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ZOONOTIC GIARDIASIS AND ITS COMPLICATIONS: A REVIEW ARTICLE

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Abstract

Giardiasis is a protozoan flagellated zoonotic parasite, causing diarrheal disease caused by *G. duodenalis* (or "Giardia" for short). Once a person or animal has been infected with *Giardia*, the parasite lives in the intestines and is passed in stool (poop). It causes mild or severe diarrhea, gas, stomach cramps, nausea (a feeling of stomach upset), or dehydration (body water loss causes weakness of dizziness). Some people experience no symptoms at all. Cysts once pass are infective, spreads easily and can spread from person to person or through contaminated water, food, surfaces, or objects. Giardiasis shares some pathogens some or less the clinical manifestations.

Keywords: Giardiasis, Epidemiology, Pathogenesis, Diagnosis, Differential diagnosis, Treatment

Introduction

Giardia duodenalis (= *G. lamblia* or *G. intestinalis*) is a zoonotic protozoan parasite capable of causing sporadic or epidemic diarrheal illness. Giardiasis is an important risky cause of water-borne and foodborne disease, daycare center outbreaks, and illness in travelers (Feng an, Xiao, 2011).

Review and Discussion

Epidemiology: *Giardia duodenalis* is the main species infecting various mammals, including domestic animals and man globally (Jian *et al,* 2021). Risk groups are infants, children, international adoptees, travelers, immunocompromised individuals, and cystic fibrosis patients (Takaoka *et al*, 2016). But, in 147 pediatric patients with acute non-dysenteric diarrhea in the United States, giardiasis was the cause in 15% of cases, seconded by rotavirus (Schlagenhauf *et al,* 2015).

Giardiasis is especially common in areas with poor sanitary conditions and limited water-treated facilities with prevalence more than 40% (Boggild *et al,* 2014). Giardiasis *duodenalis* identified in stools were asymptomatic, and detected more common in asymptomatic stools than in acute diarrhea patients (Muhsen and Levine, 2012). Giardiasis is a known cause of enteric disease in abroad travelers in the United States, Canada, and Europe (Harvey *et al,* 2013), up to15.223 cases in the United States (CDC, 2015). Caeiro *et al.* (1999) in Canada noted an adjusted incidence rate of 25.8 cases per 100,000 populations between 1990 & 1998, of which 40% of cases were in travelers; other important infection source was person-to-person transmission (Greig *et al,* 2001). Also, giardiasis was common in the Eastern Mediterranean Countries and North Africa (Hijjawi *et al,* 2022), and especially children in Egypt (Saleh *et al,* 2023), Jordan (Nimri, 1994), Lebanon (Arslanian, 1960), Saudi Arabia (Awadallah and Morsy, 1974), Syria (Almerie *et al,* 2008) and others.

Life cycle: *Giardia* has two morphological forms: cysts and trophozoites. Cysts are the infective form; they are excreted in stool and can survive in moisted soil for months especially in cold water (Fink *et al,* 2020). After cyst ingestion, excystation occurs in the proximal small bowel with release of trophozoites, which are pear-shaped, binucleate, multi-flagellated forms, and division by binary fission; they localize principally to the proximal small bowel (Pierce and Huston, 2009). An adhesive disk on the trophozoite ventral surface facilitates its attachment to mucosal

surface of the duodenum and jejunum, although it doesn't invade the mucosal epithelium. Trophozoites that do not adhere to the small bowel move forward to the large intestine, where they revert to the infectious cyst form; conjugated bile salts appear to foster encystation. Cysts pass into environment in excreted stool; in setting of diarrhea, trophozoites can also be found in stool. The incubation is a week or more before acute giardiasis symptoms development (CDC, 2017).

Routes of transmission: Infective *Giardia* cysts to man may occur via three routes: waterborne, foodborne, or fecal-oral transmission (Fakhri *et al,* 2021).

