Endemic Thoracic Infections in Latin America and the Caribbean

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KEYWORDS

• Chagas disease • Malaria • Amebiasis • Echinococcosis • Cysticercosis • Schistosomiasis • Paragonimiasis • Ascariasis

KEY POINTS

- Chronic Chagas cardiomyopathy is the most common and serious complication of Chagas disease, and the most common cause of mortality.
- Acute lung injury and adult respiratory distress syndrome are the most severe pulmonary manifestations of malaria.
- Pleuropulmonary amebiasis occur when a subcapsular amebic liver abscess ruptures though the diaphragm into the thoracic cavity.
- After the liver, the lung is the second most commonly affected organ in echinococcosis.
- Pulmonary arterial hypertension is a common complication of schistosomiasis.
- Strongyloides hyperinfection syndrome is typically seen in immunosuppressed patients.
- Imaging findings in acute paracoccidioidomycosis resembles those of primary tuberculosis infection with the combination of airspace disease and lymphadenopathy.

INTRODUCTION

Socioeconomic inequality, poor hygiene, poor sanitation, and lack of access to safe drinking water pose significant public health challenges in Latin America and the Caribbean (LAC) region, where infectious diseases continue to cause significant morbidity, disability, and mortality. Major regional health challenges include limited access to health services, lack of resources, and poor distribution of the existing ones. Diarrheal diseases, along with acute respiratory infections, remain a significant cause of infant and child mortality. Parasitic diseases in particular, which affect more than 1.5 billion people worldwide, are closely associated with income inequality and deficient sanitation. Many of these conditions, which still have significant impact in LAC, are part of the Neglected Infectious Disease (NID) initiative by the World Health Organization (WHO). Ten percent of the global burden of NID occur in LAC, where 17% of the population in rural areas do not have access to safe drinking water, and 37% lack access to sanitation facilities.[1] Developing countries,

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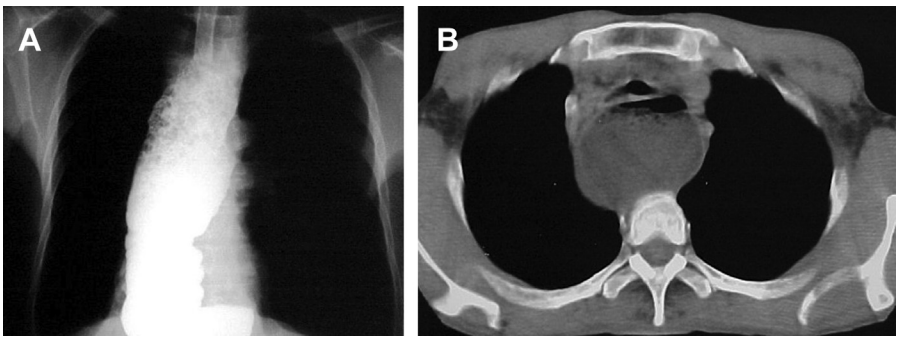

Fig. 1. Chagas megaesophagus. (*A*) Frontal radiograph after oral barium administration demonstrates dilated entire esophagus in a 54-year-old man with Chagas disease. (*B*) Noncontrast CT of the chest reveals an abnormally dilated esophagus with retained fluid content.

including those in LAC, bear 98% of the global burden of infectious parasitic diseases.[2] In this article, the thoracic imaging manifestations of several communicable diseases, including 9 parasitic infection and 1 fungal infection that have significant impact in the LAC region will be reviewed.

PROTOZOA

Chagas Disease (American Trypanosomiasis)

Chagas disease is caused by infection with *Trypanosoma cruzi*, a flagellated protozoan that circulates among hematophagous insect vectors and more than 100 species of mammals, including people. It is considered by some to be the most important parasitic disease in the Western Hemisphere, with a disease burden 7.5 times as great as that of malaria. The disease is endemic in 21 countries, from the southern United States to southern Argentina and Chile, where an estimated 6 million people are currently infected, with 28,000 new cases and 12,000 deaths every year.[3,4] The highest prevalence country is Bolivia, follow by Argentina and El Salvador. Even though vector-borne transmission occurs only in the Americas, because of increasing migration, the disease is increasingly being found in non-endemic areas including Europe and Asia, and has become a global health problem. In the United States 300,000 people live with the infection, with up to 45,000 individuals having undiagnosed Chagas cardiomyopathy.[5] Among Latin America-born patients with newly diagnosed nonischemic cardiomyopathies in Los Angeles and New York, 19% and 13% respectively, had Chagas disease.[6,7]

T cruzi penetrates in the host's cells and multiplies (amastigote form), creating pseudocysts that break, giving rise to an inflammatory reaction with scar formation and fibrosis. The pseudocyst rupture releases new amastigotes that circulate and invade new cells, in a cycle of progressive reinfection that predominantly affects the heart, followed by the esophagus and colon through a mechanism that includes inflammation and denervation.[8]

It is estimated that 30% to 50% of infected people who survive the acute phase progress to chronic Chagas' cardiomyopathy, a highly arrhythmogenic condition, also associated with dilated heart and congestive heart failure, which may develop thromboembolic complications such as stroke and systemic embolism.[9]

Chagas gastrointestinal (GI) involvement is less common (14%), and predominantly affects the esophagus and colon, resulting in visceral dilation from intramural neuronal damage with megaesophagus and megacolon (**Fig. 1**). This complication has significant regional geographic variation, and predominantly presents below the equatorial line, in the southernmost part of South America.[8]

Chronic Chagas cardiomyopathy (CCC) is the most common and serious complication of Chagas disease, and the most common cause of mortality in affected patients. CCC develops after several years or even decades of the indeterminate form of the disease, which is typically asymptomatic. The typical morphologic pattern is a dilated cardiomyopathy, with predominant fibrosis in the apical and posterior wall of the left ventricle, and electric conduction abnormalities with end result of abnormal myocardial contraction. Impaired left ventricular (LV) function with a reduced ejection fraction (<30%), segmental or global LV wall motion abnormalities, LV aneurysm, increased LV diastolic dimension, and cardiomegaly are important independent predictors of mortality.[10]

Contrast-enhanced cardiac magnetic resonance (CMR), in addition to precisely evaluating cardiac morphology and function, may also demonstrate myocardial scar of fibrosis when present. In a substantial number of patients, this

