk considering that the virus has been detected in 128 countries in endemic regions.

Chikungunya is another virus recognized as an NTD, but it is primarily endemic to Asia and Africa. Although it has spread to much of the Americas region as recently as 2013, relatively speaking, it has a low impact in Latin America.

**Pathophysiology**

During the rainy season in endemic regions, the breeding of *Aedes* mosquitoes is abundant. Poor water management in these areas often is coupled with a population uneducated about mosquitoes’ breeding and mosquito bite protection. *Aedes* mosquitoes spend their lifetime near a single location, traveling an average of 400 meters. This means that infected humans, rather than the mosquitoes, are the primary reason the virus spreads between communities.

The virus has 4 distinct serotypes (DEN-1, DEN-2, DEN-3, and DEN-4), and the severity of the dengue...
burden is dependent on the number of serotypes with which an individual becomes infected. The virus circulates in the blood of an infected host for 2 to 7 days over which time the host might begin to develop symptoms. For the disease to spread among people, the mosquito must feed on a person during this period. Then it must bite another person after an 8-day to 12-day incubation period.

**Clinical Manifestations**

Dengue infection can range from being asymptomatic to causing severe hemorrhagic fever or fatal shock (dengue shock syndrome). More than half of infected children are asymptomatic, whereas in adults the illness is more severe and begins more suddenly. After an incubation period of 4 to 5 days, infected individuals experience a sudden onset of "breakbone" fever including chills, aching of the head, back, and extremities accompanied by sore throat, prostration, and malaise. After 3 to 4 days of infection, a maculopapular rash sparing the palms and soles appears in more than 50% of cases.

Dengue hemorrhagic fever typically occurs in secondary infections and in infections with DEN-2. Signs of hemorrhage appear in the first few days of infection and include ecchymoses (nontraumatic bruises), gastrointestinal bleeding, and epistaxis. Dengue shock syndrome is indicated when acute fever, hemorrhagic manifestations, pleural effusions, and ascites occur. Other indicators of dengue shock syndrome include continuous abdominal pain with vomiting, mucosal bleeding, a decrease in consciousness, rash, conjunctival congestion, and hypothermia.

**Diagnosis**

Serological findings can be useful in dengue diagnosis including the nonstructural protein-1 antigen test, measurement of glycoprotein levels, and immunoglobulin G and M tests. During the fifth and sixth day of febrile illness from this disease, hemagglutination inhibition antibodies begin to appear at detectable levels.

Medical imaging of mild dengue syndromes does not reveal specific abnormalities. In severe cases, however, abdominal imaging might display the presence of associated hepatomegaly, and chest radiographs can show pleural effusions within the first week of infection. When advanced imaging is available to providers, CT scans without contrast can detect intracranial bleeding or cerebral edema from dengue hemorrhagic fever.

Ultrasoundography can show thickening of the gallbladder wall and, in some patients, ascites. A correlation exists between the severity of the illness and increasing thickness of the gallbladder wall, with 93% of patients with severe cases of dengue displaying gallbladder wall thickness exceeding 3 mm. When there is ascites, the gallbladder wall is significantly thicker than in patients without intraperitoneal fluid (see Figure 18). Sonographic findings of fluid collection typically can occur in the perirenal and pararenal, hepatic and splenic subcapsular, and pericardial regions. In addition, ultrasonography can demonstrate evidence of pancreatic enlargement and hepatosplenomegaly, with an alteration in the normal liver echo texture caused by intraparenchymal and subcapsular hemorrhages.