Water is the giardiasis major transmission source. *Giardia* cysts survive readily in mountain streams, as they are hardy in cold water. Water-dwelling mammals, such as beavers, can become infected and may serve as as sources of water contamination (Dykes *et al,* 1980). So, it is an important cause of diarrheal illness among hikers in wilderness areas who drink water without adequately filtered, treated, or boiled (McClung *et al,* 2018). Deep well water, in contrast with surface well water, is usually safe as water filtration through soil removes cysts, which are resistant to chlorination and so, bacterial coliform counts were not a reliable measure of *Giardia* contaminated in chlorinated water (Welch, 2000). Ma *et al.* (2019) in China reported that bacteria indicator for *Escherichia coli* and coliforms to test water may not be adequate for pathogens, its absence didn't mean absence of *Giardia* or *Cryptosporidum*.

Foodborne transmission of giardiasis can occur via ingestion of raw or undercooked food raw vegetables, salad bars, and fresh fruit) contaminated with cysts and food contaminated after cooking (Adam *et al,* 2016).

Person-to-person transmission can occur in settings in which there is fecal incontinence and poor hygiene, such as childcare centers (Overturf, 1994). Risk of acquisition or transmission was greatest for young children who didn't yet toilet trained; who can also serve as a source for secondary cases within households (Waldram *et al,* 2017). Also, giardiasis can be transmitted via heterosexual or homosexual anal-oral sexual contact (Escobedo *et al,* 2014).

Pathogenesis: *Giardia* infections are common in pigs, cattle, sheep, goats, elks and deer, other ruminants, and dogs and cats as well as man (Cacciò *et al,* 2018). Six known *Giardia* species are restricted to nonhuman hosts and are not recognized as causes of human disease. *Giardia duodenalis* consists of eight genetic groups (or assemblages); two assemblages are found in both humans and animals (A & B), and the remaining six are specific to non-human hosts, including canines, felines, rodents, and seals as assemblages (C to H). The genotypes varied even within assemblages A & B, and it was likely that only some genotypic variants have potential to cause infection in humans. Based on this heterogeneity, the animals' role in the human epidemiology remains poorly understood (Krumrie *et al,* 2022). Beavers have been clearly implicated in transmission of waterborne infection to humans, but the domestic dogs and cats roles as sources of human infection remain to be ascertained (Bouzid *et al,* 2015)

The relative roles of *Giardia* assemblages and the human host responses that contribute to asymptomatic and symptomatic infections are not fully understood. Experimental ingestion of 10 to 25 cysts was associated with symptomatic giardiasis in one study; some infections resolved by about three weeks, while others persisted with mild symptoms (Rendtorff, 1954) No volunteers challenged with assemblage A *Giardia* became infected, whereas all challenged with assemblage B strain became infected, although only half of these individuals were symptomatic (Nash, 2013). Assemblage B isolates occurred more frequently in patients with chronic infection than assemblage A isolates (Franzén *et al,* 2009). Assemblage A was associated with symptomatic infection, and genotype B was associated with asymptomatic infect-

ion (Sahagún *et al,* 2008), which was particularly evident in children <5 years, suggested that host factors also influence the nature of clinical manifestations.

The pathogenesis of giardiasis symptoms can occur in (acute diarrhea and longstanding malabsorption) is not fully understood. The small intestine is the site of the major structural and functional abnormalities associated with giardiasis (Buret, 2007). Cotton *et al.* (2011) in Canada reported that trophozoites attached to the epithelium of the small intestine upper part induced a series of events, as disruption of epithelial barrier function, diffuse shortening of brush border microvilli, small intestinal mal-absorption and mal-digestion, chloride hyper-secretion, and increased the rates of small intestinal transit, culminating with diarrhea. Allain and Buret (2020) in Canada reported that giardiasis the important cause of diarrhea, resulted in post-infectious and extra-intestinal complications by the pathophysiological responses triggered recapitulating by the effects of its membrane-bound and the secreted cysteine proteases.

The light microscopy may demonstrate no abnormalities, mild or moderate partial villous atrophy, or subtotal villous atrophy in severe cases. An increase in crypt depth may be seen, and microvilli shortening or disruption may occur. Intestinal epithelial tight junctions may be disrupted leading to increased permeability and altered epithelial cell survival. Thus, deficiencies in small bowel epithelial brush border enzymes, including disaccharidases such as lactase, may develop (Solaymani-Mohammadi, 2022). Such local epithelial enzyme deficiencies likely contribute to symptoms of acute and chronic giardiasis and are slow to recover even with effective treatment (Gardner and Hill, 2001).

