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The Public Health and Clinical Importance of Amoebiasis

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Abstract

Amoebiasis also called amoebic dysentery, first described by Fedor A. Lösch in 1875, caused by Entamoeba histolytic has great clinical impotence and is of public health significance. Histolytica has a simple life cycle involving the infective cyst that ingested through contaminated food and water and vegetative trophozoite. The pathogenesis of Entamoeba histolytica have different events like cell death, inflammation, and invasion which are performed with the help of different molecules like lectin, Amoeba pores and cysteine protease, etc. 80-90% of people infected with Entamoeba histolytica are asymptomatic (intraluminal amoebiasis) and remaining to develop manifestation like amoebic colitis, toxic megacolon, ulceration, ameboma and another extraintestinal amebiasis like amoebic liver abscess, pulmonary, cardiac and cerebral abscesses if trophozoite reaches haematogenous to these sites. The global burden caused by amebiasis is widespread. Worldwide 50 million people are affected by this disease and 100000 deaths are reported annually. The highest burden of amebiasis is in developing countries, particularly in the tropics and subtropics, where there is inadequate hygiene and access to sanitation. Microscopy, serological and molecular methods can use for diagnosis. Pharmacological therapy and surgical intervention are recommended. As there is no effective vaccine, prevention emphasizes on sanitation and access to clean drinking water.

Keywords: Amoebiasis; Dysentery; Entamoeba histolytica; Contaminated food; Water; Sanitation

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Introduction

A pseudopod-forming, non-flagellated protozoan parasite Entamoeba histolytica causes an infection called amoebiasis or amoebic dysentery that can be asymptomatic and self-limiting (90%) and can also causes extensive mortality and morbidity globally by the occurrence of diarrheal disease and abscess formation in tissues of parenchyma such as liver(amoebic liver abscess), lungs (Pulmonary amoebiasis), heart (cardiac amoebiasis) and brain (cerebral amoebiasis) Amoebiasis was first described by Fedor A. Lösch in 1875, in St. Petersburg Russia Later, in 1903 Fritz Schaudinn has named Lösch's microorganism causal of the dysentery, Entamoeba histolytica **[1,2].**

Amoebiasis is the second leading cause of death disease, caused by parasite after malaria. In most areas of the world the prevalence of this infection is not known because of difficulty to identified *E. histolytica* and other amoebas with same structures as Entamoeba dispar and Entamoeba moshkovskii **[3].** Most diseases that show symptoms are caused by Entamoeba histolytica. Many years considered that Entamoeba dispar was considered non-pathogenic but later it was reported as pathogenic due to its presence in some cases of amoebic colitis and amoebic liver abscesses **[4].** Other four-nucleated structurally similar organisms, Entamoeba moshkovskii, has been seen in sewage as a free living but it is also enabled to colonize the human intestine **[5].**

Both species Entamoeba histolytica and Entamoeba dispar are found in two forms the hardy, infective cyst and the fragile, potentially pathogenic trophozoite fecal oral route is the main source of spread of these organisms. Some cases of spreading through sexually has also been found. **[6, 7]** As natural hosts of this disease are humans. When we ingest fecally-contaminated food or water then amoebic infection occur. Mature cyst encystation occurs in small intestine and trophozoite are released and move to large intestine. The trophozoite increase by process of binary fission and pass in feces and survive in external environment for some days to weeks due to cyst wall. There ingestion causes disease **[6, 7].**

The pathogenesis is mainly divided into three stages host cell death, inflammation, and parasitic invasion by involvement of different molecules like lectin, Amoeba pores, cysteine proteases and EhMIF **[8].** It causes the lysis of the tissues and proteolysis.

