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COMMON OPPORTUNISTIC PULMONARY PARASITES IN HIV PATIENTS AND THEIR IMPACT ON ORGAN TRANSPLANTATION

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Abstract

HIV (human immunodeficiency virus) is a virus that attacks the body's immune system, If not treated would lead to AIDS (acquired immunodeficiency syndrome). The HIV infection is acquired from contact with infected blood, semen, or vaginal fluids. Most patients get HIV by having unprotected sex with someone who has HIV. Another common way is by sharing drug needles with HIV infected patient. Most patients develop a pulmonary complication during the history of HIV infection. Lung is the most frequently affected site by the bacterial, fungal, viral and parasitic diseases.

Key words: HIV, Opportunistic parasites, Lung, Human immunity, Organ transplantation, Review.

Introduction

Prior to the era of potent antiretroviral therapy, parasitic pulmonary infections were more commonly seen than they are today. But, the clinicians still need to be aware of presenting symptoms and signs of these uncommon infections, which may still be diagnosed in the immunosuppressed patient with untreated or drug-resistant HIV infection. HIV-related immunosuppression significantly increases the risk for acquiring opportunistic infections due to bacteria, viruses, fungi, and protozoa. These opportunistic infections are a major source of morbidity and mortality in HIV-infected patients. Substantial advances in the prevention of opportunistic infections have been achieved. These strategies involve prophylactic antibiotics, immunizations, and public health measures. The cost effectiveness of preventing infections is variable, ranging from $16,000 per quality adjusted life-year saved for *Pneumocystis carinii* pneumonia to over $300,000 for cytomegalovirus (Freedberg *et al,* 1998). A total of 33 million people were estimated to be living with HIV/AIDS, and more than 37 million had died since the epidemic begin (UNAIDS, 2009). The HIV patients who are unaware of their diagnosis could in one way or another passively infect others or family members.

Review and Discussion

The HIV-patients opportunistic pulmonary parasites were mainly *Toxoplasma gondii*, *Strongyloides stercoralis, Cryptosporidium parvum*, *Entamoeba histolytica*, echinococcosis/hydatidosis, paragonimiasis and others, apart from *Aspergillus, Cryptococcus, Histolasma*, *Microsporidium* Coccidioides, Cytomegalovirus, and the clinical manifestations of pneumocystis found elsewhere (Skalski and Limper, 2016).

Toxoplasmosis: *Toxoplasma gondii* is a ubiquitous intracellular protozoan. Although *T. gondii* can infect a wide range of vertebrates, feral and domestic cats are the definitive hosts. The organism undergoes its complete life cycle in cat, resulting in the production of oocytes, which are passed with the feces into soil (Sabry *et al,* 2013). *T. gondii* prevalence in Egyptian feral cats denoted a high oocysts environmental contamination (Al-Kappany *et al,* 2010). Oocytes may remain infective for over one year and infection is mainly acquired by ingestion of food or water contaminated with oocysts shed by cats or by eating undercooked or raw meat containing tissue cysts or use contaminated knives, cutting boards or other utensils (Montoya and Liesenfeld, 2004), or congenitally mother to fetus (Saleh *et al*, 2014). Transmission also, occurred by blood transfusion from infected donor (Sarwat *et al,* 1993), occupational (Saleh *et al,* 2016) and needle-injury (Abdel-Motagaly *et al,* 2017). There is no evidence of any other type of man-to-man transmission. Barsoum (2004) reported that toxoplasmosis, malaria, trypanosomia-

sis, and leishmaniasis are the main parasites transmitted with bone marrow, kidney, or liver homograft. Miró *et al.* (20120 in Spain declared that parasites must be considered in the differential diagnosis of post-transplant infections in foreign-born recipients. Kim *et al.* (2020) in Korea by DNA analyzed detected *T. gondii* in ticks, but neither associated with tick species nor development stage. Percipalle *et al.* (2021) in Italy detected traces of *T. gondii* DNA in samples of dehydrated mealworm whose life cycles make them candidates for potential insect breeding substrate contamination. They concluded the need for implementing good farming and processing practices with particular care to safe storage and handling of feed and substrates used for edible insects to reduce *T. gondii* entering the human food chain

Clinical epidemiology: *T. gondii* is generally believed to cause subclinical infection in most immunocompetent hosts, but one review found that one-third of reported cases of active pneumonia were in patients with no underlying immunosuppressive illness (Pomeroy and Filice, 1992). Of the remaining two-thirds, 61% had AIDS & 39% had other forms of immunosuppression (Mariuz *et al,* 1994). Most active cases of toxoplasmosis are due to reactivation of latent infection. About one-third of adults in the United States were IgG seropositive for *T. gondii* (Evans and Schwartzman, 1991). In HIV-patients who were seropositive for *T. gondii* was estimated about 30% developed *Toxoplasma* encephalitis within 2 years of initial AIDS diagnosis; another 1% who was seronegative, but developed primary toxoplasmosis (Holliman, 1990).

Generally, toxoplasmosis is a serious and often life-threatening disease in immunodeficient patients. Although encephalitis is overwhelmingly the commonest manifestation of *T. gondii* infection in AIDS patients, pneumonitis has become its second most common presentation. Incidence of pneumonitis is unknown, but number of cases is increasing. The estimated prevalence of *T. gondii* pneumonia in France was 5%, based upon a prospective study of bronchoalveolar
on a prospective study of bronchoalveolar lavage (BAL) specimens in 169 AIDS patients. Rates in the United States were much lower, which may be due to lower rates of dormant infection or to under-diagnosis. Active pulmonary toxoplasmosis did not occur in HIV-infected patients until CD4+ count falls <100cells/mm3 (Derouin *et al,* 1990).

Clinical presentation: *Toxoplasma* pneumonitis generally presents with fever, nonproductive cough, and dyspnea. The chest radiographs generally show diffuse bilateral interstitial and alveolar infiltrates. Other abnormalities include single or bilateral pulmonary nodules, cavitary infiltrates, lobar pneumonia, and pleural effusions (Bonilla and Rosa, 1994). Pulmonary toxoplasmosis may be clinically indistinguishable from PCP, tuberculosis, cryptococcosis, or histoplasmosis.

Serologic tests for IgG, IgM, IgA, & IgE to *T. gondii* are available, but results are not always helpful in profoundly immunosuppressed patient. However, the absence of IgG antibody level to *T. gondii* did make the diagnosis much less likely, since most active disease is due to reactivation of latent infection. Gallium scans in patients with *T. gondii* pneumonitis were rare, but diffuse intense uptake was reported, and serum LDH levels markedly elevated (Pugin *et al,* 1992).