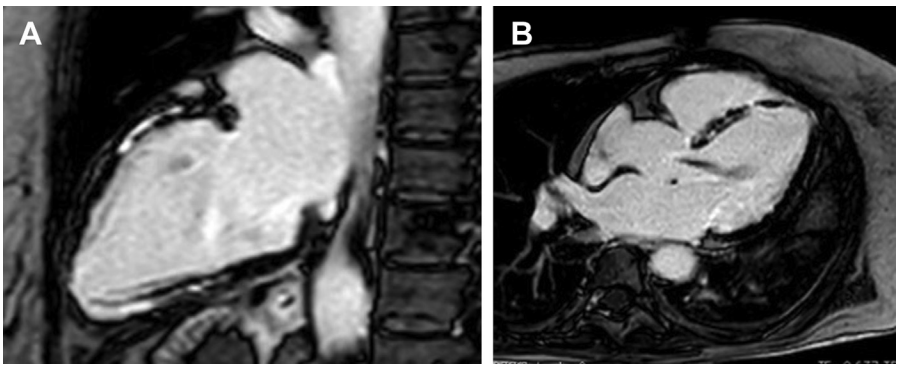

Fig. 2. Chagas cardiomyopathy. (*A*) Cardiac magnetic resonance vertical long axis image after contrast injection shows multiple areas of delayed gadolinium enhancement in a nonvascular distribution in the anterior and inferior wall of the left ventricle. (*B*) Four-chamber view in a different patient. Patchy areas of delayed enhancement are seen in the interventricular septum and apical left ventricle.

allows for risk stratification and robust prognostic evaluation. The prevalence of delayed enhancement in CCC (25%-90%) varies depending on the severity of the disease, with higher prevalence in patients with ventricular tachycardia and LV dysfunction Prevalence also tends to be higher in males.[11,12]

In CCC patients, CMR wall motion abnormalities and delayed enhancement are more commonly seen in the inferolateral and apical segments, with delayed enhancement distribution that may be transmural (36%-50%), subendocardial (27%), subepicardial (12%-23%), or midwall (14%-35%), not infrequently mimicking ischemic and nonischemic cardiomyopathies (**Fig. 2**).[11,12] CMR is particularly indicated in patients with severe ventricular arrhythmias in order to quantify the extent of myocardial fibrosis and the risk of sudden death. This information may influence the decision to place an implantable defibrillator.[13]

MALARIA

Human malaria results from the protozoal infection by 1 of the 5 *Plasmodium* species, transmitted by the *Anopheles* mosquito. More than 80% of malaria cases in South America come from the Amazon rain forest (Venezuela [30%], Brazil [24%], Peru [19%] and Colombia [10%]), where the disease is endemic, with more than half of the cases caused by *Plasmodium vivax.* The incidence of *Plasmodium falciparum* malaria, the second most common type in the region, has decreased in recent years, except in parts of the Pacific coast in Colombia, where *P falciparum* is still the dominant type.[14] According to the WHO, there were 229 million cases of malaria in 2019, with an estimated 409,000 deaths, most in the Sub-Saharan Africa and India (96%).[15]

Malaria infection begins when an infected female *Anopheles* mosquito bites a person, injecting saliva infected with *Plasmodium* sporozoites into the bloodstream, which passes quickly into the human liver, where the sporozoites multiply. The parasites, in the form of merozoites, are released from the liver cells, travel through the right side of the heart, and reach the lungs. In the bloodstream, the merozoites invade erythrocytes and multiply, invading more red cells. This cycle is repeated, causing recurrent fever each time parasites break free and invade more blood cells. Some of the merozoites in these cells develop into sexual forms (male and female gametocytes) that circulate in the blood stream. When a mosquito bites an infected person, it ingests the gametocytes that mate in the gut of the mosquito to form sporozoites. Human infection continues when the mosquito bites another person, injecting saliva with the parasite, beginning a new cycle.

Symptoms and severity of malaria vary depending on which of the 4 parasite species is the cause (or combination thereof, since a patient may contract more than 1 type of malaria at a time), with an incubation period of 10 to 15 days (but may last as long as few months), after which time, malaise, episodic fever, rattling chills, and muscular spasms manifest.

Acute lung injury and adult respiratory distress syndrome (ARDS) are the most severe pulmonary manifestations of malaria. They occur in up to 25% of patients with severe falciparum and vivax malaria, are associated with grave prognosis, and are responsible for up to 40% of malaria deaths. The exact mechanism is not entirely known, but endothelial cell injury and necrosis and altered alveolar capillary permeability are important factors. The histopathology reveals pulmonary edema, intra-alveolar hemorrhage, and

hyaline membrane formation.[16] The radiologic manifestations are those of a noncardiogenic pulmonary edema and ARDS with bilateral interstitial and alveolar opacities, which may progress to airspace consolidation associated with variable degree of pleural effusion. Interlobular septal thickening, ground-glass opacities, and small pleural effusion are better appreciated on computed tomography (CT) (**Fig. 3**) (see also article by Ryzdak and colleagues in this issue).[17,18] Differentiation from multilobar pneumonia may be a challenge in these patients with respiratory symptoms, fever, and multifocal pulmonary radiographic abnormalities.[19]

AMEBIASIS

Amebiasis is the parasitic disease caused by the pseudopod forming nonflagellated protozoan *Entamoeba histolytica* (*E histolytica*). It is the third leading cause of death from a parasitic disease worldwide after malaria and schistosomiasis, with an estimated death toll between 40,000 to 100,000 people annually.[20,21]

Amebic colitis, the most common clinical manifestation, is a leading cause of severe diarrhea worldwide and among the leading causes of death in children. Endemic in developing countries, particularly in tropical and subtropical regions, the seroprevalence of *E histolytica* in some rural communities of Latin America with inadequate hygiene is above 40%. In the United States, the incidence of amebiasis is low, with most cases seen in immigrants or returning travelers from endemic regions. Three different types of *Entamoeba* that can cause asymptomatic infestation have been described: *E dispar, E moshkovskii,* and*E bangladeshi*, are morphologically identical to *E hystolytica,* and can potentially infect the intestinal mucosa, but *E histolytica* is the most invasive and responsible of nearly all cases of human amebiasis.[21–23]