Immunity: Chronic exposure to *G. duodenalis* may induce partial immunity; in endemic areas, children <10 years have higher rates of giardiasis than older individuals (Giman *et al,* 1985). Besides, travelers to endemic areas have higher rates of symptomatic disease than long-term residents (Istre *et al,* 1984). Buret (2005) in Canada reported that the T lymphocyte-mediated pathogenesis is common to a variety of enteropathies, including giardiasis, cryptosporidiosis, bacterial enteritis, celiac's disease, food anaphylaxis, and Crohn's disease. In giardiasis as well as in the other disorders, a diffuse loss of microvillous brush border, combined or not with villus atrophy, is responsible for disaccharidase insufficiencies and malabsorption of electrolytes, nutrients, and water, which ultimately cause diarrheal symptoms. In endemic regions, however, reinfections can be frequent, so any acquired immunity is limited. Humoral immunity is important for host defense against giardiasis. Secretory IgA antibodies are an important response to infection, since trophozoites are localized to intestinal lumen (Stark *et al,* 2009). Patients with cystic fibrosis or immunoglobulin deficiencies (such as common variable immunodeficiency or X-linked agammaglobulinemia) tend to have more severe disease, may be due to deficiencies in secretory IgA and cell-mediated immunity (Oksenhendler *et al,* 2008).

Patients with HIV infection have impaired immune response to parasite but don't develop more severe disease; giardiasis is not a major cause of enteritis in HIV-infected patients (Smith *et al,* 198). Asymptomatic infections occur with the HIV, but with progressive immunosuppression, symptomatic infection risk increases (Nash *et al,* 2001). Giardiasis can cause diarrhea in both HIV-positive and HIV-negative patients (Yancheva *et al,* 2016).

Clinical manifestations: Giardiasis severity of clinical manifestations is variable. In general, about half of exposed individuals clear the infection in the absence of clinical symptoms, approximately 15% of individuals shed cysts asymptomatically, and the remaining 35 to 45% of individuals with symptomatic infection (Nash *et al*, 1987). Nature of clinical manifestations in an individual depends on a number of factors including

the virulence of the isolate, parasite load, and the host immune response (Pickering *et al*, 1984).

Asymptomatic infection: Asymptomatic infection occurs in both children and adults, and asymptomatic cyst shedding can last six months or more (Donowitz *et al,* 2016). In resource-limited settings, most children were encountered *Giardia* by age two years without it being associated with diarrhea, but infected children might have impaired growth (Kotloff *et al,* 2013).

Acute giardiasis: Symptoms include (Hill and Nash, 2011): Diarrhea- 90%, malaise- 86%, foul-smelling and fatty stools (steatorrhea)- 75%, abdominal cramps and bloating- 71%, flatulence- 75%, nausea- 69%, weight loss- 66%, vomiting- 23%, fever- 15%, constipation- 13%, and urticarial- 10%. Onset of acute gastrointestinal symptoms within one week of exposure is not likely attributable to infection with *Giardia*. Symptoms may last two to four weeks.

Chronic giardiasis: Chronic giardiasis may follow the acute phase of illness or may develop in the absence of an antecedent acute illness. Chronic symptoms can develop in up to half of symptomatic individuals (Cantey et *al,* 2011). In experimentally infected individuals, 84% had a self-limited illness (mean duration 18 days); the remainder became chronically infected.

Chronic giardiasis symptoms may include: Loose stools but usually not diarrhea, steatorrhea, profound weight loss (10 to 20% of body weight), malabsorption, malaise, fatigue, depression, abdominal cramping, borborygmi, flatulence, and burping. These manifestations may wax and wane over several months (Singh *et al,* 2000). Malabsorption may be responsible for significant weight loss that can occur in giardiasis. Even in cases of otherwise asymptomatic infection, malabsorption of fats, sugars, carbohydrates, and vitamins may occur. This can lead to hypoalbuminemia and deficiencies of vitamin A, B12, and folate (Rana *et al,* 2005).