Diagnosis: Bronchoscopy with bronchialveolar lavage (with or without transbronchial biopsy) was the preferred diagnostic method, but its sensitivity and specificity were unknown (Oksenhendler *et al,* 1990). Diagnosis was confirmed by observing the tachyzoite form of the organism in the BAL fluid or transbronchial biopsy. The reliable diagnostic methods are Giemsa stained or eosin/ methylene blue stains. Tachyzoite is crescent-shaped 5 to 7 microns in length. Immunofluorescence staining with a monoclonal antibody, inoculation of mice followed by traditional culture, or PCR increased the yie-

ld of BAL, but most of these methods were not available (Derouin *et al,* 1989).

If bronchoscopy is not diagnostic, then an open lung biopsy can be performed, either by video-assisted thoracoscopic surgery or traditional thoracotomy. Pathologically, a fibrinous exudate can be seen in the bronchi and alveoli, with an inflammatory cell interstitial infiltrate and areas of parenchymal necrosis. Organism may be seen within alveolar macrophages or freely floating within the alveoli. But, diagnosis of *Toxoplasma* pneumonitis was usually made by postmortem examination of the lungs, because it is often not considered premortem and with the lack of special stains for diagnosis, a review estimated mortality to be 40% in immunosuppressed hosts (Burg *et al,* 1989).

Treatment: Most healthy people don't require treatment. But, if health one otherwise with signs and symptoms of acute toxoplasmosis, his doctor may prescribe the following drugs: Pyrimethamine (Daraprim®) typically used for malaria is a folic acid antagonist. Other potential side effects of pyrimethamine include bone marrow suppression and liver toxicity. Also, Sulfadiazine® was used with pyrimethamine to treat children toxoplasmosis (Wishahy *et al*, 1972). The combination of Pyrimethamine and Sulfadiazine is the regimen of choice for treatment of extrapulmonary toxoplasmosis. This regimen was used for pulmonary toxoplasmosis as there were no controlled studies specifically designed for lung infection. A 200mg loading dose of pyrimethamine was given initially and followed by 50 to 75mg/day, but sulfadiazine was given at 4 to 6gm/day (McCabe and Oster, 1989). Leucovorin calcium (10 to 20mg/day orally) usually given to reduce hematologic toxicity of these drugs. Clindamycin (in dose of 600mg every 6hrs.) was used combined with pyrimethamine in patients with sulfa intolerance. Treatment time of toxoplasmosis pneumonitis was unknown, however, at least three to six weeks was given, depending upon the disease severity and the response rate (Dannemann *et al,* 1992).

The acute toxoplasmosis during pregnancy is detrimental to the developing fetus. Thus, treating pregnant women and babies varied. If infection occurred before the 16th week of pregnancy, spiramycin® was recommended, which may reduce fetus's risk of neurological problems, but if occurred after the 16th week of pregnancy, or fetus had toxoplasmosis, pyrimethamine and sulfadiazine and folinic acid (leucovorin) was recommended. Besides, prenatal care must include health education about prevention of toxoplasmosis (Paquet and Yudin, 20180). Other drugs included atovaquone, azithromycin, clarithromycin, dapsone, pyrimethamine or trimethoprim-sulfamethoxazole alone or in combination with others (Dunay *et al*, 2018).

Some studies suggest successful treatment outcomes in 50 to 77% of patients, although the number of cases of pulmonary toxoplasmosis is small (Maguire *et al,* 1986).

Secondary prophylaxis: Secondary prophylaxis or maintenance therapy is prudent because relapses of toxoplasmosis are reported in up to 80% of patients after successful treatment (Lane *et al,* 1994). The lowest *Toxoplasma* encephalitis relapse rate was reported with Pyrimethamine (25 to 75mg/day) & sulfadiazine (1.0 to 1.5gm four times daily) with Leucovorin (15mg/day). Clindamycin with Pyrimethamine can be used in sulfa intolerant patients. There was, however, a high relapse rate with low doses of Clindamycin; as a result, 1200mg/day in divided doses is suggested if tolerated by the patient (Luft and Remington, 1992).

According to guidelines issued by CDC, and the Infectious Diseases Society of America, discontinuation of secondary prophylaxis was considered if the patient successfully completed treatment, asymptomatic and CD4 count was maintained above 200cells/ microL for six months (Benson *et al,* 2005).

Primary prophylaxis: Primary prophylaxis against *Toxoplasma* encephalitis should be considered in patients with CD4+ counts below 100cell/mm3 and positive *T. gondii* serology. TMP/SMX is the recommended first-

line prophylactic agent, as it also gave prophylaxis against PCP. Other effective prophylactic drugs include pyrimethamine as a single agent, pyrimethamine-dapsone, fansidar, as well as clarithromycin, azithromycin, and atovaquone (Furrer *et al,* 2000).

Seronegative persons who are not taking a PCP prophylactic agent with known activity against toxoplasmosis should be retested for IgG antibody when their CD4+ count drops below 100cells/mm3. They should receive appropriate prophylaxis if they have seroconverted (CDC, 1995). If the CD4 count rises above 200cells/microL for three months, primary prophylaxis for toxoplasmosis may be discontinued (Mussini *et al,* 2000).

Strongyloidiasis: *Strongyloides stercoralis* is an intestinal parasite that has a worldwide distribution. Low estimates postulate it to affect 30-100 million people worldwide mainly in the tropical and subtropical countries (Buonfrate *et al,* 2015), while higher estimated conservatively extrapolate that infection is upwards to or above 370 million people (Varatharajalu and Kakuturu, 2016), but was predominantly found in the tropical and subtropical areas as well as the southeastern United States (Grove, 1989). The primary mode of transmission occurred when larvae from contaminated feces penetrate the skin, although infection can also occur via the fecal-oral route and from sexual transmission (Jaleta *et al,* 2017). Most infected persons remain either asymptomatic or have low grade abdominal symptoms. Some patients, particularly those who were immunosuppressed, could develop disseminated strongyloidiasis or hyperinfection syndrome, both of which were considered as systemic strongyloidiasis (Satoh *et al,* 2003). The free cycle of *Strongyloides* allowed them for residency in the lungs (Viney, 2006). Strongyloidiasis dissemination occurred when chronic strongyloidiasis patients became immunosuppressed or when the larval form was found outside the usual migration pattern. Hyper-infection is an augmentation of life cycle, resulted in heavy worms' lung infections (Lessnau *et al,* 1993). The hyper-infection of various levels of severe dissemination may present with abdominal pain, distensions, shock, pulmonary and neurologic complications, sepsis, hemorrhage, malabsorption, and depending on the combination, degree, number, and severity of symptoms, was fatal (Arthur and Shelley, 1958). Worms enter bloodstream via bowel wall, simultaneously allowed entry of bowel bacteria such as *Escherichia coli* (Ghoshal *et al,* 2002), which caused symptoms such as sepsis to bloodstream infection (Graeff-Teixeira *et al,* 2002), or bacteria spread to other organs where they caused localized infection as meningitis (El-Bahnasawy *et al,* 2016). Wołyniec *et al.* (2018) found that solid organ and bone marrow (toxoplasmosis, malaria, & leishmaniasis) transplantations, blood transfusions and immunosuppressive treatment were associated with a small, but real risk of giardiasis and cryptosporidiosis causing diarrhea in the European Citizens. Fürnkranz and Walochnik (2021) in Austria reported nosocomial *S. stercoralis* transmission by organ transplantation