E histolytica has cytolytic, phagocytic, and proteolytic capabilities that allow invasion through the mucosa and submucosal tissues and even into the portal circulation. Up to 10% of asymptomatic individuals infected with *E histolytica* develop symptomatic disease during the following year, typically amebic colitis with bloody diarrhea and abdominal pain.[20]

Infection with *E histolytica,* typically from ingestion of quadrinucleated cyst from fecally contaminated food or water (fecal-oral transmission), may be asymptomatic or may cause dysentery or extraintestinal disease, with amebic liver abscess being the most common extraintestinal location. It typically occurs in young men between 20 and 40 years old. Pleuropulmonary amebiasis can occur as a complication when the liver abscess ruptures into the thoracic cavity, which is more likely to occur when the abscess is in a subcapsular location. Not surprisingly, most pleuropulmonary complications of amebic liver abscess occur in the right hemithorax (90%). Imaging manifestations include elevation of the right hemidiaphragm, pleural effusion, parenchymal consolidation in the right lower lobe and/or right middle lobe, and occasionally hydropneumothorax if bronchopleural fistula is present. A different location may result in case of hematogenous or lymphatic spread. Intrapericardial rupture may occur, particularly with left hepatic lobe abscess, but is rare (2%). CT may demonstrate abnormal appearance of the hemidiaphragm with thickening and transdiaphragmatic extension of the abscess, with a characteristic hourglass configuration (**Fig. 4**).[24–26] Amebic liver abscess are commonly unilocular (70%), occasionally with internal septations (30%), and demonstrate rim peripheral enhancement after contrast injection on CT (target sign).[27] Pulmonary amebiasis may rarely result from hematogenous or lymphatic spread from an intestinal infection or from inhalation of dust containing cysts or trophozoites of *E histolytica* (**Fig. 5**).[28]

Pleuropulmonary amebiasis is a serious disease with significant mortality (5%–16%), especially in patients with poor health or malnutrition, in cases with delayed diagnosis, or with inadequate treatment.

CESTODES

Echinococcosis

Recently, a much-needed international consensus on terminology to be used in the field of Echinococcoses has been published with an agreement on 3 names for the disease caused by *Echinococcus spp*: cystic echinococcosis (CE), caused by *E granulosus sensu lato*; alveolar echinococcosis (AE) caused by *E multilocularis*, and neotropical echinococcosis (NE) caused by *E Vogeli* and *E Oligarthra*. Confusing terms like hydatidosis, polycistic echinococcosis, and other, that have been used for centuries are considered to be either confusing or entirely incorrect and should be avoided. Hydatid disease, if used should be only in reference to CE. Because DNA sequencing has identified several subtypes of *E granulosus*, the current recommendation is to add sensu lato (meaning in a wider sense) when in reference to them as a group and sensu stricto when in reference to one subtype in particular.[29]

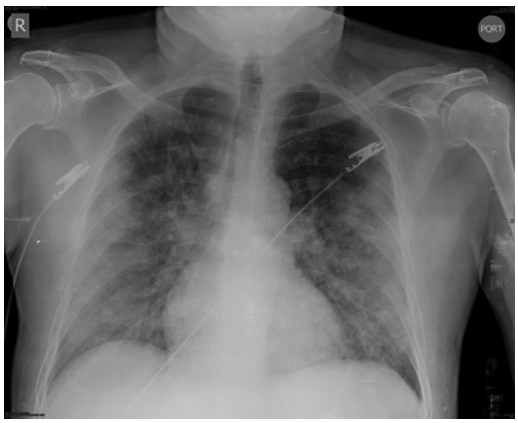

Fig. 3. Acute malaria in a 21-year-old man with plasmodium vivax infection with respiratory distress, thrombocytopenia, and hemoptysis. Chest radiograph demonstrates multifocal ground-glass opacities in the bilateral midlung zones. (Courtesy Tatiana Suarez MD, Medellin, Colombia)

Echinococcosis is a neglected parasitic zoonosis secondary to infection by the larval stage of the cestode (tapeworm) of the genus *Echinococcus.* CE and AE are the most important clinical forms because of their more extensive geographic distribution, with a substantial health and economic burden. CE is found in Africa, Europe, Asia, the Middle East, North America, Central America, and South America. AE has a worldwide distribution, with higher distribution in northern latitudes of Europe, Asia, and North America. NE is endemic and limited to certain areas of Central America and South America.[30] *E granulosus* is endemic in Argentina, Chile, Peru, Uruguay, and southern Brazil, with around 5000 new cases of CE reported annually and 2.9% mortality.[31,32] People are considered to be aberrant intermediate hosts, who become infected by the ingestion of food contaminated with eggs (fecal-oral route), which in the intestine release onchospheres that penetrate the intestinal wall to reach the vasculature and though the circulatory system reach different end organs (especially liver and lungs), where thick-wall multilayer unilocular cysts develop. NE secondary to *Echinococcus vogeli* causes a multicystic disease, whereas *E Oligarthra* produces a unilocular cystic disease.[33]

Initial infection is always asymptomatic. Later, clinical manifestations vary depending on the number and size of the cystic lesions and the location within the affected organ, with most small (<7 cm) encapsulated lesions remaining asymptomatic. Cysts may slowly grow and become symptomatic with abdominal pain and hepatobiliary manifestation caused by mass effect in case of liver lesions and pleuropulmonary manifestation (cough, pain, hemoptysis) in case of pulmonary infection.

The lung is the second most commonly affected organ (20%-30%) after the liver (60%–70%). Other locations (eg, peritoneum, kidney, or brain) are less common. Extrapulmonary intrathoracic disease involving the pleura, mediastinum, heart and great vessels, diaphragm, and chest wall may also occur.[34–36]

A hydatid cyst consists of 3 layers with a variable amount of fluid inside: the pericyst (outermost layer), the exocyst (middle layer), and the endocyst (the innermost layer). In CE, lung involvement typically reveals a single cyst (70%-85%) 1 to 20 cm in diameter, with a predominant lower lobe distribution. Uncomplicated cysts are smooth-wall homogeneous low-density round or oval lesions. Complicated cysts (ruptured or infected) reveal separation between the pericyst and endocyst, creating a variety of imaging signs, including air crescent sign, meniscus sign, inverse crescent sign, air bubble sign, and signet ring sign.