Acquired lactose intolerance occurs in up to 40% of patients; clinically manifests with exacerbation in intestinal symptoms following ingestion of dairy products. Recovery can take many weeks, even after clearance of the parasite (Vega-Franco *et al,* 1987)

Complications: In a small number of patients, persistent infection was associated with malabsorption development and weight loss (Lengerich *et al,* 1994). Chronic giardiasis as other diseases associated with malabsorption, such as inflammatory bowel disease, and travelers acquired giardiasis suffered persisting diarrhea (Gunasekaran and Hassall, 1992), and in some patients persisted after initial treatment (Mørch *et al,* 2008). Prado *et al.* (2005) reported that growth and decreased growth was impeded among giardiasis children with or without symptoms. Also, giardiasis was a strong predictor of stunted growth in Colombian children (Botero-Garcés *et al,* 2009). Duplessis *et al.* (2017) reported that chronic giardiasis affected children growth and development. This is due to impacting such biological processes as iron absorption, retinal morphology, and hepatic and pancreatic functionality (Lehto *et al,* 2019).

Hypersensitivity, as rash, urticaria, aphthous ulceration, and reactive arthritis or synovitis, although rare, was reported in giardiasis setting (Halliez and Buret, 2013). Hanevik *et al.* (2014) in Norway, after a waterborne giardiasis epidemic a cohort study of >800 individuals exposed to *Giardia* showed irritable bowel syndrome (39%) and chronic fatigue (31%) were significantly increased six years after exposure (relative to unexposed controls) and the symptoms frequency declined with time after the initial exposure

Giardiasis and *Helobacter pylori*: Moreira *et al.* (2005) in Brazil reported that *G. intestinalis* and *H. pylori* infect humans' gastroint estinal tracts early life, *H. pylori* caused pathologies and hypochlorhydria in stomach facilitating *Giardia* colonization in the gastric mucosa, and significantly influence the patient immune response.

Abou Holw *et al.* (2009) in Alexandria reported significant upper gastrointestinal sy-

mptoms (epigastric pain and anorexia) in the giardiasis patients with *H. pylori*, with significant gastric lesions by endoscopic and histopathologic examination in such patients as compared to those only with G. *lamblia* infection. Isaeva and Efimova (2010) in Russia reported strict association between the *H. pylori* and *G. lamblia* in the stomach--100% of *H. pylori*-infection combined with giardiasis. Ankarklev *et al.* (2012) in Uganda reported that *Giardia* assemblage B dominated in children in Kampala, and that the *H. pylori* presence was an associated risk factor for *G. intestinalis* infection. Fouad *et al.* (2014) in Egypt reported that *Heliobacter pylori*, *G. intestinalis* and coeliac disease were the common causes of dyspepsia, and *G. intestinalis* genotype A, which highly associated with dyspeptic symptoms. Gerbaba *et al.* (2015) in Canada reported that *Giardia* induced functional changes in commensal bacteria, making them to be opportunistic pathogens, and alter host-microbe homeostatic interactions assessing the human microbiota toxicity. El-Badry *et al.* (2017) in Egypt with variables *Giardia* assemblage type, sex, or harboring multi-parasites, reported that only school aged children, markedly associated with giardiasis and *H. pylori*. Painter *et al.* (2017) reported that *Giardia* can spread from duodenum to biliary and pancreatic ducts, causing cholecystitis, cholangitis, or granulomatous hepatitis, impaired exocrine pancreatic function with diminished trypsin and lipase secretion. Nakao *et al.* (2017) in USA noted that individuals with giardiasis were more likely to have irritable bowel syndrome.

Abd Elbagi *et al.* (2019) in Sudan reported that among 100 *H. pylori* patients that 23% had *H. pylori* and 10% of healthy individuals had gastrointestinal parasites; *Entamoeba histolytica* was detected in 12% of *H. pylori* cases followed by *Entamoeba coli* (7%) and *Giardia lamblia* (4%). The control ones (100), *E. histolytica* was detected in 5% followed by *G. lamblia* in 3% and *E. coli* in 2% of individuals. They concluded that the intestinal protozoa parasites were more common in the *H. pylori* infected patients.

Again, Hurník *et al.* (2019) in Czech Republic in an old-man found *Giardia* co-infected with pancreatic cancer. Tilahun *et al.* (2022) in Ethiopia reported that *G. lamblia* and *H. pylori* together, have a synergistic effect on man causing serious damage. They added that nevertheless, previously infected persons may report symptoms even years after effective treatment. Chen *et al.* (2022) in China reported that *G. lamblia* is the risk factors among colorectal cancer patients.