This disease is mainly found in developing countries like parts of Central and South America, Africa, and Asia because of poor sanitation, inadequate water treatment and low socio-economic status. **[9]** Worldwide 50 million people are affected by this disease and 100000 deaths reported annually **[10].** Demographic, behavioural, environmental and clinical characteristics that linked with disease are counted among the risk factors in developing countries **[11].** The amoebiasis also occurs in people in developed countries in travellers, immigrants and men who have sex with men **[2].**

Diagnosis can be done by direct microscopy of stools, body fluid or tissue sample for cysts and trophozoite. However, this organism is seen in only 30% of patient and is nonspecific for Entamoeba histolytica. Other test like culturing and is enzyme analysis are also used but these methods have limitations. These limitations are overcome by use of antigen detection or molecular method i.e., highly sensitive and important in differentiating Entamoeba histolytica from Entamoeba dispar and Entamoeba moshkovskii. (Tanyuksel and Petri, 2003). Symptomatic amoebiasis requires hydration and use of drugs such as metronidazole, tinidazole and diloxanide furoate etc. To prevent this disease avoid using contaminated water and foods and maintain good environment sanitation **[11].**

Life cycle

The life cycle of E. histolytica is simple and revolves around two stages, either cyst that cause infection or invasive trophozoite **(Figure 1).**

Infection begins when humans (natural host) ingest the cyst found in food or water contaminated with feces. **[12, 13]** It can also occur through person to person contact, swimming in contaminated bodies of water and exposure in endemic areas **[13-16]** where it can found on various material or on surfaces including human hand that can be contaminated with feces and lead to oral intake either directly or indirectly **[12, 14 , 15].** Once the cyst is ingested, through gastrointestinal track it reaches to small intestine and then to large intestine and undergo encystation, which releases trophozoites of 10–20µm in diameter in the lumen of the intestinal wall. The trophozoite reproduces through binary fission then penetrates the colonic mucosa, forming distinct flask-shaped ulcers. The trophozoites can then gain access to the hepatic portal circulation and spread to the liver, which produces an inflammatory reaction leading to necrotic hepatocytes and subsequent abscess formation with a characteristic 'anchovy-paste' exudate It also disseminates to distant sites such as the brain and lungs hematogenously. Symptoms may occur within weeks or years after ingestion. Cysts and trophozoites are passed in stools. As cyst has protective all so it can survive in environment from days to week and is responsible for further transmission of the parasite. but trophozoite cannot **[17, 18].**

Pathogenesis

The pathogenies of E. histolytica can be classified as host cell death, inflammation, and parasitic invasion. The different mechanism like induction of programmed cell death, phagocytosis, and trogocytosis are used by trophozoite to kill the host cell **[19].**

The pathogenesis by *E. Histolytica* begins when parasite adhere to colonic mucosal layer through Gal / GalNAc lectin k by targeting the O-linked polysaccharide side chains of mucin on colonic epithelial

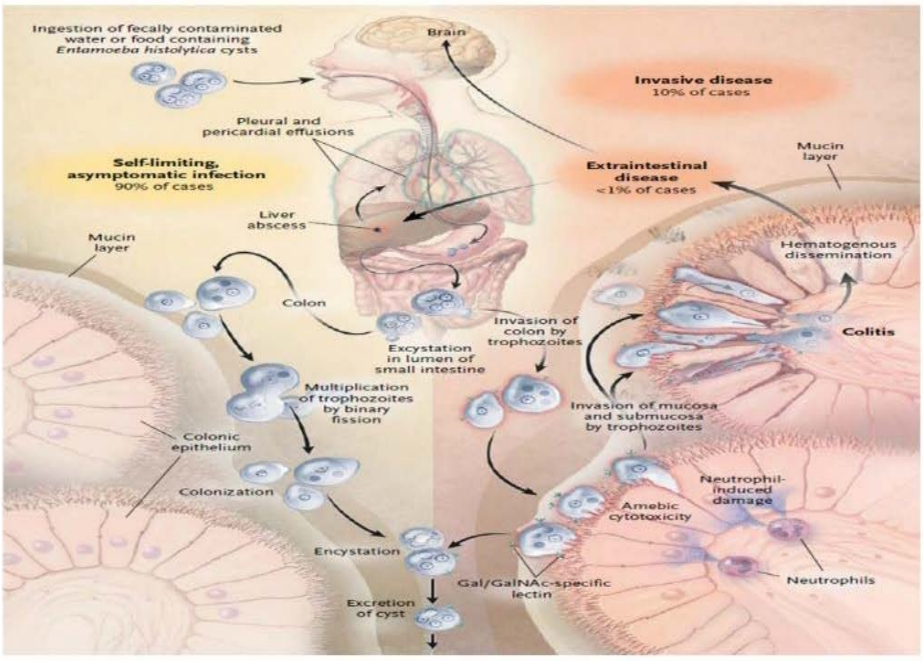

Figure 1 Life cycle of E. histolytic (Ayed L Ben and Sabbahi, 2017).