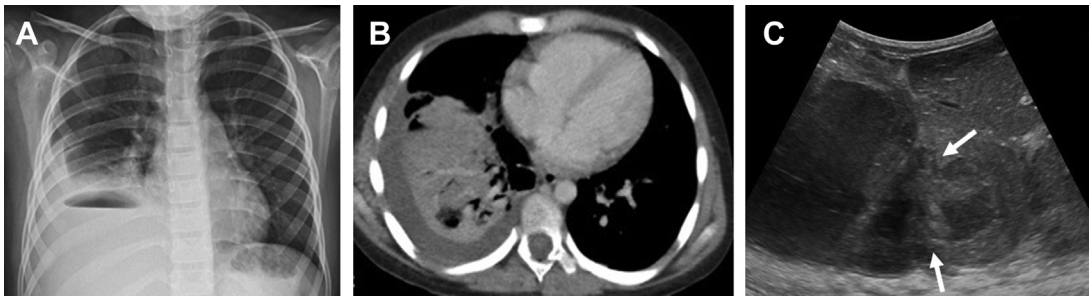

Fig. 4. Pleuropulmonary amebiasis in an11 year old boy. (*A*) Frontal chest radiograph shows an air fluid level in the right upper quadrant of the abdomen, with a right-side pleural effusion and pulmonary opacity. (*B*) CT shows a consolidative opacity in the right lung surrounded by pleural fluid. (*C*) Right upper quadrant ultrasound, sagittal view reveals the diaphragmatic rupture (*arrows*) with transdiaphragmatic extension of the amebic liver abscess.

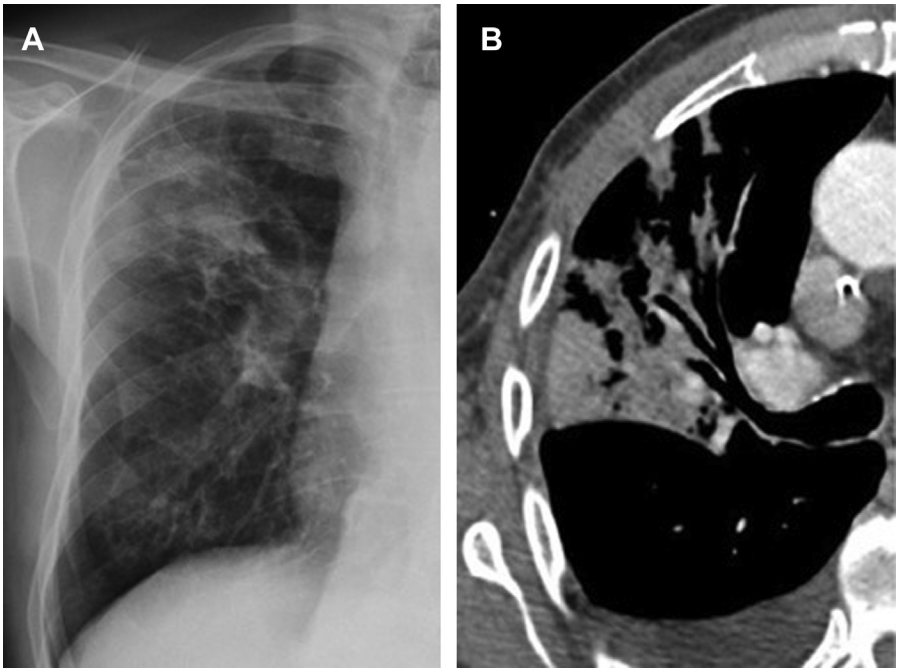

Fig. 5. Pleuropulmonary amebiasis secondary to hematogenous spread. (*A*) Patchy consolidative opacities are appreciated on the chest radiography. (*B*) Contrast-enhanced chest CT soft tissue window demonstrates airspace consolidation and layering right side pleural effusion in a patient with proven amebic pneumonia, and colitis without liver abscess.

Complete rupture is suspected when the cyst is seen connected to a bronchus, also with several imaging signs having been reported, including cumbo sign, whirl sign, and waterlily sign. The air crescent sign or meniscus sign, for example, refers to a thin crescent of air seen between the pericyst and endocyst, whereas the waterlily sign refers to the appearance of a complete collapse of the endocyst, floating in the residual fluid. High-density content and peripheral enhancement suggest superimposed bacterial infection. Larger cysts may present additional abnormalities like atelectasis, bronchiectasis and pleural effusion (**Fig. 6**).[35,37,38]

Cardiac involvement is a rare manifestation of CE (<2%), and occurs through the coronary artery circulation, with LV involvement being the most common location, followed by the right ventricle (60% and 15% respectively). Pericardial disease is seen in less than 10% of the cases.[39] Similar to other organs, the cysts may have variable presentation as single or multiple, with variable wall thickness, rarely becoming solid lesions, difficult to differentiate from a cardiac tumor.[40]

Involvement of the great vessels may occur, either as extrinsic compression from a large mediastinal or pleuropulmonary lesion, or as an infiltrative vascular wall lesion with potential risk for thrombosis, erosion, or rupture (**Fig. 7**).

CYSTICERCOSIS

Cysticercosis, considered by the WHO an NTD, is caused by infection with the larval stage of the pork tapeworm *Taenia solium*, acquired through fecal-oral contamination with eggs from tapeworm carriers. The invasive oncospheres in the eggs cross the small bowel wall, reaching the bloodstream to be carried to the brain, muscles, and other organs where they encyst as cysticerci.[41] Cysticercosis is endemic in Latin America, India, Asia, and Africa in rural and urban areas with poor sanitation and is not uncommonly seen outside of endemic areas in communities with large number of immigrants. Areas of high prevalence are found in Mexico, Bolivia, Ecuador, Peru, Honduras, and Guatemala.[42]

Neurocysticercosis is the most serious clinical form of the disease, manifesting as epilepsy, intracranial hypertension, and hydrocephalus. Nearly 15 million people are estimated to have neurocysticercosis in Latin America and the Caribbean. Pulmonary and heart infection are rare despite the