Diagnosis: CDC (2021) declared many tests to detect *Giardia* and some are more sensitive and specific than others. Microscopy with direct fluorescent antibody test is considered the test of choice for giardiasis diagnosis since it increased sensitivity over non-fluorescent microscopy techniques. Other alternate methods include: 1- Rapid immunochromatographic cartridge assays, 2- Enzyme immunoassay (EIA) kits, 3- Microscopy with trichrome staining, & 4-Molecular assays (Only DNA sequencing, for example) can be used to identify the *Giardia* strains, retesting for *Giardia* is only re-commended if symptoms persist after treatment. However, Oberhuber *et al.* (2016) in Austria reported that *G. lamblia* was only proven histological examination of mucosal biopsy specimens taken from terminal ileum. Groudan *et al.* (2021) in USA reported a patient diagnosed incidentally with *Giardia* from a duodenal biopsy specimen obtained during a gastrointestinal bleed. Pessarelli *et al.* (2022) in Italy reported intestinal pseudo-obstruction by *G. lamblia* in a woman with malaise, nausea, reduced appetite, abdominal distention, loose stools and weight loss, in whom diagnosis was basis of dilated small bowel loops with air-fluid levels and in absence of any mechanical obstruction assessed by cross-sectional imaging and endoscopy.

Antigen detection assays: A number of immunoassays using antibodies against cyst or trophozoite antigens have been developed for stool analysis. Available kits include direct immunofluorescent assays (DFA) using

fluorescein-tagged monoclonal antibodies, immunochromatographic assays, and ELISA assays (Garcia and Shimizu, 1997).

Generally, these methods have greater sensitivity and faster turn-around time than conventional stool microscopy methods. Specificity and cost are usually relatively comparable. Some studies showed DFA to have the highest sensitivity (Al *et al,* 2006). Most of the commercially available assays can detect both *Giardia* and *Cryptosporidium* simultaneously. Of the 325 stool samples demonstrated that an ELISA against a specific *Giardia* antigen (antigen 65) detected 30% *Giardia* cases more than the stool microscopy (Rosoff *et al,* 1989). Stool samples from patients with abdominal symptoms by using different assays for detection of *Giardia*, the sensitivities obtained by Ridascreen *Giardia*, Rida Quick *Giardia*, Rida Quick Combi, and *Giardia*-Strip were 82, 80, 80, & 44%, respectively & all specificity was ≥98% (Weitzel *et al,* 2006). Also, Ridascreen *Giardia* ELISA was 100% sensitive and 91.5% specific, including children (Jahan *et al,* 2014). Comparing stool microscopy, DFA, and three *Giardia* immunodiagnostic techniques agreed with these methods in 76% of cases, but immunologic methods detected more positive cases than stool microscopy by 12% (Aziz *et al,* 2001)

Immunoassays are of limited use following treatment of infection. Loss of detectable stool antigens is suggestive of effective treatment, but continued stool antigen shedding could reflect shedding of killed parasites (Vasoo and Pritt, 2013).

Nucleic acid amplification assays: Nucleic acid amplification assays (NAAT) were developed to detect *Giardia* in stool samples, some remain research tools, following were commercially available (Boadi *et al,* 2014): 1- The Bio-Fire Film-Array gastrointestinal panel can detect 22 bacterial, viral, & parasitic (including *G. duodenalis*) causes of infectious diarrhea (Buss *et al,* 2015). 2- The Luminex xTAG Gastrointestinal Pathogen Panel can detect various viral, bacterial, and protozoan (including *G. duodenalis*) intestinal pathogens (Claas *et al,* 2013). 3- The BD MAX Enteric Parasite Panel detected *G. duodenalis, Cryptosporidium parvis*/*hominis,* and *Entameba histolytica* (Perry *et al,* 2017). The NAATs were of limited use for post-treatment, Van den Bijllaardt *et al.* (2014) in the Netherlands reported that *Giardia* DNA is rapidly cleared after successful treatment, but uncertainties remain regarding clearance of *Giardia a*DNA following treatment and whether residual detection may show killed or viable parasites (Mengelle *et al,* 2013). The NAAT-based tools were used to detect *Giardia* and other zoonotic pathogens in water supplies (Dreelin *et al,* 2014).