cells (Tsukui and Nozaki, 2016). The mucin secretagogue is produce by *E. Histolytica* causes hyper secretion of mucus from goblet cell for the diminution of mucin stores **[20].** Several other molecules involved in the pathogenesis of amebiasis, namely Amoeba pores that insertion into the membrane of target cells causing their lysis by forming ion channels or pores in it. The hydrolytic enzymes, cysteine proteases cause hosts epithelial and inflammatory cells destruction and subsequent invasion. During E. histolytica infection, 90% of all cysteine protease activities is by CP-1, CP2 and CP-5. The CP-5 is the most predominant in degradation of the epithelial mucus layer. The secretion of cysteine protease-5 induces the cleavage of C-terminus of the MUC2 protein, which results in protective mucin barrier degradation, later invasion and increased hyper permeability of the gut. *E. Histolytica* can also invade the host defense by Peroxiredoxin, alcohol dehydrogenase, and lipopeptidophosphoglycan **[21].** For invasive disease, the destruction of extracellular matrix is necessary. E. histolytica produces E. histolytica migration inhibitory factor (EhMIF), which causes mucosal inflammation and production of matrix metalloproteinase (MMPs) **[22].** MMPs break down the extracellular matrix in the gut to promote cell migration and are overexpressed, thus EhMIF further contributes to disseminate disease by producing MMPs **[23, 24]** during the amoebic pathogenesis, and trogocytosis and phagocytosis of cellular debris are important processes. A trogocytosis is new mechanism of pathogenicity, reported for E. histolytica trophozoites, which consists of the ingestion of pieces of living cells It begins to occur within one minute of contact with the host cell. Host cells are still alive when the process starts, but eventually die due to loss of membrane integrity. Trogocytosis of amoeba include physiological temperature, rearrangement of amoeba actin, Gal / GalNAc lectin, EhC2PK, and signaling-PI3K. Cell death after amoeba trogocytosis can be caused by accumulated physical damage to bitten cells (Ralston, 2015). According to the current studies the interaction among the host's intestinal flora and *E. histolytica* can facilitate the pathogenic behavior, generating more virulent and enter pathogenic bacteria cause increase in Gal/GalNAc lectin expression in trophozoites of *E. histolytica* which results in increased capacity of adhesion and cytopathic effects. In the presence of certain gut bacteria, production of proinflammatory cytokines was also increased, causing more impairment of epithelium and facilitate trophozoite invasion **[25, 26].**

Clinical manifestation

In *E. Histolytic* infection, only 10%–20% people develop symptomatic infection remaining 80%-90% are asymptomatic. The reasons for this are poorly understood but result from an interaction of several factors related to parasite, host, and environment **[27].**

Intraluminal Amebiasis

Luminal amebiasis is an asymptomatic infection. In case of personal history of travel to/arrival from an endemic area or a history of this in either a household or sexual contact, Screening for asymptomatic infection is recommended **[28].**