Clinical manifestations: Majority of strongyloidiasis infected people do not have symptoms. Those who developed symptoms often have non-specific, or generalized complaints, as abdominal pain, bloating, heartburn, intermittent episodes of diarrhea and constipation, a dry cough, vomiting, and weight loss, red hives near the anus, and skin rashes. The acute manifestations are associated with the pre-patent period, defined as the time from penetration of infective larvae to production of new larvae by a mature female adult (Freedman, 1991). Infected individuals frequently experience irritation at the site of skin penetration that appears immediately followed occasionally by localized edema or urticaria that could last up to 3 weeks. Urticaria particularly involving the perianal skin and buttocks were the commonest chronic strongyloidiasis symptom (O'Brien, 1975). Within a week following infection, a dry cough may occur. Gastrointestinal symptoms such as diarrhea, constipation, abdominal

pain or anorexia can occur following the infection establishment in the small intestine as early as the 3rd week of infection (Keiser and Nutman, 2004). Once larval production by adults starts (~1 month following initiation of infection) new cycles of infection can be initiated through autoinfection (whether within the intestinal mucosa or in perianal skin) that often presents as a non-specific urticarial rash or pathognomonic *larva currens* (Gaus *et al,* 2011). Barsoum (2004) in Egypt reported that strongyloidiasis was among the parasites reactivated in the immunocompromised host (organ transplantation).

Clinical epidemiology: Surprisingly, there have been scattered cases of systemic strongyloidiasis in HIV-infected patients (Maayan *et al,* 1987). The only associated risk factor in the HIV-infected population is previous or current residence in an endemic area. Risk factors for the strongyloidiasis development in the general population include: Race (white) Gender (male) Use of steroids Hematologic malignancy Prior gastric surgery (Davidson *et al,* 1984). Moreover, the presence of co-infection with schistosomiasis or ascariasis may be an additional risk factor that was relevant in rural, underdeveloped countries where these infections were common (Nucci *et al,* 1995).

Clinical presentation: Systemic strongyloidiasis is manifested by fever, malaise, and gastro-intestinal &/or respiratory symptoms. Eosinophilia is common in immunocompetent patients with chronic strongyloidiasis, but it may be absent in immunocompromised patients who develop systemic strongyloidiasis. Eosinopenia was a poor prognostic indicator as Gram negative bacteremia and meningitis may complicate these infections (Igra-Siegman *et al,* 1981).

Diagnosis: The organisms may be identified through examination of wet preparations of stool, sputum, or bronchoalveolar lavage fluid. Diagnosis may also be made serologically, but the tests were not widely available: ELISA has a sensitivity of 85 to 90% and a specificity of 90%. However, there were false positive tests in patients with other parasitic infections **(**Celedon *et al,* 1994). Serial antibody titers may be followed in patients who have been treated, but with some difficulties in distinguishing acute from previous infection.

Treatment: Ivermectin (200mcg/kg/day) is the first-line agent for treatment, and a course of 5 to 7 days was suggested in immunosuppressed patients with systemic disease. But, ivermectin did not kill the larvae, only adults; therefore repeat dosing was necessary to properly eradicate the infection and auto-infective cycle of roughly two weeks (Repetto *et al,* 2018). Lifelong suppressive therapy may be indicated for both gastrointestinal and pulmonary infections in patients with relapses (Igual-Adell *et al,* 2004). Other effective strongyloidiasis drugs are Albendazole &Thiabendazole (25mg/kg twice daily for 5 days to 400mg maximum (Gompels *et al,* 1991). Massoud *et al.* (2006) in Egypt treated 28 parasitological proved strongyloidiasis male patients (18-65 years old) with Mirazid® given for one month except three resistant cases. One of them responded to repeated course of Mirazid, while the other two cases still had larvae in their stool by agar culture plate. On combined therapy of both Mirazid & Mebendazole®, larvae were eliminated from their stool as approved by agar plate culture.

Empiric therapy: Prophylaxis should be considered in patients from endemic areas, especially those with unexplained eosinophilia. Ivermectin was given as a single dose (200mcg/kg/day) or a multi-dose schedule (200mcg/kg/day for four days). Remissions induced by the multi-dose regimen of ivermectin were maintained for up to 3 years (Torres *et al,* 1993).

Entamoeba histolytica: Amebic liver abscess always preceded lung infections; early diagnosis and treatment prevent complications (Del Campo and Del Campo, 1982). Invasive amebiasis was more common in immunosuppressed, pregnant women, children, and alcoholics. Pleuropulmonary form occ-

urred exclusively with complications included right-sided sympathetic effusions, empyema, basilar atelectasis, lung infiltration, & abscess (Lyche and Jensen, 1977). Shamsuzzaman and Hashiguchi (2002) in Japan detected *Acanthamoeba*, a free-living ameba, infected the lungs forming pulmonary nodular infiltration and edema in association with amebic meningoencephalitis in immunocompromised patients. They concluded that HIV/AIDS patients were not prone to *E. histolytica* infection and that minimal incidence of intestinal infection among HIV-seropositive or AIDS patients did not mean any more amebiasis invasive.

Echinococcosis/hydatidosis: *Echinococcus granulosus* and *E. multilocularis* are the commonest organisms responsible for pulmonary hydatid cysts (Polat *et al,* 2003) of worldwide distribution (Schwartz, 1994). Dogs stray (Sarkari and Rezaei, 2015) or pet ones (Sabry *et al,* 2012) as well as other carnivores are the definitive hosts. All vertebrate mammals including man are the intermediate hosts (Eckert and Deplazes, 2004). Iyigun *et al.* (2004) considered hydatidosis an occupational infectious disease. Hydatidosis is acquired by ingesting echinococcosis eggs in food or water or by close contact with an infected dogs, foxes and wolves (Turkyilmaz *et al,* 2004). Hydatid cysts consist of three layers, pericyst, ectocyst, and endocyst, which can be ruptured or un-ruptured (Haridy *et al,* 2008). Human lungs were the second most common organ of involvement in adults, after the liver (Wilson, 1991). Hydatidosis of organs outside of liver or lung were unusual as CNS (Mazyad *et al*, 1999), heart, kidney, bone and ocular (Gogus *et al,* 2003), brain (Farahmand *et al,* 2010), ovary causing ovarian neoplasm (Sharma *et al,* 2012), pancreas (Akbulut *et al,* 2014), pelvic (Bhatnagar *et al,* 2017), breast (Temiz *et al,* 2017), uterus (Kakaei *et al,* 2017), foot (Ewnte, 2020), or even anal fossa (Abdalla *et al,* 2020). However, lungs were the most common site of involvement in children (Polat *et al,* 2003). Un-ruptured hydatids on CT appeared as well demarcated spherical or oval homogeneous cystic lesions with enhancing walls in the lung, more commonly in mid and lower zones (Pedrosa *et al,* 2017). Daughter cysts may be seen as curved septations within it, but calcification was rare in pulmonary hydatid cysts (El-Sayed *et al,* 2020). Cyst rupture can also lead to secondary infection or lung abscess formation with a type of liquefactive necrosis of lung tissue and formation of cavities (more than 2 cm) containing necrotic debris or fluid caused by microbial infection (Kuhajda *et al,* 2015).

