REVIEW ARTICLE

INTERNATIONAL JOURNAL OF RESEARCH IN PHARMACEUTICAL SCIENCES

Published by JK Welfare & Pharmascope Foundation Journal Home Page: https://ijrps.com

A general review on the amoebiasis

Fatima Ibrahim Mohammad*

Department of Environment, College of science, University of Al-Qadisiyah, AL-Diwaniyah, Qadisiyah, Iraq

Article History:

Received on: 11.09.2018
Revised on: 18.12.2018
Accepted on: 22.12.2018

Keywords:

Entamoeba histolytica,
Amoebiasis,
Parasitic diseases

ABSTRACT

Entamoeba histolytica parasite causes amoebiasis or Amoebic dysentery, this parasite spreads all over the world, especially in the tropical and subtropical regions and spreads among the poor. This may be due to poor nutrition or unhealthy conditions. This disease is one of the most common parasitic diseases in the world. *E. histolytica* is responsible for the deaths of more than 100, 000 people annually, and this ranks third after malaria and Schistosoma in increasing the number of deaths caused by the infection of the primary parasite. *E.histolytica* has the ability to invade the intestinal mucus layer and spread to other organs especially the liver, causing the amoebic liver abscess.

* Corresponding Author

Name: Fatima Ibrahim Mohammad
Email: fatima.mohammad@qu.edu.iq

ISSN: 0975-7538

DOI: https://doi.org/10.26452/ijrps.v10i1.1872

Production and Hosted by
IJRPS | https://ijrps.com

INTRODUCTION

Amoebiasis is a gastrointestinal disease transmitted to humans by ingested food and water contaminated with the faecal amoeba (Botelho *et al.,* 2011), also the weather and the extreme events such as floods, drought and rainfall can impact the prevalence of infection (Jaykus *et al.,* 2009). By through flow of the wild animals and pet dung with surface and groundwater (Moreno, 2006). This disease occurs without symptomatic in an infected person, but this can be their life-threating because it diarrhoea may lead to severe drought (Botelho *et al.,* 2011). This is disease causes deaths and rank third after malaria and Schistosoma (Stanely, 2003).

World Health Organization (WHO) refered to that E. *histolytica* cause mortality in 100, 000 persons and symptomatic disease in 50 million (Lozano *et al.,* 2012). There are many species belong to Entamoeba that is: *Entamoeba histolytica*, *E. polecki, E.hartmani*, *E.coli*, *E.dispar* and *E.gingivalis* (Ash and oriheli, 1980).amoebiasis is a disease, in fact, human and other animals. In animals, the cysts multiply in the small intestine and then move to the colon where the amoeba attack the epithelial lining (Schuster and Visvesvara, 2004), infection of animals such as (dogs, cats, pigs, apes) is rare and these hosts act either sources or reservoirs, and this refers to the animals do not excrete cysts with their faeces therefore cysts cannot survive outside the animal body, the humans consider are the only reservoir of *E. histolytica* (Ayed and sabbahi, 2015).

Morphological features of the parasite

Entamoeba histolytica pass through its life cycle in three distinct forms that are trophozoite, precyst and cyst

Trophozoite stage: This phase is moving, nourishing and active with a diameter of 10-60 µm and is present in the tissues and the liquid faeces (Brooks *et al.,* 2004) and causes the pathogenicity of humans by attacking the epithelial cells of the mucous layer lining the intestinal cavity as it has the ability to migrate to other organs of the body to cause injuries outside the bowel. The parasite movement is the main factor in pathogenicity, attack on tissues and acute disease development. The trophozoite movement occurs through an ex-