Amebic Colitis

Amebic Colitis is symptomatic intestinal infection with sub-acute onset. The characteristic symptoms of amoebic colitis are watery or bloody diarrhoea, with abdominal cramps, pain/tenderness, and weight loss. Disease may be limited to the ascending colon or cecum. If diagnosis and treatment is not timely, serious complications such as fulminant infection which can result in massive areas of colonic involvement with perforation and peritonitis, toxic megacolon and fistulising perianal ulcerations can occur. A risk factor for the development of fulminant forms of amebic colitis is corticosteroid use diabetes mellitus, alcoholism, malignancy/chemotherapy, and pregnancy. Patients could be toxic in appearance, febrile, and hypotensive, with profuse bloody diarrhoea, abdominal pain, distension, and signs of peritonism. Toxic megacolon occurs in a patient with amebic colitis and heavy use of the ant motility agent lope amide The toxic megacolon development has linked to corticosteroid usage and is impassive to anti amoebic therapy, requiring instant surgery The most uncommon manifestation occur in amoebic colitis is formation of ameboma. the development of tumor-like granulation tissue in the colonic lumen, can mimic colonic cancer It is characterized by pain and swelling in the right iliac fossa, or with symptoms of bowel obstruction.

Disseminated Amebic Disease

The most common extraintestinal amoebiasis is amebic liver abscess. Trophozoites enter to the liver by mean of hepatic circulation, forming micro abscesses that finally combine to form a well-circumscribed amebic liver abscess, generally in the posterior right lobe **[28].** The abscess contains inflammatory debris, dead hepatocytes, and amoebic trophozoites surrounded by a rim of connective tissue, with a characteristic chocolate-colored fluid "anchovy paste" exudate **[29]** The two most common symptoms of ALA are right hypochondriac pain or constant, aching right upper quadrant pain , and fever (38.5 to 39.5°C), which generally presents within 2-4 weeks in 50-80% of individuals(Kannathasan et al., 2018; Bhatia and Sundaram, 2019; Patients may also suffer with nausea, vomiting, weakness, weight loss, and referred pain to the shoulder in some cases. Patients may or may not present with jaundice **[30].** It also has gastrointestinal symptoms typically without concurrent dysentery. Hepatomegaly with point tenderness over the liver can often be detected **[31]** Another uncommon complain in ALA is cough and right sided pleural pain. It is generally due to associated pleural effusion and compression collapse of the underlying lung parenchyma (Sharma *et al*., 2010). Leucocytosis, transaminitis, and elevated alkaline phosphatase occur during ALA and it can use in laboratory evaluation. Imaging reveals an abscess, typically on the right hepatic lobe [31, 32]. Anemia and hypoalbuminemia are quite common in amoebic liver abbesses in comparison to pyogenic or bacterial abscesses **[33]** the second most common extraintestinal organ affected are lungs. Pulmonary amoebiasis generally occurs by direct extension of an ALA or it also occur by direct haematogenous spread from intestinal lesions or by lymphatic spread **[34,35]** The most affected part of the lung is the right lower or middle lobe. Patients present the symptoms that are fever, haemoptysis, right upper quadrant pain, and referred pain to the right shoulder

or intrascapular region. When a liver abscess ruptures into the pleural space, pulmonary abscesses, broncho hepatic fistula and empyema can occur. Patients usually present with brown colour "anchovy sauce-like" pus or sputum **[36].**

Cardiac infection is another occasional complication in amoebiasis. It occurs due to amoebic liver abscess rupture and spread to the pericardium, which causes end pericardial rupture which leads to cardiac tamponade, or slow onset of pericardial effusion. It generally occurs when abscess is in the left lobe of the liver, which is rare, as most cases posterior right lob is affected It can present acutely with cardiac tamponade resulting from purulent pericarditis, or with a slowly accumulating pericardial effusion. Symptoms include severe chest pain, shortness of breath, and edema from congestive heart failure or constrictive pericarditis Inferior vena cava (IVC) thrombosis is another extremely rare complication of ALA Mechanical compression of the IVC by a large hepatic abscess or by erosion from a posterior liver abscess can lead to embolism of the IVC and thromboembolic disease of the lungs (Shamsuzzaman and Hashiguchi, 2002; McKenzie *et al*., 2015). The exceedingly rare and lethal complication is cerebral amoebiasis. Which occur by Haematogenous spread to the brain? Worldwide, the frequency of cerebral infections caused by *E. histolytica* is from 0.6% to 8.1% (Barrera *et al*., 2012; Cruz *et al*., 2004). *E. histolytica* produces brain abscess, with a period of incubation from a few days to several months