Alveolar echinococcosis (AE) was a rare but potentially life-threatening infection due to the accidental ingestion of the egg of the parasite *Echinococcus multilocularis*. It is encountered only in the northern hemisphere (Wen *et al,* 2019). Its natural history is characterized by a slow parasitic growth over several years, as Immunomodulation of host immunity toward energy was triggered by parasite metabolites (Gottstein *et al,* 2015). Dupont *et al.* (2020) reported an exceptionally fast growing and aggressive *E. multilocularis* in a 41-year old Caucasian female veterinarian who underwent a right lung transplantation (LT) for pulmonary fibrosis, and Ten months after transplantation, irregular hepatomegaly was detected associated with persisting pain in the right hypochondrium. They concluded that clinicians dealing with immunosuppressed patients living in endemic areas should not only be aware of the need to search for preexisting lesions, but also help prevent new cases of infections.

Treatment: Sayek *et al.* (2004) in Turkey mentioned that although certain types of hydatid cysts were successfully treated by percutaneous aspiration, injection, and reaspiration, treatment of choice is surgery. Thapa *et al.* (2018) in United Kingdom reported that controversies about the best surgical technique, complications brought on by cyst rupture, multiplicity and multi-organ involvement add complexity to treatment decisions. El-Sayed *et al.* (2020) in Egypt successfully used staging surgery for patient with bilateral

pulmonary cyst or pulmonary and liver cysts. Albendazole in dose10mg/ kg/day was given pre- and post-operative. Also, Ibrahim and Morsy (2020) reported that both PAIR and laparoscopic procedure were recommended as staging treatment surgery of liver hydatidosis, with albendazole 10mg/kg/d was given as prophylaxis during PAIR technique**.**

Paragonimiasis is a foodborne parasite caused by the lung fluke, most commonly *Paragonimus westermani* infecting about 22 million people yearly worldwide (Lane *et al,* 2009). Acute phase (invasion and migration) caused diarrhea, abdominal pain, fever, cough, urticaria, hepatosplenomegaly, pulmonary abnormalities, and eosinophilia. In chronic phase, pulmonary manifestations included cough, expectoration of discolored sputum containing clumps of eggs (CDC, 2013). Pozio (1991) in Western Africa reported human paragonimiasis (2 to 31%).Diaz (2011) in USA reported paragonimiasis after eating raw crayfish. Song *et al.* (2011) in Korea reported that surgeons must include paragonimiasis in differential diagnosis of asymptomatic nodular lesions in patients' lungs. Field *et al.* (2018) in Canada reported that cough is common in pulmonary TB and other chronic respiratory infections due to mycobacteria, fungus, and paragonimiasis.

Cryptosporidium is causative agents of gastrointestinal disease in HIV-infected patients (Mannheimer and Soave, 1994). Lung disease due to *Cryptosporidium* was reported in the early AIDS era, before anti-riretroviral therapy, and fever and cough were the dominant symptoms (Forgacs *et al,* 1983).

Cryptosporidium is a microscopic parasite that causes the diarrheal disease cryptosporidiosis. Both the parasite and the disease are commonly known as "Crypto." By genotypes within *Cryptosporidium* isolates showed about 20 species (Robinson *et al*, 2008). *Cryptosporidium* species infect all the vertebrates including mammals, reptiles, birds, and fish, some of which also infect humans (Sponseller *et al,* 2014). *C. parvum* was divided into two separate species: *C. hominis* (previously *C. parvum* genotype 1) and *C. parvum* (formerly *C. parvum* genotype 2). *C. hominis* apparently infects only man, and *C. parvum* was found in many animals and in man (Morgan-Ryan *et al*, 2002). Animal cryptosporidiosis as *C. canis, C. felis, C. meleagridis, C. muris,* and *C. suis* were reported in some patients (Cacciò, 2005). Pathogenic agents was associated with *C. parvum* were *Cyclospora cayetanensis Microsporidium* and cytomegalovirus (Teixidor *et al*, 1991).

People at risk: many people are at exposure risks: 1- People who swim regularly in pools with insufficient sanitation, as certain *Cryptosporidium* strains are chlorine-resistant (CDC, 2009). 2- The person-to-person transmission is common particularly among family members, sexual partners, children in daycare centers (Musher and Musher, 2004). 3- Nursing of cryptosporidiosis pati-ents. 4- People consuming contaminated water from unfiltered or untreated sources as well as contaminated raw vegetables or fruits (Ethelberg *et al*, 2009). 5- Farmers and visitors visiting petting farms and open farms with public access (Walker, 2018), an outbreak of cryptosporidiosis was reported among firefighters responded to a fire in the barn housing calves, where many waterbor-ne outbreaks were reported (CDC, 2012). 6- People exposed to human feces (fecal-oral), as well as insertive anal sex (Black *et* al, 2003) and oral-genital sex (Borchardt and Noble, 1997). 7- Respiratory transmission might arise if oocysts were aerosolized during coughing (Højlyng *et al,* 1987), and 8- People exposed to human feces.

Prevention: *Cryptosporidium* is resistant to chlorine disinfection, so it is tougher to be killed than most disease-causing germs. The usual disinfectants, including most commonly used bleach solutions gave little effect on the parasite. An application of hydrogen peroxide was the best (Chalmers *et al,* 2019).

Blood parasites: babesiosis and malaria are parasites of worldwide distribution. Babesiosis is transmitted by hard-ticks, and malaria by female *Anopheles*. Also, both are transm-

itted by blood transfusion, needle-stick injury and congenitally. Some *Babesia* species cause potentially fatal disease in many mammalian species including man. it causes hemolysis accounting for clinical complications as hemolytic anemia, cardiorespiratory /renal failure, disseminated intravascular coagulation and even death (Vannier and Krause, 2012). Malaria (*Plasmodium falciparum, P. vivax, P. malariae, P. ovale*, and *P. knowlesi*), is a risky human disease. The major clinical complications are severe anemia, cerebral malaria, placental malaria, acute renal failure and acute lung injury/acute respiratory distress (Taylor *et al,* 2012). Pulmonary edema due to increased pulmonary capillary permeability is the risky pulmonary manifestation (Adam and Elbashir, 2004).

