

Symptomatic Assessment of Human Giardiasis in Basrah

Ghazi Yaqoob Al-Emarah¹ and Rasah Khalil Al-Saad^{2*}

¹Dean and Assistant Professor, Parasitology / Veterinary Medicine / University of Basrah, Basrah, Iraq

²Lecture, Parasitology / Veterinary Medicine / University of Basrah, Basrah, Iraq

*Corresponding author: Rasah Khalil Al-Saad, Lecture, Parasitology / Veterinary Medicine / University of Basrah, Basrah, Iraq, Tel: 964-40 413828; Fax: 964-40 410574; E-mail: masterr78@ymail.com

Rec date: Jun 13, 2014; Acc date: Jul 14, 2014; Pub date: Jul 20, 2014

Copyright: © 2014 Al-Emarah GY, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

Background: Giardia was single celled microscopic protozoan parasites that cause enteric disease in human. It was recognized as the most common parasitological cause of diarrhea in human patients and is a major concern to drinking water authorities, as it is a frequently diagnosed waterborne infection.

Aim: To assess symptomatically human giardiasis in Basrah population.

Materials and Methods: This work was conducted from September 2012 to May 2013. A total 1344 stool samples were collected from human cases.

Results: About 405 cases of human giardiasis were diagnosed out from 1344 patients. The percentages of each symptoms are as follow diarrhea 75%; foul smelling greasy stool 31.8%; bloating 22%; abdominal cramp 85%; anorexia 6.4%; fever 25%; vomiting 9% and constipation 16%.

Conclusion: Clinical examination data in human shown variety of presentation among symptoms and signs.

Keywords: Giardia; Diarrhea; Steatorrhea; Giardiasis; waterborne

Introduction

Antony Van Leeuwenhoek was the first one described Giardia in his own stool in 1681 [1]. The World Health Organization added Giardia to the list of parasitic diseases in 1981 [2,3]. *G. lamblia* was a worldwide health problem that requires better solutions, it was the causative agent of giardiasis, a disease which affected over 200 million people per year in 2002 [4-6]. Giardiasis was the most frequently diagnosed water borne disease and the major public health concern of water utilities in the developed and developing nations, water is an important vehicle for the transmission of Giardia to human and livestock [7]. The life cycle of *G. duodenalis* comprises two main stages: a trophozoite stage which colonizes the intestinal epithelium of the host and causes disease, and an infectious cyst stage which is resistant in the environment [8]. *G. duodenalis* trophozoites have been reported to rarely invade the mucosa of the duodenum and jejunum, but normally they are considered non-invasive and attach to the microvillous and baso-lateral membranes of the enterocyte [9-11]. In addition, the normal villus structure is affected in some patients like in villus blunting (atrophy) and crypt cell hypertrophy and increases of crypt depth [12]. The colonization of trophozoites in the small intestine results in a reduction in the height of the microvilli and therefore a loss of absorptive surface area [9]. This loss of absorptive surface leads to mal-absorption of glucose, electrolytes and water, with reduces disaccharidase activity [13]. This results in the small intestine filling with mucous and fluid, and ultimately mal-digestion and hypermotility, all responsible for the clinical manifestation of diarrhea [14]. As a result, enterocytic brush border is damaged, the increased