Epidemiology of amebiasis

E. Histolytica has miscellaneous distribution and the global burden caused by amebiasis is widespread. The highest burden of amebiasis in developing countries, particularly in the tropics and subtropics, where there is inadequate hygiene practices and access to sanitation **[35].** Worldwide, 35–50 million symptomatic cases occur annually, leading to approximately 55,000 deaths In the developing parts of Central and South America, Africa, and Asia, amebiasis is endemic. The incidence of amebiasis is low, but amebiasis-related deaths still usually occur, accounting for at least 5 deaths per year in the United States The travellers and immigrants, who returns from endemic countries are mostly affected from amebiasis in united states In developing countries, the exact burden of amebiasis is difficult to measure. The geographic region, study design, size of sample, incubation, symptom and symptom, and the sensitivity of the diagnostic modality used can affect the report. In addition, diagnostic capacities and surveillance are often less in areas where E. histolytica is endemic (Esquivel et al., 2015). The estimated prevalence in Pakistan is 13.6% to 63.8%. Studies on amoebiasis in Pakistan are based on microscopic examination of feces which results in some misinterpretation, but still significant work has done in Pakistan **[35-38].**

Diagnosis of Amoebiasis

In endemic areas, patients can be diagnosed for intestinal amoebiasis by observing the clinical signs and symptoms like gastrointestinal discomfort and watery or bloody diarrhoea **[38]** The researchers had used several laboratories based for the diagnosis of amoebiasis like microscopic examination, serological methods including ELISA, indirect hem agglutination assay and latex agglutination assay **[39]** Antigen detection and molecular tests. Often a combination of tests is required to diagnose the amoebiasis.

Microscopy

Different microscopic techniques like wet preparation e.g direct saline wet mount, concentration, and permanently stained smears are employed in a diagnostic clinical laboratory for the identification of *E. histolytica/E. dispar/E. moshkovskii* in feces **[40].** The stool sample should be examined within one hour of collection for identification of motile trophozoite but it there is any delay in examination then stool sample should be preserved in polyvinyl alcohol (PVA), Schaudinn's fixative or sodium acetate-acetic acid-formalin (SAF) (Garcia and Shimizu, 1998). The trophozoites are more likely to be observed in loose stools which contain mucous, pus and occult blood. Whereas cyst can be observed in both formed and loose stool. Stool specimens can be observed either unstained or stained with Lugol's or D'Antoni's iodine which makes the nucleus perfectly visible By using stains like methylene blue, Giemsa, Wright's and iodine-trichrome, the morphology, size and number of nuclei can be clearly observed. The modified iron haematoxylin and Wheatley's trichrome stains are recommended for routine use Presence of ingested red blood cell in cytoplasm (erythrophagocytosis) is considered as diagnostics for dysenteric patients. This can also use to distinguish the *E. histolytica* and *E. dispar* In case of ALA, microscopic examination of the thick brown pus (anchovy paste) contains dead and deformed hepatocytes, red blood cells and some polymorphs. The common staining techniques hematoxylins and eosin (H&E), periodic-acid Schiff (PAS)and immune staining are used to visualize the morphological changes in the liver tissue and differentiates the amoebas against the surrounding cells (Bancroft and Gamble, 2008). Advantage of microscopy is that it is widely available and require Minimal equipment and reagents but it is time-consuming, has poor sensitivity and specificity, multiple stools need to be submitted, cannot differentiate from other Entamoeba spp and skilled observer required because inadequate training and diagnostic testing may lead to misdiagnosis.