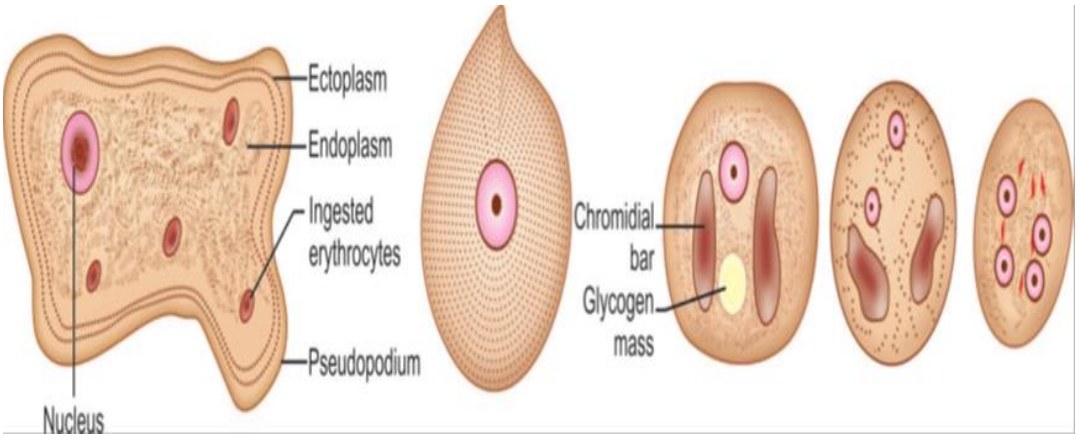

Figure 1: Entamoeba histolytica. A. Trophozoite; B. Precystic stage; C. Uninucleate cyst; D. Binucleate cyst; E. Mature quadrinucleate cyst

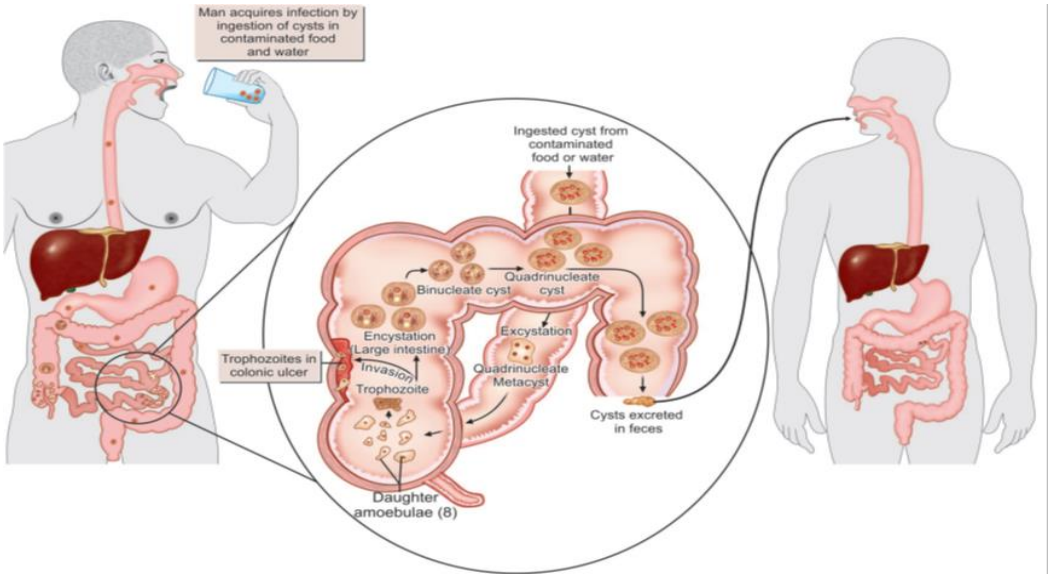

Figure 2: Life cycle of Entamoeba histolytica

tension of the cytoplasmic growth known as pseudopodium and movement are dependent on the dynamic of actin-myosin.