epithelial permeability leads to an inflammatory response, digestive and absorptive changes, that correlate with the brush border injury and disaccharidase deficiencies [15]. It also triggers apoptosis causing loss of epithelium barrier function with a subsequent increase in permeability [16]. It is established that the increased intestinal permeability could also result from increased luminal antigens, this could provoke the appearance of allergic reactions—a complication, often observed in humans infected with Giardia [15,17,18]. There are three types of giardiasis the asymptomatic, acute, and chronic infections [19]. Asymptomatic infection occurs in human adult and children, and other mammalian hosts and is probably the most common form of giardiasis [19,20]. Factors such as variation in parasite virulence and host immune defense may contribute to asymptomatic giardiasis [21]. The clinical manifestations of human giardiasis are individual and depend on various factors such as the route of infection, the duration of infection and the physiological condition of the host and probably, parasitic factors [12]. The acute stage generally begins with intestinal troubles, colic, followed by nausea and anorexia, early signs could be low-degree fever and lethargy. Later symptoms include profuse, watery, foul-smelling diarrhea, meteorism and enhanced peristalsis with extensive flatulence, eructation and bad taste, epigastric cramping. Rarely, feces could contain mucus or blood [22]. Although some acute episodes of giardiasis could resolve spontaneously, they usually pass into a sub-acute or chronic stage [23]. During the chronic stage, lethargy, headache and muscle pain with progressive weight loss, loss of appetite and mal-absorption could be present [24]. Chronic disease is often dominated by symptoms of mal-absorption [22].

Materials and Methods

About 1344 stool samples were collected from human cases for different age, sex, address, economic state and service level, during period from 10 September 2012 to 30 May 2013.

Questionnaires sheet

The patient's name:
 Date:
 Sample:
 Number:
 Age:
 Gender: Male/Female
 Address:
 The living situation of the individual: Poor/Medium/Good
 The level of services: Poor/Medium/Good
 Symptoms: Yes/No
 Mention:
 Infection:
 The first number frequent case:
 Have animal? Yes type of animal: (cows, sheep, dogs..... etc.)
 Type of drinking water and washing: River/water/liquefaction/RO

All samples were taken from patients complain of diarrhea, abdominal discomfort, nausea and abdominal cramp whose attended to Al-Qurnah General Hospital, Al-Qurnah sector centers for Primary Health Care , Al-Medina General Hospital and Al-Medina sector centers for Primary Health Care and all samples collected in sterilized cups and taken up to laboratory of above centers, then the results transported to laboratory of Parasitology / Collage of Veterinary

Medicine - University of Basrah for confirmed diagnosis. We used a Questionnaires sheet by which take a careful history from each case. The sheet consists of detailed location, level of services, social, clinical, environmental and source of water supply about every patient examined.

Direct smear with normal saline

Comminute 5-6 stool or faecal balls or 2-4 gm with pestle and mortar.

Transfer a loop-fall of the material to a slide matchstick, and a drop of diluted fluid (normal saline) were placed on glass microscopic slide to form a uniform suspension.

Spread it on the slide and apply cover slip.

Examine the slide microscopically under low power (10X) and high power (40X) according [25,26].

Direct smear with Lugol's iodine (Himedia/India)

After prepare the direct smear, used Lugol's iodine to kill and staining the trophozoites and cysts. Adding of one drop of Lugol's iodine to prepared slide and examination by light microscope under, low (10X) and high power (40X) according [25].

Results

405 cases of human giardiasis were diagnosed out from 1344 patients. Symptoms and signs varied from diarrhea, Foul smelling greasy stool (steatorrhea), abdominal crump, bloating, fever, anorexia, constipation and vomiting, there number of cases examined list in Table 1 below.

Sings	No of cases examined	Percentage
Diarrhea	304	75
Foul smelling greasy stool (steatorrhea)	129	31.8
Abdominal crump	345	85
Bloating	89	21.9
anorexia	26	6.4
Fever	101	24.9
Constipation	64	15.8
Vomiting	39	9.6

Table 1: Clinical features of giardiasis among 405 human cases with percentage

Discussion

In this work recorded symptomatic human giardiasis cases, this as a resulting from examined all human cases were obtained from peoples suffering from different clinical features attended to hospitals and health centers for treatment. These were agreeing with most studies like the infection with *G. lamblia* ranges from asymptomatic passage of cysts, to acute diarrhea, to a syndrome of chronic diarrhea and mal-absorption [27]. There are two types of giardiasis: the asymptomatic and symptomatic infections [19].