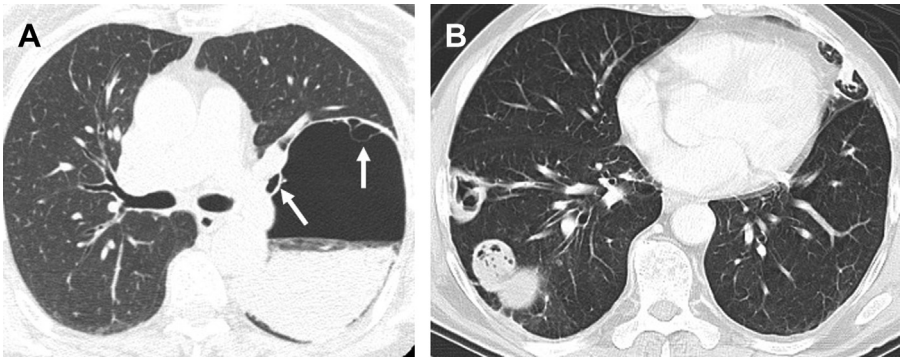

Fig. 6. Pulmonary cystic echinococcosis in 2 different patients. (*A*) CT chest axial image shows a large left side intrapulmonary cyst containing a large air fluid level in a 65-year-old woman. Smaller cysts are visible in the periphery of the dominant cyst's wall (*arrows*). (*B*) CT chest in a 42-year-old man shows multiple air and fluid containing intrapulmonary cysts in the right lower lobe.

high prevalence of cysticercosis in endemic areas, and typically manifest as solitary or multiple nodules in the lung parenchyma, or in the myocardium around 1 cm in diameter. In such cases the possibility of cysticercosis should be considered, in particular if associated with chest wall soft tissue and subcutaneous nodules and brain involvement (**Fig. 8**).[43,44]

TREMATODES

Schistosomiasis

Schistosomiasis or bilharziasis is a parasitic infection caused by trematode parasites of the genus *Schistosoma*, endemic in tropical and subtropical regions. The schistosomes are a group of blood trematodes (fluke worms), with 3 main species infecting people: *Schistosoma haematobium* (Africa, eastern Mediterranean region, and the Arabian peninsula), *S japonicum* (China, the Philippines, and Indonesia), and *Schistosoma mansoni* (Africa, Arabian peninsula, and South America). The females produce hundreds to thousands of eggs per day, with each egg containing a ciliated larva. Infection occurs in fresh water containing larval forms (cercariae). Snails become infected from eggs excreted in human feces or urine. The larvae develop in water snails (intermediate host). The cercariae penetrates the skin of the individual in contact with the water. The parasite migrates in the blood via the lungs to the liver, where they transform into young worms that mature and migrate to the perivesical or mesenteric destination, where they colonize blood vessels for years, producing *Schistosoma* eggs daily, which are eliminated with urine or feces, depending on the species, for the cycle to start again.[45,46] *S mansoni* is found in Latin America primarily in Brazil (90%), Venezuela, Suriname, and the Caribbean, where it is endemic mainly in rural

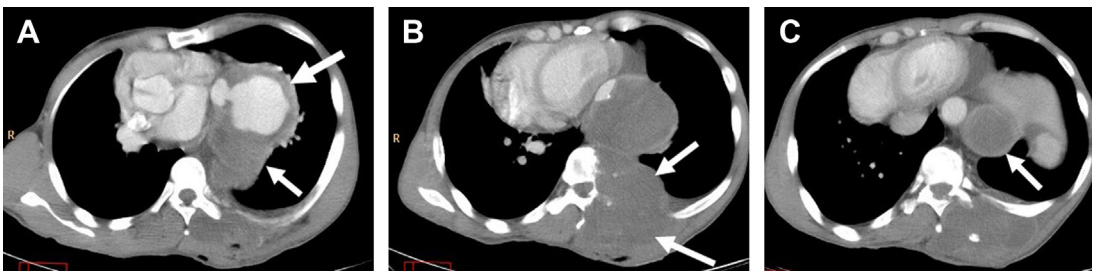

Fig. 7. Cystic echinococcosis with chest wall, mediastinal and aortic involvement. *(A)* Contrast-enhanced chest CT shows an anteriorly displaced aorta adjacent to the heart with a large pseudoaneurysm on the left side of the mid-descending aorta (large *arrow*), and a low-density posterior para-aortic lesion (small *arrow*). *(B)* Axial image at a lower level demonstrates erosive changes on the spine, and posterior chest wall involvement (*arrows*). *(C)* The displaced and compressed aortic lumen is seen adjacent to the lateral wall of the left ventricle. Axial image at the level of the left hemidiaphragm shows the lower aspect of the mediastinal hydatid cyst (*arrow*). (Courtesy Liliana Vega, MD, Mar del Plata, Argentina)

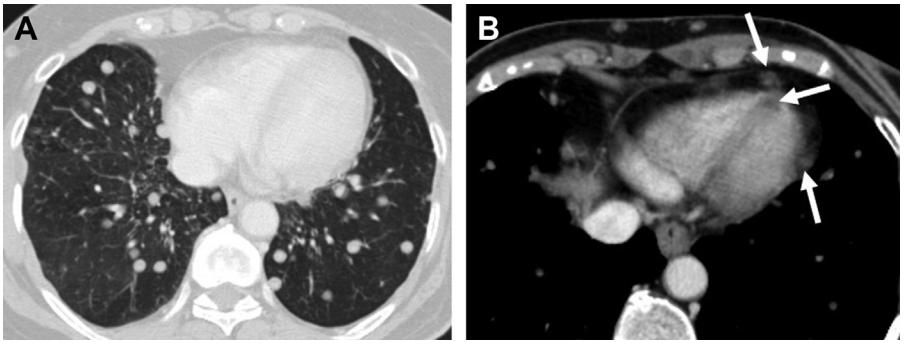

Fig. 8. Chest CT in a female patient with disseminated cysticercosis with pulmonary, cardiac, and soft tissue involvement. (*A*) Lung window axial image shows numerous soft tissue density pulmonary nodules, and subcutaneous nodules in the anterior chest wall. (*B*) Soft tissue window axial image shows additional nodular lesions in the interventricular septum and pericardium (*arrows*). (Courtesy Yashant Aswani, MD.)