The trophozoites are clear, colourless and irregular, surrounded by a rubbery, semi-permeable and viscous membrane is known as plasmalemma, the cytoplasm is characterized by two regions Ectoplasm is clear, Endoplasm is granular and contains many food vacuoles containing red blood cells and bacteria that are seen in the stool and ulcers as well as contractile vacuoles, Endoplasm contain a single spherical or vesicular nucleus surrounded by thin nuclear membrane. The inner edge of this membrane is lined with thin and regular chromatin granules, the nucleus is characterised by the presence of a small central body is called Endosome. The trophozoite is fragile and easy to destroy in the external environment and can convert inside the intestinal into precyst that contain one nucleus and these mature to the cystic stage (Tanyuksel and Petri, 2003).

Pre cystic stage: It is characterised by being small in size, oval shaped, nonmoving, non-nutritious and does not cause human disease and no contains food vacuoles. Only red blood cells, exist in the colon cavity and is rarely found in tissues. This stage contains one nucleus, glycogen vacuoles and chromatid bodies with rounded ends and then the nucleus is divided inside the cyst to produce cysts contain 4 nuclei that represent the infectious phase (Kotpal, 1994).

Cystic stage: It is responsible for infection and its diameter 10-15 µm and contains 1-4 nuclei, centrosome and regular peripheral chromatin, cytoplasm is clear and contains glycogen vacuoles, one or more chromatin bodies with round ends that disappear later. The parasite surrounds itself by layer or wall of carbohydrate, that contains a mixture of protein and kitin so it can survive outside the host body for several weeks or months especially in wet conditions (Tanyuksel and Petri, 2003). In inadequately treated wastewater, the cysts can remain alive for several weeks (Fletcher *et al.,* 2012).

Life cycle: *Entamoeba histolytica* completes its life cycle in one host and does not need an intermediate host, its infect human and other animals like monkeys (Verweji *et al.,* 2003; Regan *et al.,* 2014).

The infection occurs when ingestion of mature cysts with contaminated water, food or hands, when the mature cysts reach the terminal part of small intestine occurs excystation (Haque *et al.,* 2003).

When the cyst reaches the small intestine cavity, the wall destroys by the trypsin enzyme and the parasite comes out of the cysts to give quadrinucleate amoeba (Cho and Eichinger, 1998), divided again to produce 8 trophozoite uninucleated and migrate to the large intestine, attacks the mucous lining then the trophozoite that multiplied by the binary fission where the nucleus is divided by mitosis followed by cytokinesis to produce two daughter cells grow rapidly. This is called amoebic dysentery; amoeba may migrate through the bloodstream to infect sites outside the intestine especially liver, when exposure to inappropriate conditions occurs Encystment in large intestine then cysts move out with host stool to outside to infect a new host by ingestion contaminated food and water (Laughlin and Temesvari, 2005).

Pathogenesis

Amoebiasis usually occurs without symptoms but the trophozoites in some cases invasive the intestinal mucosa and causes dysentery, ulcers and that may be threat life of the infected persons (Sateriale and Hutson, 2011).

The most common infection invasion of amoebiasis is an amoebic liver abscess but may extend to other organs such as cerebral, renal, pleuropulmonary, genitourinary and cutaneous sites (Gamboa *et al.,* 2011). There are three levels governed of invasive trophozoites that are adherence, lysis and phagocytosis of the target cell (Sehgal *et al.,* 1996). In the first invasion, the trophozoite adheres to epithelial cells inside the intestinal mucous by lectine which consider the main factor to the adhesion (Lejeune *et al.,* 2009).

Amoebapores are polypeptides released by the parasite and present in the trophozoites cytoplasm (Gonzalez *et al.,* 2008). amoebapore which is capa- ble of depolarizing target cells, and forming ion channels, or pores in lipid membranes, was discov- ered in *E. histolytica* (Leippe and Herbst, 2004 ; Young *et al.,* 1982). Amoebapore assembles inside host cell membrane to trigger cell death (Rosen- berg *et al.,* 1989). Amoebapore form pores in the membrane of eukaryotic cell or bacteria and re- sults in lysis of the target cell and cell necrosis of eukaryotic cells (Blessmann *et al.,* 2002).