Also, visceral leishmaniasis (VL) is a severe, systemic and potentially lethal parasites. Lung, like any other organ, can be affected in VL, and interstitial pneumonitis was described in past decades (Morsy, 1997). Typical manifestations include fever, weight loss, hepatosplenomegaly, and pancytopenia resulting from *Leishmania* amastigotes replication in macrophages mainly in liver, spleen, and bone marrow, but as splenomegaly may be absent in VL/HIV-co-infected patients, but atypical organ involvement, such as of lungs or gastrointestinal system (Bispo *et al,* 2020). Amyloid A (AA) amyloidosis leading to renal failure was associated with chronic VL in HIV patients

Prevention of HIV is spread by having sex or sharing syringes and other injection equipment with HIV infected someone. Substance use can contribute to these risks indirectly because alcohol and other drugs can lower people's inhibitions and make them less likely to use condoms (CDC, 2020).

Conclusion

Generally speaking, human immunodeficiency viruses are two species of *Lentivirus* (a subgroup of retrovirus) that infect human. Over time, they cause acquired immunodeficiency syndrome (AIDS), a condition in which progressive failure of immune system allows life threatening opportunistic infections and cancers to thrive. Without treatment, average survival time after HIV infection was about 9 to 11 years, depended on the HIV subtype. Lungs provide a protected environment for the survival of vulnerable stages of some parasites development, which were omnipresent and exerted persistent selective pressure on the human immune response. Invasive parasites including lung ones are increasing in HIV patients. Patients with immunodeficiency syndromes (HIV infection, organ transplantation and immunosuppressive drugs; as corticosteroids) must be evaluated for early diagnosis of lung parasites and proper treatment. No doubt, respiratory diseases are a risky cause of morbidity and mortality.

References

Abdalla, KM, Al Sharie, AH, ALzu'bi, YO, Daoud, MN, Aleshawi, AJ, *et al,* 2020: Ischioanal fossa hydatid cyst. Int. J. Infect. Dis. 92: 181-3.

Abdel-Motagaly, AME, Ibrahim, AMA, Morsy, TA, 2017: An intervention program on blood protozoa acquired by needle stick injury and infection control. J. Egypt. Soc. Parasitol. 47, 2: 309-22

Adam, I, Elbashir, MI, 2004: Maternal death due to severe pulmonary oedema caused by falciparum malaria: a case report. East. Mediterr. Hlth. J. 10, 4/5:685-7.

Akbulut, S, Yavuz, R, Sogutcu, N, Kaya, B, Hatipoglu, S, *et al,* 2014: Hydatid cyst of the pancreas: Report of an undiagnosed case of pancreatic hydatid cyst and brief literature review. World J. Gastrointest. Surg. 6, 10:190-200

Al-Kappany, YM, Rajendran, C, Ferreira, L R, Kwok, OC, Abu-Elwafa, SA, *et al,* 2010: High prevalence of toxoplasmosis in cats from Egypt: Isolation of viable *Toxoplasma gondii,* tissue distribution, and isolate designation. J. Parasitol. 96, 6:1115-8

Arthur, RP, Shelley, WB, 1958: Larva currens: A distinctive variant of cutaneous larva migrans due to *Strongyloides stercoralis*. AMA Arch. Dermatol. 78, 2:18-21

Barsoum, RS, 2004: Parasitic infections in organ transplantation. Exp. Clin. Transplant. 2, 2: 258-67.

Benson, CA, Kaplan, JE, Masur, HC, *et al,*

2005: Treating opportunistic infections among HIV-infected adults and adolescents: Recommendations from CDC, the NIH, & HIV Medicine Association/Infectious Diseases Society of America. Clin. Infect. Dis. 40:S131.

Bhatnagar, N, Kishan, H, Sura, S, Lingaiah, P, Jaikumar, K, 2017: Pelvic hydatid disease: A case report and review of literature. J. Orthop. Case Rep. 7:25-8

Bispo, AJB, Almeida, MLD, de Almeida, RP, Bispo, NJ, de Oliveira Brito, AV, *et al,* 2020: Pulmonary involvement in human visceral leishmaniasis: Clinical and tomographic evaluation. PLoS One Jan 30;15(1):e0228176.

Black, ER, Mushlin, AI, Griner, PF, Suchman, AL, James, RL, *et al,* 2003: Predicting the need for hospitalization of ambulatory patients with pneumonia. J. General Inter. Med. **6**, 5:394-400.

Bonilla, CA, Rosa, UW, 1994: *Toxoplasma gondii* pneumonia in patients with the acquired immuno-deficiency syndrome: Diagnosis by bronchoalveolar lavage. South Med. J. 87:659-63.

Borchardt, KA, Noble, MA, 1997: Sexually Transmitted Diseases: Epidemiology, Pathology, Diagnosis, and Treatment. CRC Press.

Buonfrate, D, Formenti, F, Perandin, F, Bisoffi, Z, 2015: Novel approaches to the diagnosis of *Strongyloides stercoralis* infection. Clin. Microbiol. Infect. 21, 6:543-52

Burg, JL, Grover, CM, Pouletty, P, Boothroyd, JC, 1989: Direct and sensitive detection of a pathogenic protozoan, *Toxoplasma gondii*, by polymerase chain reaction. J. Clin. Microbiol. 27:1787-92.

Cacciò, SM, 2005: Molecular epidemiology of human cryptosporidiosis. Parassitologia 47:185-92.

CDC, 1995: USPHS/IDSA guidelines for the prevention of opportunistic infections in persons' infected with human immunodeficiency virus: A summary. MMWR Morb. Mortal. Wkly. Rep. 44:1.

CDC, 2009: Outbreak of cryptosporidiosis associated with a splash park-Idaho, 2007. MMWR Morb. Mortal. Wkly. Rep. 58:615.

CDC, 2012: Outbreak of cryptosporidiosis associated with a firefighting response: Indiana and Michigan, June 2011. MMWR Morb. Mortal. Wkly. Rep. 61:153.

CDC, 2013: Paragonimiasis, clinical features. Archived from retrieved 2012-09-06.

CDC, 2020: HIV Risk and Prevention. Advanced Research.

Celedon, JC, Mathur-Wagh, U, Fox, J, *et al,* 1994: Systemic *Strongyloides* in patients infected with the human immunodeficiency virus. Medicine 73:256-60.