Culture method

From more than 80 years, culture techniques are being used for isolation of Entamoeba species by using xenic and axenic cultural media. The xenic cultivation was introduce by Boeck and Dr bohlav in 1925. The xenic diphasic media include egg slant medium now modified and known as Locke-egg (Clark and Dimond, 2002) still in use today particularly in research studies. The axenic cultivation of *E. Histolytica* was introduced by Diamond in 1961 Fecal specimens, rectal biopsy specimens, and liver abscess aspirates can be used for culturing of *E. Histolytica* (Blessmann, 2002). The cultivation of *E. Histolytica* in a clinical diagnostic laboratory is not feasible as a standard procedure. It is difficult, expensive, labour-intensive and. less sensitive than microscopy. It is not recommended as routine diagnostic procedure for the detection of **Entamoeba** species because of overgrowth of bacteria fungi and other protozoans **[40].**

Isoenzyme analysis

Iso enzyme analysis of cultured amebae by means of zymodeme enzymes assist in the differentiation of *Entamoeba* species A zymodeme is a group of amoeba strains that have the same electrophoretic pattern and mobilities for numerous enzymes such as malic enzyme, hexokinase, glucose phosphate isomerase, and phosphoglucomutase isoenzyme Out of 24 different zymodemes 21 are from human isolates (nine *E. histolytica* and twelve *E. dispar*) and three from experimentally cultured amoeba strains. To differentiate the two Entamoeba species, band are counted e.g. there are three zymodeme bands for *E. histolytica* (II, XIV, and XIX) and one for *E. dispar* (The isoenzyme analysis is difficult and time consuming It requires four to ten days to grow significant number of trophozoite and is not always successful The overgrowth of bacteria, fungi and protozoan is another major problem during isoenzyme analysis. It sometime also gives false negative results of some microscopy positive stool sample **[34, 36].** Because of its less sensitivity it is not recommended for routine use and is replaced by molecular diagnosis

Antigen Detection

The limitations regarding diagnosis of amoebiasis through methods like stool microscopy is overcome by antigen detection test that is easy to use but particularly in in low endemic areas, it has variable sensitivity and specificity For antigen detection in fecal samples, ELISA is developed by investigators. It has advantages than other methods being used for diagnosis of amoebiasis like It can differentiate *E. histolytica* from *E. dispar,* It has excellent sensitivity and specificity and Non experience laboratory personnel can also use this test *E. Histolytica* specific antigen-based ELISA kits use monoclonal antibodies for detection of antigen like Tech Lab *E. Histolytica* I and Entamoeba CELISA-PATH which detect Gal/GalNAc lectin, Optimum S kit which detect Serine-rich antigen and ProSpecT *E. Histolytica* micro plate assay which detect EHSA antigen (Tanyuksel and Recently, an *E. Histolytica* QUIK CHEK immunochromatographic (IC) assay, was approved by the US Food and Drug Administration (FDA). It is simple to perform and has a quick turnaround time **[26]** Antigen detection test may have high sensitivity in endemic areas but reduced sensitivity in nonendemic areas. It is simple to perform, have rapid turnaround time, and is commercially available but disadvantage is that it has poor sensitivity for amebic liver abscess. It requires fresh, not fixative preserved stool for analysis.

Antibody Detection test

For the detection of anti-amoebic antibodies several assays are commonly used such as ELISA, indirect hem agglutination (IHA), indirect immunofluorescence assay (IFA), latex agglutination, immune electrophoresis, counter immune electrophoresis (CIE), amoebic gel diffusion test, immunodiffusion and complement fixation test **[26, 28]** The most common technique that has been used to investigate the epidemiology of symptomatic amoebiasis due to its reliability and ease of performance is ELISA **[40]** Some commercially available antibody detection assays for extraintestinal amoebiasis are IHA Cellognost-Amoebiasis, Novagnost Entamoeba IgG, Bichro-Latex Amibe, I.H.A. Amoebiasis, Amoebiasis Serology microwell II EIA and RIDASCREEN IgG Entamoeba. Antibody detection method has high sensitivity and specificity but serology remains positive for years after resolution of infection, so less helpful in endemic areas and more useful for travelers. Antibody response is often detectable by the time of presentation but may need to be repeated in 7–10 days if initially negative (Shirley et al., 2018).