The clinical picture of giardiasis varies ranging from asymptomatic infection or acute self-limiting to severe, chronic one [28]. Many factors such as variation in parasite virulence and host immune defense may contribute to asymptomatic giardiasis [21]. The parasite adaptations promoting cyst survival in the external environment and trophozoite infectiveness and persistence in the mammalian small intestine each contribute to being key virulence properties for this parasite to cause symptomatic disease [29].

In this work eight common symptoms recorded in human were diarrhea 75%; foul smelling greasy stool 31.8%; bloating 22%; abdominal cramp 85%; anorexia 6.4%; fever 25%; vomiting 9% and constipation 16%. These were agreeing with the most important clinical symptoms are diarrhea and mal-absorption [2]. Other mention that intestinal troubles followed by nausea and anorexia, other signs could be low-degree fever, flatulence and abdominal distension with cramps occurred in acute stage, later symptoms include profuse, watery and foul-smelling diarrhea [22]. During the chronic stage lethargy, headache and muscle pain with progressive weight loss, loss of appetite and mal-absorption could be present [22]. Impaired fat absorption and steatorrhea are common in symptomatic giardiasis, stools are usually greasy and malodorous and float in water [20]. Typically, the symptoms accompanying infection include diarrhea, flatulence, upper intestinal cramps, abdominal distension, nausea, weight loss and mal-absorption [30].

Host factors such as immune status, nutritional status and age, as well as differences in virulence and pathogenicity of *G. duodenalis* isolates are recognized as important determinants for the severity of infection [9,12]. The colonization of trophozoites in the small intestine results in a reduction in the height of the microvilli and therefore a loss of absorptive surface area [9]. This loss of absorptive surface leads to mal-absorption of glucose, electrolytes and water, and reduces disaccharidases activity [13]. This results in the small intestine filling with mucous and fluid, and ultimately mal-digestion and hypermotility, all responsible for the clinical manifestation of diarrhea [14]. As a result, enterocytic brush border is damaged, the increased epithelial permeability leads to an inflammatory response, digestive and absorptive changes, that correlate with the brush border injury and disaccharidases deficiencies [15].