Chalmers, RM, Davies, AP, Tyler, K, 2019: *Cryptosporidium*. Microbiology (Reading) 165, 5:500-2

Dannemann, B, McCutchan, JA, Israelski, D, *et al,* 1992: Treatment of toxoplasmic encephalitis in patients with AIDS, a randomized trial comparing pyrimethamine plus clindamycin to pyrimethamine plus sulfadiazine. Ann. Intern. Med. 116:3-8.

Davidson, RA, Fletcher, RH, Chapman, LS, 1984: Risk factors for *Strongyloides*: A case-control study. Arch. Intern. Med. 144:321-6.

Del Campo, C, Del Campo, M, 1982: Thoracic complications of amebiasis. Can. J. Surg. 25, 2: 119-21.

Derouin, F, Sarfati, C, Beauvais, B, *et al,* 1989: Laboratory diagnosis of pulmonary toxoplasmosis in patients with acquired immunodeficiency syndrome. J. Clin. Microbiol. 27:1661-8.

Derouin, F, Sarfati, C, Beauvais, B, *et al,* 1990: Prevalence of pulmonary toxoplasmosis in HIV-infected patients. AIDS 4:1036-42.

Diaz, JH, 2011: Boil before eating: Paragonimiasis after eating raw crayfish in the Mississippi River Basin. ***J. La State Med Soc.*** 163, 5:261-6.

Dunay, IR, Gajurel, K, Dhakal, R, Liesenfeld, O, Montoya, J, 2018: Treatment of toxoplasmosis: Historical perspective, animal models, and current clinical practice. Clin. Microbiol. Rev. 31, 4:e00057-17.

Dupont, C, Grenouillet, F, Mabrut, JY, Gay, F, Persat, F, *et al,* 2020: Fast-growing alveolar echinococcosis following lung transplantation, Pathogens. 9(9):756.https://doi.org/10.3390/pathogens9090756.

Eckert, J, Deplazes, P, 2004: Biological, Epidemiological, and Clinical Aspects of Echinococcosis, a Zoonosis of Increasing Concern. Clin. Microbiol. Rev. 17, 1:107-35.

El-Bahnasawy, MMN, El Feky, MR, Morsy, ATA, Ismail, MAM, Morsy, TA, 2016: Egyptian eosinophilic and infectious meningoencephalitis &their impact on psychological aspects. J. Egypt. Soc. Parasitol. 46, 1:67-80.

El-Sayed, YA, Ibrahim, EA, Morsy, TA, 2020: Peri-cystectomy versus cystotomy and enucleation of germinal layer by tissue spearing technique for lung hydatid in Egypt. J. Egypt. Soc.

Parasitol. 50, 3:494-503.
Ethelberg, S, Lisby, M, Vestergaard, LS, *et al*, 2009: A foodborne outbreak of *Cryptosporidium hominis* infection. Epidemiol. Infect. 137:348-52
Evans, GT, Schwartzman, JD, 1991: Pulmonary toxoplasmosis. Semin. Respir. Infect. 6:51-8.
Ewnte, B, 2020: Hydatid cyst of the foot: A case report. J. Med. Case Rep. 14, 1:6. doi:10.1186/
Farahmand, M, Yadollahi, M, 2010: Echinococcosis: An occupational disease. Int. J. Occup. Environ. Med. 1, 2:88-91.
Field, SK, Escalante, P, Fisher, DA, Ireland, B, Irwin, RS, 2018: Cough due to tb and other chronic infections: Chest guideline and expert panel report. Chest 153, 2:467-97.
Forgacs, P, Tarshis, A, Ma, P, *et al*, 1983: Intestinal and bronchial cryptosporidiosis in an immuno-deficient homosexual man. Ann. Intern. Med. 99:793-5.
Freedberg, KA, Scharfstein, JA, Seage, GA, *et al*, 1998: The cost effectiveness of preventing AIDS-related opportunistic infections. JAMA 279:130-6.
Freedman, DO, 1991*:* Experimental infection of human subject with *Strongyloides* species, a review. Rev. Infect. Dis. 13, 6:1221-6.
Fürnkranz, U, Walochnik, J, 2021: Nosocomial infections: Do not forget the parasites! Pathogens 10, 2:238. doi:10.3390/10020238.
Furrer, H, Opravil, M, Bernasconi, E, *et al,* 2000: Stopping primary prophylaxis in HIV-1 infected patients at high risk of toxoplasma encephalitis: Swiss HIV Cohort Study. Lancet 355: 2217-21.
Gaus, B, Toberer, F, Kapaun A, Hartmann, M, 2011: Chronic *Strongyloides stercoralis* infection: Larva *currens* as skin manifestation. Hautarzt 62, 5:380-3.
Ghoshal, UC, Ghoshal, U, Jain, M, Kumar, A, Aggarwal, R, *et al,* 2002: *Strongyloides stercoralis* infestation associated with septicemia due to intestinal transmural migration of bacteria. J. Gastroenterol. Hepatol. 17, 12:1331-3.
Gogus, C, Safak, M, Baltaci, S, Turkolmez, K, 2003: Isolated renal hydatidosis: Experience with 20 cases. J. Urol. 169:186.
Gompels, MM, Todd, J, Peters, BS, Main, J, Pinching, AJ, 1991: Disseminated strongyloidiasis in AIDS: Uncommon but important. AIDS 5, 3:329-32.
Gottstein, B, Wang, J, Boubaker, G, Marinova, I, Spiliotis, M, *et al*, 2015: Susceptibility versus resistance in alveolar echinococcosis (larval infection with *Echinococcus multilocularis).* Vet. Parasitol. 213:103-9
Graeff-Teixeira, C, da Silva, AC, Yoshimura, K, 2009: Update on eosinophilic meningoencephalitis and its clinical relevance. Clin. Microbiol. Rev. **22**, 2: 322-48.
Grove, DI, 1989: Strongyloidiasis: A Major Roundworm Infection of Man. Philadelphia, PA: Taylor & Francis.
Haridy, FM, Abou Holw, SA, Hassan, AA, Morsy, TA, 2008: Cystic hydatidosis: A zoonotic silent health problem. J. Egypt. Soc. Parasitol. 38, 2:635-44**.**
Højlyng, N, Anderson, W, Jepson, S, 1987: Cryptosporidiosis: A case of airborne transmission. Lancet 2:271-2.
Holliman, RE, 1990: Serological study of the prevalence of toxoplasmosis in asymptomatic patients infected with human immunodeficiency virus. Epidemiol. Infect.105:415-9.
Ibrahim, EA, Morsy, TA, 2020: Tissue sparing technique in liver hydatid diseases: Three-way technique versus hydatid cone technique. J. Egypt. Soc. Parasitol. 50, 2:378-89.
Igra-Siegman, Y, Kapila, R, Sen, P, *et al,* 1981: Syndrome of hyperinfection with *Strongyloides stercoralis*. Rev. Infect. Dis. 3:397-400.
Igual-Adell, R, Oltra-Alcaraz, C, Soler, E, *et al,* 2004: Efficacy and safety of ivermectin and thiabendazole in the treatment of *Strongyloides*. Expert. Opin. Pharmacother. 5: 2615-20.
Iyigun, O, Uysal, S, Sancak, R, Hokelek, M, Uyar, Y, *et al,* 2004: Multiple organ involvement hydatid cysts in a 2-year-old boy. J. Trop. Pediatr. 250, 6:374-6
Jaleta, TG, Zhou, S, Bemm, FM, Schär, F, Khieu, V, *et al,* 2017: Different but overlapping populations of *Strongyloides stercoralis* in dogs and humans-dogs as a possible source for zoonotic strongyloidiasis. PLoS Negl. Trop. Dis. 11, 8: e0005752.
Kakaei, F, Asvadi Kermani, T, Tarvirdizade, K, 2017: A case report: Primary hydatid cyst of uterus. Int. J. Surg. Case Rep. 42:67-9.
Keiser, PB, Nutman, TB, 2004: *Strongyloides stercoralis* in the immunocompromised population. Clin. Microbiol. Rev. 17, 1:208-17.
Kim, JY, Kwak, YS, Lee, IY, Yong, TS, 2020: Molecular detection of *Toxoplasma gondii* in *Haemaphysalis* ticks in Korea, Kor. J. Parasitol. 58, 3:327-31.
Kuhajda, I, Zarogoulidis, K, Tsirgogianni, K,