Stool microscopy: Stool microscopy to detect *Giardia* can be specific and may also be useful for detecting other potential parasitic causes of gastrointestinal symptoms (El Shazly *et al,* 2007). Limitations include intermittent excretion of *Giardia* cysts (necessitateng up to 3 stool examinations), cumbersome processing procedures, and technician expertise. Infectious Disease Society of America diagnostic guidelines recommend taking stool to diagnose *Giardia*; when stool examinations are negative but suspicion remains high, duodenal aspirate microscopy is the only alternative diagnostic strategy suggested.

Laboratory processing of stool samples consists of a saline suspension to look for trophozoites and cysts and a polyvinyl alcohol and/or formalin preparation for staining. Loose, watery stool is more likely to be positive for trophozoites; a semiformed or form ed stool would likely contain cysts only.

Other tests: In general, giardiasis patients don't have peripheral leukocytosis or eosinophilia. White cells in stool specimens are usually absent. Fecal fat excretion and other laboratory tests of malabsorption may be abnormal. Upper gastrointestinal series are usually normal but may demonstrate mucosal edema in some cases.

Differential diagnosis: Differential diagnosis of giardiasis includes: 1- Travelers' diarrhea: Travelers' diarrhea can be caused by a

range of pathogens, including enterotoxigenic *Escherichia coli* and *Campylobacter* spp; it consists of malaise, anorexia, and abdominal cramps followed by watery diarrhea in travel setting to developing setting (Casburn-Jones and Farthing, 2004). Diagnosis is usually based on clinical history; the illness is generally self-limited. Travelers' diarrhea onset is usually within days, whereas symptomatic giardiasis develops only after a week or more after infection. Evaluation for *Giardia* is warranted in the setting of delayed onset (at least a week after exposure) of upper gastrointestinal manifestations (such as bloating, gas, or nausea) and in the setting of persistent symptoms.

2- Cryptosporidiosis: *Cryptosporidium* is similar to *Giardia* in that it can cause a diarrheal illness with associated malaise, nausea and anorexia, crampy abdominal pain, and low-grade fever associated with a secretory diarrhea and with malabsorption. Infection can be asymptomatic, a mild diarrheal illness, or severe enteritis with or without biliary tract involvement. In immunocompetent hosts, illness usually spontaneously resolves without therapy, but among immunosuppressed host infection can be a chronic debilitating illness with wasting and persistent diarrhea. Diagnosis depends on enzyme immunoassay or by microscopic identification of oocysts in stool or tissue. Organisms may be present in duodenal aspirates, bile secretions, biopsy specimens from gastrointestinal tract, or respiratory secretions (El-Bahnasawy *et al,* 2018).

3- Strongyloidiasis: *Strongyloides stercoralis* infection can range from asymptomatic eosinophilia in immunocompetent host to disseminated disease with septic shock in immunocompromised host, being endemic in tropical and subtropical regions and occurs sporadically in temperate areas. Adult burden in infected man can increase substantially via autoinfection. Among immunocompromised hosts, autoinfection leads to hyperinfection syndrome, with massive dissemination of filariform larvae to the lungs, liver, heart, CNS, and endocrine glands. Most infected patients don't have prominent symptoms. The commonest manifestations are mild waxing and waning gastrointestinal, cutaneous, or pulmonary symptoms persisting for years (cancer); others simply have eosinophilia in absence of symptoms (Zaky *et al,* 2019)

4- *Dientamoeba fragilis*: Its clinical manifestations are similar to those of giardiasis and include abdominal pain, flatulence, and diarrhea. In human stool specimens, it is almost always found solely as a trophozoite. However, the rare presence of putative cyst and precyst forms in clinical specimens has been reported; their transmission potential is being investigated. Other aspects of transmission and pathogenicity also are poorly under stood. It may be associated with eosinophilia; diagnosis is established by stool micrroscopy. Its pathogenicity and clinical importance continue to be investigated, including whether particular genotypes, subtypes, or strains of *D. fragilis* are associated with symptomatic infection in man. Both asymptomatic and symptomatic infection (e.g., with various nonspecific gastrointestinal symptoms) have been reported. The reported clinical manifestations have sometimes been described as similar to those of colitis, appendIcitis, or irritable bowel syndrome (CDC, 2019).