Although serology is used to confirm dengue fever, ultrasonography can be used to assess the infection’s...
severity and often serves as a catalyst to disease management when serology testing is unavailable. The severity of dengue infection is linked directly to platelet count, with patients demonstrating all sonographic features associated with dengue likely having platelet counts less than 40,000/µL and potentially requiring blood transfusion. In addition, sonographic features also have a direct relationship with the degree of thrombocytopenia affecting dengue patients.95

**Treatment**

No pharmaceutical treatment for dengue infections is available. A person experiencing associated symptoms should use analgesics with acetaminophen and avoid use of aspirin-based medications because they can compound bleeding issues. Rest and hydration are recommended until a physician can be consulted, but worsening symptoms, such as vomiting and severe abdominal pain within 24 hours of fever decline, require emergent evaluation. In-hospital treatment involves fluid volume support, blood products, vasopressor agents, acetaminophen, and endoscopic therapy to assist in managing gastrointestinal hemorrhage.91

Recovery from infection by a dengue virus results in lifelong immunity against that particular virus serotype; however this immunity provides only partial and transient protection against subsequent infection by the 3 other serotypes. In addition, studies show that sequential infection increases the risk of developing a severe dengue infection.97

**Conclusion**

With WHO’s official recognition of the 17 global NTDs and the 66th World Health Assembly’s 2013 adoption of resolution WHA66.12 calling for intensified, integrated measures and planned investments to improve the health and social well-being of populations affected by NTDs, member states are focusing increased attention on these diseases. Although the majority of countries endemic for NTDs within the Americas are in Central and South America, these conditions exist in the Caribbean and the southern portions of North America including the states bordering the U.S. Gulf Coast. For this reason, it is important for radiologic technologists to understand the role medical imaging plays in initial disease diagnosis and confirmation, as well as recognize the radiologic manifestations of the NTDs specific to the Americas region.

Reviewing a patient’s history for recent travel to an endemic country can provide insight into symptoms that initially might seem to be benign. Performing a basic physical examination for signs of disease can trigger a need for laboratory analyses and imaging assessment. Immunosorbent assays, such as ELISA, as well as stool sample testing often can lead directly to diagnosis when an NTD is being considered. When such technology is unavailable, an understanding of the basic modes of transmission for each NTD, related pathophysiology, and common clinical manifestations can aid in selecting the appropriate imaging modality and technical parameters necessary to make an accurate diagnosis.

In Latin America and the Caribbean, ultrasonography has been the most widely used imaging modality given its low cost, noninvasiveness, and portability; however, this modality can be limited by user-dependency and considerable interobserver variability. Ultrasonography generally is used for screening purposes to identify nonspecific evidence of parasitic presence and invasion. In urban settings, CT and MR technology are more likely to be available. These high-resolution imaging techniques, often with the aid of contrast enhancement, can help characterize parasitic lesions and offer advanced assessment of organ involvement and damage.

It also is important to recognize the need for greater investment into radiology outreach initiatives within endemic regions of the Americas to control and reverse the spread of diseases at their points of origin. Although an “imaging gap” continues to exist between developed and developing countries with regard to access to reliable imaging services and radiology-specific training, the presence of NTDs in endemic regions of the Americas remains a persistent risk to the resource-rich countries that border them.
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1. Chagas disease is caused by the parasite _______ and is transmitted by triatomine bugs.
   a. Giardia lamblia
   b. Trypanosoma cruzi
   c. Entamoeba histolytica
   d. Plasmodium knowlesi

2. Medical imaging of Chagas disease can reveal:
   1. cardiomyopathy.
   2. calcifications in the apical vasculature.
   3. delayed esophageal motility.
   a. 1 and 2
   b. 1 and 3
   c. 2 and 3
   d. 1, 2, and 3

3. Cysticercosis infections are caused by:
   a. helminths.
   b. filarial worms.
   c. flatworms.
   d. mosquitoes.

4. The predominant means of spreading cysticercosis infections is:
   a. person to person.
   b. via environmental sources.
   c. animal to person.
   d. insect to person.