***et al,* 2015:** Lung abscess-etiology, diagnostic & treatment options. Ann. Trans. Med. 3, 13:183-9.

Lane, HC, Laughon, BE, Falloon, J, *et al,* 1994: NIH Conference. Recent advances in the management of AIDS-related opportunistic infections. Ann. Intern. Med. 120:945-52.

Lane, MA, Barsanti, MC, Santos, CA, Yeung, M, Lubner, SJ, *et al,* 2009: Human paragonimiasis *in* North America following ingestion of raw crayfish. Clin. Infecti. Dis. **49**, 6:e55-61

Lessnau, K, Can, S, Talavera, W, 1993: Disseminated *Strongyloides stercoralis* in human immunod-eficiency virus-infected patients. Chest 104:119-24.

Luft, BJ, Remington, JS, 1992: Toxoplasmic encephalitis in AIDS. Clin. Infect. Dis. 15:211-8.

Lyche, KD, Jensen, WA, 1997: Pleuropulmonary amebiasis. Semin. Respir. Infect. 12, 2:106-12.

Maayan, S, Wormser, G, Widerhorn, J, *et al,* 1987: *Strongyloides stercoralis* hyperinfection in a patient with the acquired immune deficiency syndrome. Am. J. Med. 83:945-50.

Maguire, GP, Tatz, J, Giosa, R, Ahmed, T, 1986: Diagnosis of pulmonary toxoplasmosis by bronchoalveolar lavage. N Y State J. Med. 86: 204-8.

Mannheimer, SB, Soave, R, 1994: Protozoal infections in patients with AIDS: Cryptosporidiosis, isosporiasis, cyclosporiasis, and microsporidiosis. Infect. Dis. Clin. North Am. 8:483-7.

Mariuz, P, Bosley, EM, Luft, BJ, 1994: Toxoplasmosis in individuals with AIDS. Infect. Dis. Clin. 8:365-0.

Massoud, AM, El-Shazly, AM, Awad, SE, Morsy, ATA, Sadek, GS, *et al,* 2006: New trends in diagnosis and treatment of chronic intestinal strongyloidiasis *stercoralis* in Egyptian patients. J. Egypt. Soc. Parasitol. 36, 3:827-44.

Mazyad, MAM, Morsy, TA, Habib, KSM, 1999: Vertebral unilocular hydatidosis in a shepherd and his wife. J. Egypt. Soc. Parasitol. 29, 2:547-50.

McCabe, RE, Oster, S, 1989: Current recommendation and future prospects in the treatment of toxoplasmosis. Drugs 38:973-6.

Miró, JM, Blanes, M, Norman, F, Martín-Dávila, P, 2012: Infections in solid organ transplantation in special situations: HIV-infection and immigration. Enferm. Infec. Microbiol. Clin. 30, 2:S76-85.

Montoya, JG, Liesenfeld, O, 2004: Toxoplasmosis. Lancet 363, 9425:1965-76.

Morgan-Ryan, UM, Fall, A, Ward, LA, *et al*, 2002: *Cryptosporidium hominis* n. sp. (Apicomplexa: Cryptosporidiidae) from Homo sapiens. J. Eukaryot. Microbiol. 49:433-9.

Morsy, TA, 1997: Visceral leishmaniasis with special reference to Egypt (Rev-iew and Comment). J. Egypt. Soc. Parasitol. 27, 2:373-96.

Musher, DM, Musher, BL, 2004: Contagious acute gastrointestinal infections. N. Engl. J. Med. 351: 2417-22.

Mussini, C, Pezzott, P, Govoni, A, *et al,* 2000: Discontinuation of primary prophylaxis for *Pneumocystis carinii* pneumonia and toxoplasmic encephalitis in human immunodeficiency virus type-1-infected patients: Changes in opportunisitic prophylaxis study. J. Infect. Dis. 181:1635-9.

Nucci, M, Portugal, R, Pulcheri, W, *et al,* 1995: *Strongyloides* in patients with hematological malignancies. Clin. Infect. Dis. 21:675-8.

O'Brien, W, 1975: Intestinal malabsorption in acute infection with *S. stercoralis*. Trans. R. Soc. Trop. Med. Hyg., 69: 69-77.

Oksenhendler, E, Cadranel, J, Sarfati, C, *et al,* 1990: *Toxoplasma gondii* pneumonia in patients with acquired immunodeficiency syndrome. Am. J. Med. 88:18-21.

Paquet, C, Yudin, MH, 2018: No. 285-toxoplasmosis in pregnancy: Prevention, screening, and treatment. J. Obstet. Gynaecol 40, 8:e687-93.

Pedrosa, I, Saíz, A, Arrazola, J, Ferreirós, J, Pedrosa, CS, 2000: Hydatid disease: Radiologic and pathologic features and complications. Radiographics 20, 3:795-817.

Percipalle, M, Salvaggio, A, Pitari, GM, Giunta, RP, Aparo, A, *et al,* 2021: Edible insects & *Toxoplasma gondii*: Is it something we need to be concerned about? J. Food Prot. 84, 3:437-41.