areas with poor sanitation. Acute schistosomiasis (Katayama fever) is a systemic hypersensitivity reaction after the primary infection, with patchy pulmonary ground-glass opacities, small pulmonary nodules (2–15 mm) and interlobular septal thickening on imaging examination.[47,48] In chronic infection, the parasites are trapped in tissue, where they induce granulomatous inflammation and fibrosis resulting in 2 main clinical forms: genitourinary and GI schistosomiasis. The fibrotic liver disease derived portal-caval shunting allows ova to leak into the pulmonary capillary bed, resulting in necrotizing and obliterative endarteritis with fibrosis and pulmonary arterial hypertension (PAH), complications seen in as much of 20% of patients with schistosomiasis. In endemic areas with high prevalence of the disease, more than 30% of all cases of PAH are secondary to schistosomiasis.[49,50] Imaging studies may reveal a cardiomegaly with right ventricular enlargement, enlarged pulmonary trunk, and central pulmonary arteries accompanied by a fibrotic liver. No diffuse parenchymal pulmonary disease (eg, fibrosis, emphysema, or interstitial lung disease) or additional cardiovascular abnormality (eg, intracardiac shunt or chronic thromboembolism) is present to account for PAH. Small pulmonary nodules and small patchy consolidations can also be seen in patients with chronic pulmonary involvement (**Fig. 9**).[51,52]

PARAGONIMIASIS

Paragonimiasis is a foodborne zoonotic disease caused by trematodes of the genus *Paragonimus*. The infection develops after the ingestion of raw or insufficiently cooked meat from freshwater crab, crayfish or from a mammalian host (pigs, wild boar) containing the encysted metacercaria of the flatworm. Of the nearly 50 *Paragonimus* species described, 8 can produce disease in humans, with 3 being responsible for most cases: *Plasmodium westermani* in Asia, *Plasmodium mexicanus* in Central and South America with highest

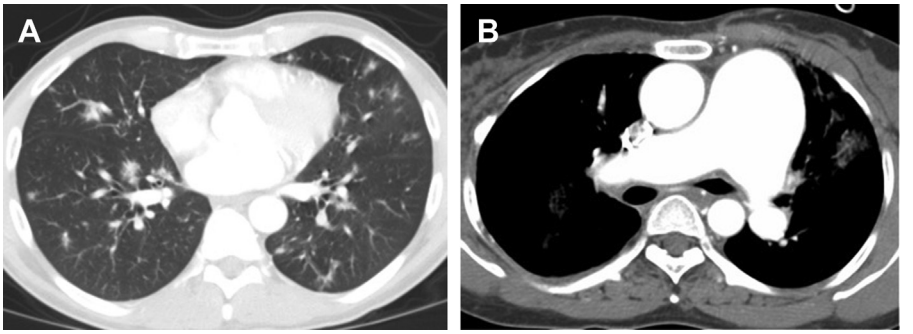

Fig. 9. Acute and chronic schistosomiasis in 2 different patients. (*A*) Acute pulmonary schistosomiasis in a young male. Contrast-enhanced chest CT, lung window axial image reveals small lung nodules with peripheral ground-glass halo scattered in the bilateral lungs. (*B*) Chronic schistosomiasis in a female patient. Contrast-enhanced CT shows pulmonary hypertension with significantly enlarged pulmonary trunk and central pulmonary arteries (*arrows*).

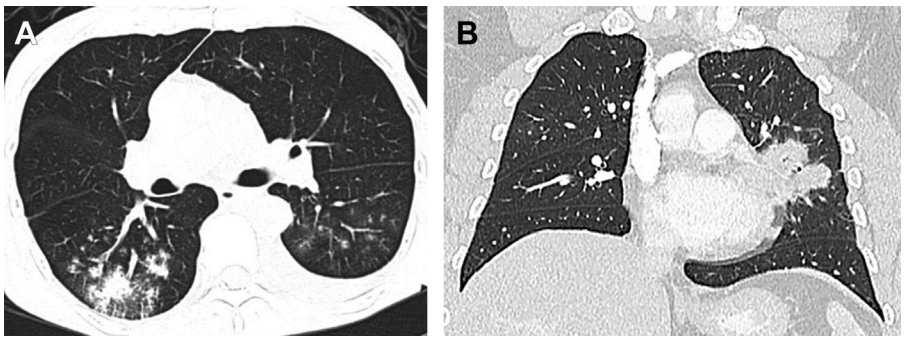

Fig. 10. Acute pulmonary paragonimiasis in 2 different patients. (*A*) Chest CT lung window axial image demonstrates patchy and nodular bibasilar bilateral pulmonary opacities, with interlobular septal thickening in the right lower lobe. (*B*) Coronal reformatted contrast-enhanced chest CT. Lung window image shows patchy rounded opacities in the left upper lobe.

incidence in Ecuador and Peru, and *P. kellicotti* in North America. It is estimated that 1 million people worldwide get infected annually.[53,54]

Paragonimus has a complex life cycle, with 2 intermediate hosts and a definitive mammalian host. After human ingestion, the parasite infective larvae migrate to the peritoneal cavity after penetrating the duodenal wall, ultimately reaching the pleura and lung through the diaphragm. In the lung, the larvae mature into adult flukes, with development of pulmonary cystic cavities.[55] Clinical presentation includes fever, dyspnea, hemoptysis, cough, pleuritic chest pain, and eosinophilic pleural effusion. Abnormalities on imaging examination, which are present in most cases (>90%), vary according to the stage of the disease and geographic distribution. During the transdiaphragmatic and pleural migration, effusion (20%-60%), pneumothorax (5%-17%), and pleural thickening (7%) may occur. Intrapulmonary migration of the worms manifests with airspace consolidation (50%) and linear or bandlike opacities (40%) representing worm migration tracts. Once the parasite ceases migration, lung nodules and thin-walled cysts 1 to 3 cm in diameter appear, some revealing ovoid internal structure from the presence of the worm within. Pulmonary cysts may appear fluid- or air-filled depending on the presence of bronchial communication (**Figs. 10 and 11**).[56–58] Pericardial effusion and omental inflammation are additional findings that have been reported in North American paragonimiasis but are uncommon in Asian paragonimiasis.[59]

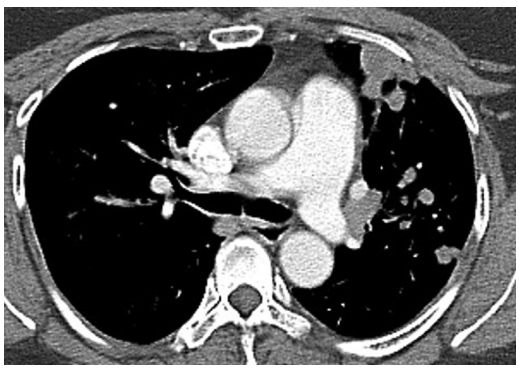

Fig. 11. Chronic pulmonary paragonimiasis in a 44-year-old patient. Contrast-enhanced chest CT axial image shows fluid density cyst scattered throughout the left lung.