Cysteine proteinases consider as virulence factors of *E.histolytica* by breach of the mucous barrier (Lejeune *et al.,* 2009). The parasite breaches the epithelial barrier and the mucous by secreted the proteolytic enzymes and thus facilitate the parasite penetration inside the tissue (Que and Reed, 2000) ulcers are formed by the combination of these molecules and followed transmission amoeba to the liver and other organs (Stanley, 2001).

Treatment: used to treatment amoebiasis three classes of the drug.

Tissue amoebicides: used chloroquine and Emetine to systemic treatment infection while it's less effective in the intestine.

Luminal amoebiasis: used iodoquinol, tetracycline, paromomycin and Diloxanide furoate to treatment the intestinal lumen while not used in tissues.

Tissues and luminal amoebicides: used Metronidazole, ornidazole and tinidazole to treatment amoebic liver abscess and amebic colitis (Paniker, 2013).

CONCLUSION

Amoebiasis is intestinal protozoan infections occur without symptoms. Symptoms can range from mild diarrhoea to dysentery with blood and mucus. Amoebiasis is caused by amoebic liver abscess but may extend to other organs. *Entamoeba* species are classified based on the characteristics of this parasite such as the size of trophozoites, cysts, the numbers of nuclei in mature cysts, the composition and location of the nucleus, *E. histolytica* is only humans pathogen while other species are not pathogenic.

REFERENCES

Al-Grawi, E.D.C., and G.R.L. Al-Awsi. 2018. "Expression of CDKN2A (P16/Ink4a) among Colorectal Cancer Patients: A Cohort Study." Journal of Pharmaceutical Sciences and Research 10 (5(.

Ash, L. and Orihel, C. (1980). Atlas of human parasitology. American society of clinical pathologists, Chicago 2nd edition, pp 22-32.

Ayed, L.B. and Sabbahi, S. (2015). Entamoeba histo- lytica. National Institute of Agronomy, Tunis, pp:36.

Blessmann, J.; VanLinh, P.; Nu, P.A.; Thi, H.D.; Muller-Myhsok, B; Buss, H. and Tannich, E. (2002). Epidemiology of amoebiasis in a region of high incidence of amebic liver abscess in central Vietnam.Am.J. Trop.Med. Hyg;66:578-583.

Botelho, F., Lopes, R., Franca, J. and Gomes, M. (2011). Advances in Amoebiasis Research Emphasizing Immunological and Oxidative Aspects. Research Journal of Parasitology, 6: 1-17

Brooks, G.F.; Butel, J.S. and Morse, S.A. (2004). Medical microbiology.23 th ed.McGraw-Hill companies, United States of American, New York.PP:671-674.

Cho, J and Eichinger, D (1998). Crithidia fasciculata induces encystation of Entamoeba invadens in a galactose-dependent manner. J Parasitol. 84, pp. 705–710.

Falah Mahdi Ali, Ahmed Ali Obaid, 2018. Prostate Vaporization by Diode Laser for Patients With Benign Prostatic Enlargement. Asian Journal of Pharmaceutical and Clinical Research, Vol 11 Issue 12 December 2018 Page: 523-525. DOI: 10.22159/ajpcr. 2018.v11i12.29572

Fletcher, SM, Stark, D, Harkness, J and Ellis, J (2012). Enteric protozoa in the developed world: a public health perspective. Clin Microbiol Rev. 25, pp. 420–449.

Gamboa, N., Rohana, P. and Martinez, A. (2011). Differentiation of E. histolytica; a possible role of enolase. Experimental Parasitology Journal, Centre for research and advanced studies, Mexico city, 123: 65-71.

Gonzalez, A., Monterrubio, D., Nequiz, M., Lopez, R. and Olivos, A. (2008). A localisation of Entamoeba histolytica amebopore in amebic liver abscesses in hamsters. Annals of New York Academia of Science, 1149: 375-379.

Haque, R, Huston, CD, Hughes, M, Houpt, E and Petri, Jr, WA (2003). Amebiasis. N Engl J Med. 348, pp. 1565–1573.