Polat, P, Kantarci, M, *et al,* 2003: Hydatid disease from head to toe. RadioGraphics 23:475-94.

Pomeroy, C, Filice, GA, 1992: Pulmonary toxoplasmosis, a review. Clin. Infect. Dis. 14:863-6.

Pozio, E, 1991: Current status of food-borne parasitic zoonoses in Mediterranean &African regions. Southeast Asian J. Trop. Med. Publ. Hlth. 22:S85-7.

Pugin, J, Vanhems, P, Hirschel, B, *et al,* 1992: Extreme elevations of serum lactic dehydrogenase differentiating pulmonary toxoplasmosis from *Pneumocystis pneumonia*. N. Engl. J. Med. 326:1226-9.

Repetto, SA, Ruybal, P, Batalla, E, López, C, Fridman, V, *et al,* 2018: Strongyloidiasis outsi-

de endemic areas: Long-term parasitological and clinical follow-up after ivermectin treatment. Clin. Infect. Dis. 66, 10:1558-65.

Robinson, G, Elwin, K, Chalmers, RM, 2008: Unusual *Cryptosporidium* genotypes in human cases of diarrhea. Emerg. Infect. Dis. 14:1800-4.

Sabry, AA, Morsy, ATA, Morsy, TA, 2012: Zoonosis from dogs with special reference to Egypt. J. Egypt. Soc. Parasitol. 42, 3:583-604.

Sabry, AA, Fouad, MAH, Morsy, ATA, 2013: Zoonosis from cats: With special reference to Egypt. J. Egypt. Soc. Parasitol. 43, 2:429-46.

Saleh, AMA, Al-Agroudi, MA, Morsy, TA, 2016: Occupational, nosocomial or hospital acquired toxoplasmosis. J. Egypt. Soc. Parasitol. 46, 2:407-18.

Saleh, AMA, Ali, HA, Ahmed, SAM, Hosny, S M, Morsy, TA, 2014: Screening of *Toxoplasma gondii* infection among childbearing age females and assessment of nurses' role in prevention and control of toxoplasmosis. J. Egypt. Soc. Parasitol. 44, 2:329-42.

Sarkari, B, Rezaei, Z, 2015: Immunodiagnosis of human hydatid disease: Where do we stand? Wld, J. Methodol. 5, 4:185-95.

Sarwat, MA, Ahmed, AB, Zamzami, OM, Fawzy, AF, Morsy, TA, 1993: *Toxoplasma gondii* in Saudi blood donors: A serological study by 3 tests. J. Egypt. Soc. Parasitol. 23, 3:751-7.

Satoh, M, Kiyuna, S, Shiroma, Y, *et al,* 2003: Predictive markers for development of strongyloidiasis in patients infected with both *Strongyloides stercoralis* and HTLV-1. Clin. Exp. Immunol. 133:391-6.

Sayek, I, Tirnaksiz, MB, Dogan, R, 2004: Cystic hydatid disease: current trends in diagnosis and management. Surg. Today 34, 12:987-96

Schwartz, SI, 1994: Liver. In: Principles of Surgery; Schwartz, SI, Shires, GT, *et al,* 6th ed.; New York, McGraw-Hill, Inc.

Shamsuzzaman, SM, Hashiguchi, Y, 2002: Thoracic amebiasis. Clin Chest Med. 23 2:479-92

Sharma, A, Sengupta, P, Mondal, S, Raychaudhuri, G, 2012: Hydatid cyst of ovary mimicking ovarian neoplasm with its imprint cytology. Am. J. Case Rep. 13:276-8.

Skalski, JH, Limper, AH, 2016: Fungal, viral, and parasitic pneumonias associated with human immunodeficiency virus. Semin. Respir. Crit. Care Med. 37, 2:257-66.

Song, JU, Um, SW, Koh, WJ, Suh, G, Chung, M, *et al,* 2011: Pulmonary paragonimiasis mimicking lung cancer in a tertiary referral centre in Korea. Int. J. Tuberc. Lung Dis. 15, 5:674-9.

Sponseller, J, Griffiths, J, Tripuri, S, 2014: The evolution of respiratory cryptosporidiosis: Evidence for transmission by inhalation. Clin. Microbiol. Rev. 27, 3:575-86

Taylor, WRJ, White, NJ, 2002: Malaria and the lung. Clin. Chest. Med. 23, 2:457-68.

Teixidor, HS, Godwin, TA, Ramirez, EA, 1991: Cryptosporidiosis of the biliary tract in AIDS. Radiology 180:51-8.

Thapa, B, Sapkota, R, Kim, M, Barnett, SA, Sayami, P, 2018: Surgery for parasitic lung inf-infestations: Roles in diagnosis and treatment. J. Thorac. Dis. 2018 10, 28:S3446-57

Temiz, A, Albayrak, Y, Akalp, SÖ, Yalçın, A, Albayrak, A, 2017: Breast recurrent hydatid cyst disease. Chirurgia (Bucur). 112, 4:482-5.

Torres, JR, Isturiz, R, Murillo, J, *et al,* 1993: Efficacy of ivermectin in the treatment of *Strongyloides* complicating AIDS. Clin. Infect. Dis. 17:900-6.

Turkyilmaz, Z, Sonmez, K, Karabult, R, Demiroglari, B, Gol, H, *et al,* 2004: Conservative surgery for treatment of hydatid cysts in children. Wld. J. Surg. 28:597-601.

UNAIDS, 2009: Annual Report towards Universal Access. http://data.unaids.org/pub/Report/jc

Varatharajalu, R, Kakuturu, R, 2016: *Strongyloides stercoralis*: Current perspectives. Reports in Parasitology: 23. doi:10.2147/RIP.S75839

Vannier, E, Krause, PJ, 2012: Human babesiosis. N. Engl. J/ Med. 366, 25:2397–407.

Viney, ME, 2006: The biology and genomics of *Strongyloides*. Med. Microbiol. Immunol. 195: 49-54.

Walker, MD, 2018: Reducing the risk of zoonotic infection. Conserv. Land Manag. 16, 2:19-22.

Wen, H, Vuitton, L, Tuxun, T, Li, J, *et al,* 2019: Echinococcosis: Advances in the 21st Century. Clin. Microbiol. Rev. 32: e00075-18.

Wilson, ME, 1991: A World Guide to Infections, Diseases, Distribution, Diagnosis. NY, Oxford, Oxford University Press.

Wishahy, AO, Rifaat, MA, Morsy, TA, El Naggar, BA, 1972: Toxoplasmosis in children with some neurological manifestations. J. Trop. Med. Hyg.75, 12:255-6, London.

Wołyniec, W, Sulima, M, Renke, M, Dębska-Ślizień, A, 2018: Parasitic infections associated with unfavorable outcomes in transplant recipients. Medicina (Kaunas). 54, 2:27-31.