NEMATODES

Ascariasis

Ascariasis is the infection produced by the roundworm *Ascaris lumbricoides*, a soil-transmitted helminth with a worldwide distribution in tropical and subtropical areas with poor sanitation and fecal contamination of food and water. Ingested eggs hatch larvae in the intestinal lumen, which are absorbed into the portal circulation reaching the liver initially and later the heart and lung parenchyma through the pulmonary circulation. Larvae are coughed up to the tracheobronchial tree and end swallowed, re-entering the GII tract.[24] Roughly 1 billion people are affected worldwide, with prevalence greater than 20% in several Latin America and Caribbean countries and the highest rates among school-age children.[60,61] Typically during the second week of infection, as the larvae invade the lung, there may be tissue damage and an allergic response. This manifests as respiratory symptoms, which may be associated with eosinophilia in blood with pulmonary radiographic abnormality, also known as Löffler syndrome (in recognition of Dr. Wilhelm Löffler, who described it)[31]

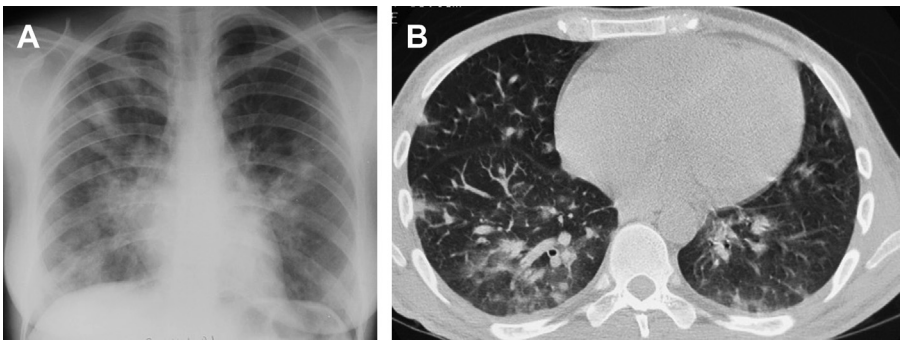

Fig. 12. Pulmonary ascariasis in 2 different patients. (*A*) Frontal chest radiograph shows multifocal patchy ground-glass and nodular bilateral opacities in a parahilar distribution. (*B*) Noncontrast chest CT demonstrates nodular and ground-glass opacities in the bilateral lower lobes.

During this phase, transient patchy nodular or consolidative pulmonary opacities with either unilateral or bilateral distribution may be evident, and typically resolve in about 2 to 4 weeks. Opacities are ground-glass in density, but frank lobar consolidation may occur (**Fig. 12**).[24,25]

In most patients, this stage is asymptomatic; with respiratory manifestation in less than 15% of patients. The severity and magnitude of the pulmonary abnormalities are in part related to the extent of worm burden, but the process is typically transient and self-limited.[62]

STRONGYLOIDIASIS

Strongyloidiasis is a chronic parasitic infection caused by the nematode (roundworm) *Strongyloides stercoralis*, a filariform larva that inhabits the soil and infects people via skin penetration. The parasite is present worldwide in tropical and subtropical regions and remains endemic in the southeastern United States, with between 30 million and 100 million people estimated to be infected worldwide, although estimates as high as 370 million people infected have been reported.[63] Among Latin American countries. the highest prevalence (>20%) is found in Argentina, Ecuador, Venezuela, Peru, and Brazil.[64,65]

After skin penetration, the filariform larvae travel hematogenously to the lungs, reaching the alveolar space, later migrating to the pharynx, where they are swallowed into the proximal small bowel, where they burrow and lay their eggs. In some ways, the life cycle of this parasite is unique. Unlike other soil-transmitted helminths such as hookworm and whipworm, whose eggs do not hatch until they are in the environment, the eggs of *S stercoralis* hatch into larvae in the intestine, allowing permanent cycles of reinfection or autoinfection by which the parasite completes its life cycle within a single host. Most of these larvae will be excreted in the stool, but some of the larvae may mature into filariform larvae and immediately reinfect the host either by burrowing into the intestinal mucosa and bowel wall, or by penetrating the perianal skin.[63] The infection may be entirely asymptomatic or manifest with minimal nonspecific respiratory symptoms. In immunosuppressed patients (eg, acquired immunodeficiency syndrome [AIDS], corticosteroid therapy, or post-transplant immunosuppression therapy) the parasite may undergo uncontrolled proliferation and dissemination spreading more significantly throughout the lungs, and into to multiple organs (eg, skin, liver, kidneys, lymphatics, and brain), in what is known as hyperinfection syndrome. Affected patients may develop intra-alveolar hemorrhage and adult respiratory distress syndrome.[24,66] Imaging studies may be normal in some patients, but more often will reveal small pulmonary nodules and/or interstitial opacities, or a bronchopneumonia pattern of multifocal patchy alveolar parenchymal opacities that may appear to migrate on follow-up examination. Occasionally a lobar consolidation may occur.[67] On CT, ground-glass opacities and interlobular septal thickening in addition to patchy airspace consolidation are common. In case of hyperinfection syndrome, more extensive multifocal ground-glass and consolidative opacities are seen. These appear disseminated throughout both lungs, commonly associated with interlobular septal thickening, and may be associated with small pulmonary nodules and variable amounts of pleural effusion (**Fig. 13**).[63,68]

FUNGAL INFECTION

Paracoccidioidomycosis

Paracoccidioidomycosis (PCM) is a fungal disease caused by the dimorphic fungus of the genus *Paracoccidioides*, endemic in Latin American countries. Of the 2 species identified, *Plasmodium*