5. In regions where computed tomography (CT) and magnetic resonance (MR) imaging are available, these scans can provide information on:
   1. morphology and localization of cysticercosis cysts.
   2. stage of cysts.
   3. inflammation.
   a. 1 and 2
   b. 1 and 3
   c. 2 and 3
   d. 1, 2, and 3
6. The majority (50%-70%) of echinococcosis cysts develop in the liver.
   a. true
   b. false

7. A patient presenting with a cyst-like mass and a history of recent exposure to sheepdogs while in an area in which *Echinococcus granulosus* is endemic is suggestive of:
   a. Chagas disease.
   b. cystic echinococcosis.
   c. neurocysticercosis.
   d. paragonimiasis.

8. All of the following are descriptions of Gharbi classification type for echinococcosis cysts, except:
   a. pure fluid collection in the cyst.
   b. fluid collection with septa.
   c. reflecting thick walls.
   d. multiple cysts with calcified walls.

9. The most common site of a paragonimiasis infection is the:
   a. heart.
   b. lung.
   c. brain.
   d. extremities.

10. Chest radiographs of people suspected of having a paragonimiasis infection show abnormalities of the lungs or pleura similar to tuberculosis including all of the following except:
    a. cavities.
    b. fibrosis.
    c. infiltrates.
    d. “air bubble” sign.

11. Which is the most characteristic radiologic sign of the mature stage of paragonimiasis and represents worms attached to the wall of a cyst?
   a. air bubble
   b. Cumbo
   c. solar eclipse
   d. double-arch

12. The characteristic convoluted channels of fascioliasis are less identifiable on ultrasonography, and therefore, ______ is the preferred imaging modality for the hepatic form of this disease.
   a. CT
   b. MR
   c. positron emission tomography
   d. radiography

13. Changes in skin thickness, pigmentation, edema, and scarring of the epidermis are key indicators of:
   a. dengue.
   b. fascioliasis.
   c. onchoceriasis.
   d. schistosomiasis.

14. Which modality is being used increasingly in the developing world to diagnose onchocerciasis?
   a. ultrasonography
   b. radiography
   c. CT
   d. MR

15. This disease is considered the most lethal of all neglected tropical diseases leaving many chronically ill and causing 100 000 deaths annually.
   a. onchocerciasis
   b. schistosomiasis
   c. dengue
   d. chorioretinitis
16. Which imaging modality might be more sensitive to show disease progression, stage, and response to therapy for *Schistosoma mansoni* infections?
   a. CT
   b. MR
   c. radiography
   d. ultrasonography

17. Once ______ is transmitted to a host, the adult form can live in the body for 1 to 2 years, and grow as long as 40 cm with the thickness of a pencil.
   a. *Ascaris*
   b. *Schistosoma mansoni*
   c. *Simulium damnosum*
   d. *Trypanosoma cruzi*

18. Heavy infestations of ______ can involve masses of worms that can cause intestinal obstruction, volvulus, intussusception, or death.
   a. *Ascaris*
   b. *Fasciola hepaticus*
   c. *Onchocera volvulus*
   d. *Schistosoma japonicum*

19. Identifying whipworm in the colon using an air-contrast barium enema enables visualization of which of the following?
   1. radiolucent outlines of small worms
   2. S-shaped configurations of female worms
   3. tightly coiled male worms
   a. 1 and 2
   b. 1 and 3
   c. 2 and 3
   d. 1, 2, and 3

20. *Aedes aegypti* mosquitoes transmit all of the following viruses except:
   a. malaria.
   b. dengue.
   c. chikungunya.
   d. yellow fever.

21. Ultrasonography can show which signs of dengue fever?
   1. thickening of the wall of the gallbladder
   2. ascites
   3. kidney stones
   a. 1 and 2
   b. 1 and 3
   c. 2 and 3
   d. 1, 2, and 3

22. Recommendations to treat dengue symptoms include:
   1. aspirin-based medications.
   2. rest.
   3. hydration.
   a. 1 and 2
   b. 1 and 3
   c. 2 and 3
   d. 1, 2, and 3

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