References

1. Roberts LS, Janovy, J (2009) Foundations of Parasitology. (8th edn). New York, USA. McGraw-Hill Inc. p:43-94.
2. Faubert G (2000) Immune response to *Giardia duodenalis*. Clin Microbiol Rev 13: 35-54, table of contents.
3. Artzer, MA (2009) A comparison of the SNAP *Giardia* fecal antigen test and the zinc sulfate double centrifugation fecal flotation procedure to diagnose *G. intestinalis* infections in two populations of infected dogs. M.Sc. Thesis. Kansas state University.
4. Lalle M, Pozio E, Capelli G, Bruschi F, Crotti D, et al. (2005) Genetic heterogeneity at the beta-giardin locus among human and animal isolates of *Giardia duodenalis* and identification of potentially zoonotic subgenotypes. Inter J Parasitol 35: 207-213.
5. Craun GF (1990) Water born Giardiasis. In Giardiasis: Meyer EA (Ed). (1st edn). Elsevier science publisher. Netherland. p: 192-211.
6. Lane S, Lloyd D (2002) Current trends in research into the waterborne parasite *Giardia*. Crit Rev Microbiol 28: 123-147.
7. Bryce J, Boschi-Pinto C, Shibuya K, Black RE; WHO Child Health Epidemiology Reference Group (2005) WHO estimates of the causes of death in children. Lancet 365: 1147-1152.
8. Hoque ME, Hope VT, Kjellström T, Scragg R, Lay-Yee R (2002) Risk of giardiasis in Aucklanders: a case-control study. Int J Infect Dis 6: 191-197.
9. Lujan HD, Svard, S (2011) *Giardia* a Model Organism. (1st edn). Austria. Springer-Verlag/Wien. p: 3-359.
10. Buret A, Hardin JA, Olson ME, Gall DG (1992) Pathophysiology of small intestinal malabsorption in gerbils infected with *Giardia lamblia*. Gastroenterology 103: 506-513.
11. Wolfe MS (1992) Giardiasis. Clin Microbiol Rev 5: 93-100.
12. Farthing MJ (1993) Diarrhoeal disease: current concepts and future challenges. Pathogenesis of giardiasis. Trans R Soc Trop Med Hyg 87 Suppl 3: 17-21.
13. Thompson RC, Reynoldson JA, Mendis AH (1993) *Giardia* and giardiasis. Adv Parasitol 32: 71-160.
14. Olson, ME and Buret, AG (2001) *Giardia* and Giardiasis. In: Samuel WM, Pybus MJ and Kocan AA, (Ed.). Parasitic Diseases of Wild Animals. (1st edn). Iowa State University Press. USA. p: 399-416.
15. Deselliers LP, Tan DT, Scott RB, Olson ME (1997) Effects of *Giardia lamblia* infection on gastrointestinal transit and contractility in Mongolian gerbils. Dig Dis Sci 42: 2411-2419.
16. Chin AC, Teoh DA, Scott KG, Meddings JB, Macnaughton WK, et al. (2002) Strain-dependent induction of enterocyte apoptosis by *Giardia lamblia* disrupts epithelial barrier function in a caspase-3-dependent manner. Infect Immun 70: 3673-3680.
17. Scott KG, Meddings JB, Kirk DR, Lees-Miller SP, Buret AG (2002) Intestinal infection with *Giardia* spp. reduces epithelial barrier function in a myosin light chain kinase-dependent fashion. Gastroenterology 123: 1179-1190.
18. Chakarova B (2004) Clinic, treatment and dispensary control of patients with lambliosis. J Bulg Medic 12: 11-12.
19. Chakarova B, Miteva I, Stanilova S (2009) Giardiasis in humans – clinical and diagnostic study. In: Proceedings of the Eighth National Conference of Parasitology. (1st edn). Varna, Bulgaria. p: 52-53.
20. Farthing MJG (1994) Giardiasis as a disease. In: *Giardia: From Molecules to Disease*. RCA Thompson, JA Reynoldson, and AI Lymbery (Eds.). (1st edn). CAB International, Wallingford, UK. p: 15-37.
21. Fauci, AS, Braunwald, E, Kasper, DL, Hauser, SL, Longo, DL and Jameson, JL (2008) HARRISON's a Principles of Internal Medicine: Giardiasis. (17th edn). The McGraw-Hill Companies, Inc. USA. p:1312.
22. Farthing MJ (1990) Immunopathology of giardiasis. Springer Semin Immunopathol 12: 269-282.
23. Ivanov AI (2010) *Giardia* and giardiasis. Bulg J Vet Med 13: 65-80.
24. Lalova, IT (1977) Investigation upon the epidemiology and diagnostic of lambliosis Dissertation. Bulg J Vet Med 13:65-80.
25. Colledge NR, Walker BR, Ralston, SH (2010) Davidson's Principles and Practice of Medicine. (21st edn). Elsevier, Churchill Livingstone, China. p: 363.
26. Markell EK, Jone DT, Krotosk WA (1999) Markell and Voges Medical Parasitology. (8th edn), W.B. Saunders co. Philadelphia. p: 55-445.
27. Al-Emarah GYA, AL-Ali SJK, AL-Idresi SRA (2009) Atlas of Parasites. (1st edn), Al-Salam printing, Basrah, Iraq. Part1 p: 7-10.
28. Hill DR (1993) Giardiasis. Issues in diagnosis and management. Infect Dis Clin North Am 7: 503-525.
29. Read C, Walters J, Robertson ID, Thompson RC (2002) Correlation between genotype of *Giardia duodenalis* and diarrhoea. Int J Parasitol 32: 229-231.
30. Aley SB, Gillin FD (1995) Specialized surface adaptations of *Giardia lamblia*. Infect Agents Dis 4: 161-166.