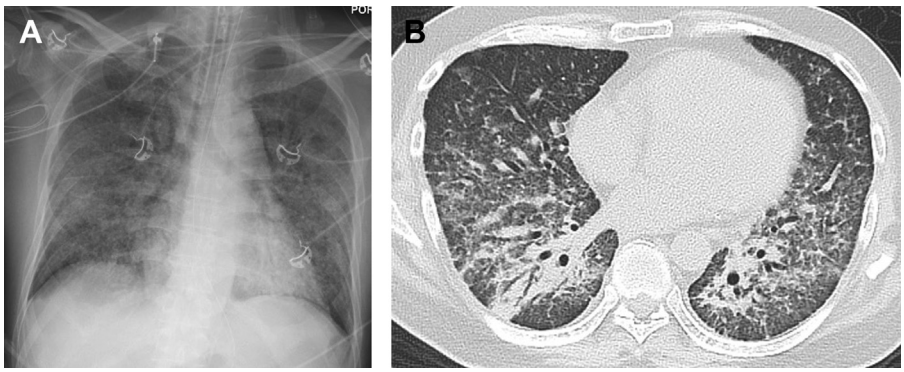

Fig. 13. Strongyloides hyperinfection syndrome in a 47-year-old immunosuppressed man. (*A*) Frontal chest radiograph shows diffuse nodular and ground-glass opacities throughout the bilateral lungs. (*B*) Noncontrast chest CT better demonstrates multifocal interstitial, patchy, and nodular alveolar bilateral pulmonary opacities with interlobular septal thickening.

brasiliensis and *Plasmodium lutzii*, the former is responsible for the majority of clinical human infections.[69]

PCM is the most frequent endemic systemic mycosis in several Latin American countries, with the largest number of cases reported in Brazil, Colombia, Venezuela, and Argentina with smaller endemic areas in Mexico, Ecuador, and Peru. Most cases are in Brazil (80%), with the highest prevalence in midwest and northern parts of the country, where the incidence may be as high as 9.4 cases per 100,000 inhabitants/year. The chronic form of the disease more commonly affects men (75%–95%) between 30 and 60 years old (male:female ratio > 20:1), typically rural workers involved in agricultural activities.[70,71]

Similar to other fungal infections, the lung is the portal of entry for this soil saprophyte. In most cases, the primary pulmonary infection is asymptomatic, with only a small proportion of cases presenting clinical manifestations. A chronic infection from reactivation of a remote primary infection most commonly affects those with history of smoking (90%) and alcoholism.[71]

The acute form (10%-25%) manifests by fever, hepatosplenomegaly, and generalized lymphadenopathy, rarely with pulmonary disease, different from the chronic form of the disease (75%-90%) that commonly affects the lung parenchyma with pulmonary nodules, airspace consolidation, cavitation and chronic scarring, and fibrosis with bronchiectasis and cicatricial emphysema.[72–74]

In the few individuals with symptomatic acute pulmonary disease, the imaging findings resemble those seen in a primary tuberculosis infection with airspace disease, consolidation, and lymphadenopathy. In the chronic stage of the disease, which is more common, the most frequent CT findings include pulmonary opacities with ground-glass attenuation (60%), nodules (50%), cavitation (40%), parenchymal scarring and fibrosis (30%), and areas of cicatricial emphysema (30%-50%),

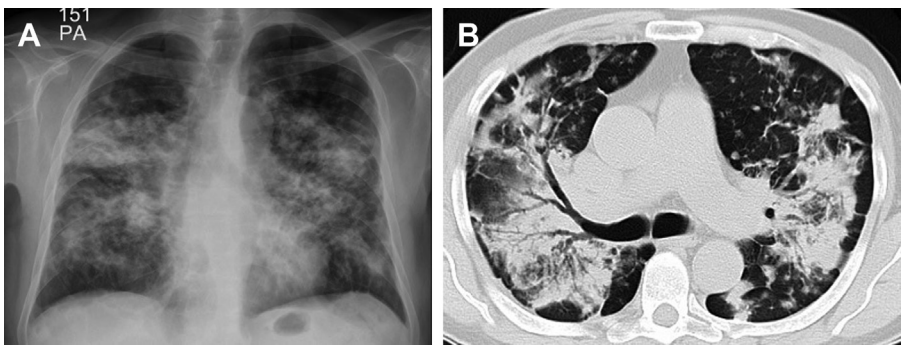

Fig. 14. Paracoccidioidomycosis in a 67-year-old male farmer. (*A*) Frontal chest radiograph shows extensive bilateral ground-glass and consolidative pulmonary opacities. (*B*) Noncontrast chest CT demonstrates air bronchogram within the bilateral areas of air space consolidation and nodular and ground opacities in the bilateral lungs.

often in a predominant peripheral and posterior distribution affecting all lung zones (**Fig. 14**).[75,76] The reverse halo sign (central ground-glass opacity surrounded by denser air–space consolidation in a crescent or ring shape) has also been reported in pulmonary PCM, but is a nonspecific imaging finding that can be seen in several infectious and noninfectious pulmonary pathologies.[77] In the long term, up to 25% of infected patients may develop precapillary pulmonary hypertension, even after declared free of the infection after appropriate antimycotic therapy.[78]

The confirmation of PCM requires the identification of *Paracoccidioides spp* through the examination of fresh sputum or other clinical specimens, such as mucocutaneous lesion, lymph node aspiration, or biopsy.[74]

SUMMARY

Transmissible diarrheal diseases, along with acute respiratory infections, remain a significant cause of morbidity and mortality in developing countries, including Latin America and the Caribbean region where poor sanitation remains a prevalent problem. Contagious and transmissible diseases are particularly affected by societal factors such as education, sociocultural level, income, housing, and access to safe drinking water. Many of the diseases discussed are associated with these deficiencies and disparities, affect the thoracic cavity, and have nonspecific clinical manifestations. Radiologists should be familiar with the epidemiology, pathophysiology, clinical, and imaging manifestations of these entities to contribute to the diagnosis and follow-up of affected patients.

DISCLOSURE

The authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or nonfinancial interest (such as personal or professional relationships, affiliations, knowledge, or beliefs) in the subject matter or materials discussed in this article.

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