Molecular Biology-Based Diagnostic Tests and PCR

To solves the problems of microscopic or culture-based diagnosis and take advantage of the sensitivity, specificity, and simplicity of newer techniques, molecular biology-based technology has become commonly used For differentiation and detection of the Entamoeba species in stools, tissues and liver lesions aspirates, different variants of DNA amplification techniques are used like conventional PCR, nested PCR, real-time PCR, multiplex PCR and loop-mediated isothermal amplification (LAMP) For recognition and discrimination of the three Entamoeba species, (*E. Histolytica/E. dispar/E. moshkovskii*) many genes are targeted like small subunit rRNA, gene encoding a 30-kDa protein, DNA highly repetitive sequences, haemolysin gene (HLY6), cysteine proteinase, gene encoding serine-rich E. histolytica SREHP protein, actin gene and tandem repeats in extra chromosomal circular DNA (Freitas et al., 2004; Vermeil et al., 2004) some examples include like Conventional PCR target Extra-chromosomal circular DNA of E. histolytica, 30-kDa antigen gene of pathogenic E. histolytica, HLY6 gene, Nested PCR target 16S-like RNA, Real-time PCR target 18S rRNA, small subunit rRNA gene 16 s rRNA

The restriction site polymorphism analysis method involving amplification followed by restriction fragment length polymorphism analyses of the small- and large-subunit rDNA, is a remarkably effective tool to evaluate different Entamoeba species Molecular methods have high sensitivity and specificity but expense and requirement for technical expertise may limit use in resource-limited settings.

Rapid diagnostic test

The preferred diagnostic tool in developing countries is rapid diagnostic tool that avoids the need of expensive equipment (Peeling and Mabey, 2010; Chin et al., 2013). Some rapid diagnostic tests for intestinal amoebiasis are RIDA®QUICK Cryptosporidium/Giardia/Entamoeba Combi, RIDA Quick Entamoeba test, *E. histolytica* Quik Chek, Triage Micro Parasite Panel and Prototype of lateral flow dipstick test (Saidin et al., 2019). The Triage Micro Parasite Panel are rapid (less than 15 min) and results can be easily read and interpreted on the test device. Furthermore, minimal training on the assay is required. Additionally, there is no cross-reactivity with the Triage Micro Parasite Panel with other intestinal parasites (*A. lumbricoides, E. coli*) that identified in stool samples (Dimond and Clark, 1993). The TPP kit has limitation that it cannot differentiate *E. histolytica, E. dispar* and *E. moshkovskii* and can only use for fresh or fresh-frozen non-preserved stools. In case of ALA, there is no rapid diagnostic test. However, two tests that are lateral flow dipstick test and immunochromatographic test seemed to have good potential for rapid diagnosis of ALA.

Imaging

To detect liver abscess ultrasonography (cystic intrahepatic hypoechoic lesion), abdominal computed tomography (non-enhancing centre surrounded by a rim of inflammation), and magnetic resonance imaging are good modalities **[36, 37].**

Treatment

Treatment of amoebiasis includes pharmacological therapy, surgical intervention, and preventives measures. Metronidazole and imidazole are drug of choice for treatment for amebic colitis and amebic liver disease. For an uncomplicated amoebic liver abscess surgical drainage is unnecessary and should avoided. If metronidazole is not effective after 72 hours of treatment the abscess should be drained in this case. Aspiration is largely being replaced by percutaneous catheter drainage **[37].** In patients unsuitable for percutaneous drainage (elderly, frail, septic shock, multilocular cysts) laparoscopy is the preferred option.

Amoebiasis can be prevented by following rudimentary hygienic habits like proper washing of hands with soap after using of toilet and before handling any food product, proper cleaning of bathrooms and toilets, use of purified drinking water either by boiling, filtration or chlorination **[40].**

Conclusion

Amoebiasis still remains problem for public health and of great clinical importance especially in developing countries. Advances in molecular epidemiology and pathogenesis have advanced our understanding of amoebiasis causative agent *E. histolytic*, but as most patients are asymptomatic (80-90%), diagnosis and treatment can be challenging for clinicians, potentially leading to continuous spread of the disease. Still there is no vaccine so preventive measures like good sanitation and hygienic lifestyle should be followed.

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