Jaykus, LA, Woolridge, M, Frank, JM, Miraglia, M, McQuatters-Gollop, A and Tirado, C (2009). Climate change: implications for food safety. Rome, Italy pp. 49.

Kotpal, R.L.(1994). Protozoa: A textbook for college and university student 11th ed. National Press, Meerut, India. PP.54-65.

Laughlin, R.C. and Temesvari, L.A.(2005).The life cycle of Entamoeba histolytica and the clinical manifestations of infections in humans. Expert. Rev.Mol.Med., 7.

Leippe, M. and Herbts, R.(2004).Ancient weapons for attack and defence: the pore-forming polypeptides of pathogenic enteric and free-living amoebid protozoa.J Eukaryot Microbiol;51:516-521.

Lejeune, M., Rybicka, J. and Chadee, K. (2009). Recent discoveries in the pathogenesis and immune response toward Entamoeba histolytica. Future Microbiology, 4: 105-110.

Lozano, R, Naghavi, M, Foreman, K, Lim, S, Shibuya, K, Aboyans, V et al. (2012). Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 380, pp. 2095–2128.

Moreno, A.R. (2006). Climate change and human health in Latin America: drivers, effects, and policies. Regional Environment Change. 6, pp. 157–164.

Paniker, CK Jayaram. (2013).paniker's Textbook of medical parasitology .seventh edition. Indian Council of Medical Research, New Delhi, India. Page 276.

Prevalence of Entamoeba histolytica and Entamoeba dispar in northern Ghana. Tropical Medicine and International Health. 8, pp. 1153–1156.

Que, Reed, S.L. (2000). Cysteine proteinases and the pathogenesis of amoebiasis. Clin. Microbial. Rev.,13:196-206.

Regan, CS, Yon, L, Hossain, M and Elsheikha, HM (2014). Prevalence of Entamoeba species in captive primates in zoological gardens in the UK. PeerJ. Pp. 2.

Rosenberg, I.; Bach, D.; Loew, L.M. and Gitler, C. (1989). Isolation characterization and partial purification of a transferable membrane channel (amoebapore) produced by Entamoeba histolytica;Mol.Biochem.parasitiol.237-248.

Sateriale, A. and Huston, C. (2011). A sequential model of host cell killing and phagocytosis by Entamoeba histolytica. Journal of Parasitology Research, Hindawi publisher's corporation, University of Vermont, USA 1: 1-10.

Schuster, FL and Visvesvara, GS (2004). Opportunistic amoebae: challenges in prophylaxis and treatment. Drug Resistance Updates. 7, pp. 41–51.

Sehgal, D., Bhattacharya, A. and Bhattacharya, S. (1996). Pathogenesis of infection by Entamoeba histolytica. Journal of Biological Sciences, 21: 423-432.

Shamran, A. R, Shaker, Z. H, Al-Awsi, G. R. L, Khamis, A. S, Tolaifeh, Z. A. and Jameel, Z. I, 2018. Rapd-PCR is a good DNA fingerprinting technique to detect phylogenetic relationships among Staphylococcus aureus isolated from different sources in hilla city, Iraq. Biochemical and Cellular Achieves. Vol. 18, Supplement 1, pp. 1157-1161.

Stanley, S., (2001). Pathophysiology of amoebiasis. Trends of Parasitology 17: 280-285.

Stanley, S.L.Jr.(2003). Amoebiasis, Lancet, 361(9362):1025-1034.

Tanyuksel, M. and Petri, W.A. (2003). Laboratory Diagnosis of Amoebiasis.Clin. Microbial. Rev., 16:713-729.

Verweij, JJ, Oostvogel, F, Brienen, EAT, Nang-Beifubah, A, Ziem, J and Polderman, AM (2003). Short communication:

Young, J.; Young, T.M.; Lu, P.L.; Unkeless, J.C.and-Cohn, Z.A. (1982). Characterisation of a membrane pore-forming protein from Entamoeba histolytica; J.Exp.Med.1677-1690.