5- Lactose intolerance: Clinical symptoms of lactose intolerance include diarrhea, abdominal pain, and flatulence after ingestion of milk or milk-containing products. Diagnosis is established by a lactose tolerance test. Lactose intolerance is a condition with digestive symptoms, such as bloating, diarrhea, and gas after consumption foods or drinks contain lactose caused by lactose malabsorption. Lactose malabsorption is a condition in which small intestine cannot digest, or break down, all the lactose eaten or drink. Not everyone with lactose malabsorption has digestive troubles after lactose consumption, only that have symptoms are lactose intolerant.

Most people with lactose intolerance can consume some amount of lactose, but with-

out clinical symptoms. Different people can tolerate different amounts of lactose before having sy-mptoms (Storhaug *et al,* 2017).

6-Tropical sprue: Tropical sprue is a chronic diarrheal disease that occurs in the tropics and involves the small intestine, characterized by nutrient malabsorption. Several recent studies from India showed TS to be the commonest cause of sporadic MAS in Indian adults. Tropical sprue (TS) is diagnosed in patients presenting with suggestive clinical presentation, which cannot be explained by another cause of MAS and investing ations showed malabsorption of two unrelated substances, abnormal small-intestinal mucosal histology, which responds to treatment with antibiotics such as tetracycline and folic acid. There is an overlap between TS and post-infectious irritable bowel syndrome, but with several advances in its epidemiology, pathogenesis, and diagnosis, hitherto an enigmatic condition (Ghoshal *et al,* 2014)

7- Crohn disease or ileitis is an inflammatory disease that may involve the entire gastrointestinal tract; most patients have small bowel involvement (usually the distal ileum); one-third have ileitis exclusively. It can cause lesions from mouth to anus and may result in extraintestinal complications, with increasing prevalence in adults and children. Genetic predispositions to Crohn's disease were identified, and specific environmental factors have been associated with its development. Common presenting symptoms include diarrhea, abdominal pain, rectal bleeding, fever, weight loss, and fatigue. Physical examination should identify unstable patients requiring immediate care, including an anorectal examination, and look for extraintestinal complications. Initial laboratory evaluation identifies inflammation and screens for alternative diagnoses. Measurement of fecal calprotectin has value to rule out disease in adults and children. Endoscopy and cross-sectional imaging are used to confirm the diagnosis and determine the extent of disease. Treatment decisions are guided by disease severity and risk of poor outcomes. Patients commonly receive corticosteroids to treat symptom flare-ups, those with high risk disease are given biologics, with or without immunomodulators, to induce and maintain remission. For children, enteral nutrition is an option for induction therapy. All Crohn's diseased patients must be counseled on smoking avoidance or cessation; they are also at higher risk of cancer, osteoporosis, anemia, nutritional deficiencies, depression, infection, and thrombotic events, with maximum prevention measure was essential in caring (Veauthier and Hornecker, 2018).

8- Irritable bowel syndrome may present with a wide array of symptoms including chronic abdominal pain, diarrhea, and/or constipation and bloating. It is chronic in nature and diagnosis is based on criteria Irritable bowel syndrome, as one of the commonest functional bowel disorders, with a substantial impact on patients' daily lives, as well as a big economic impact on society. It is characterized by abdominal pain; bloating and abdominal distention and altered bowel movements, with predominance diarrhea/constipation, or alternation of such signs that cannot be explained by a structural or biochemical abnormality, with unknown mechanism of pathogenesis and pathophysiology, affects 5%-10% of healthy persons at time, mostly with a relapsing-remitting course (Sebastián Domingo, 2022).

Treatment: Petri (2005) in USA reported that Imidazole is the first-line giardiasis drug treatment, as it requires only a single dose to cure infection in most individuals. Metronidazole is as effective, but it requires 5 to 7 days of 3times/day. Nitazoxanide® appears to be as effective as Tinidazole® or Metronidazole®, and it didn't have the bitter taste of nitroimidazoles. A good alternate for use during pregnancy is Paromomycin®. Infection cure of varies between 60% & 100% with one course of treatment. Less effective and/or less well-tolerated drugs include Albendazole®, Quinacrine®, & Furazolidone®, but these agents must be reserved for the giardiasis resisted treatment with the first-line

agents. Dyab *et al.* (2016) in Egypt reported that of *Zingiber officinale* (ginger), and *Curcuma longa* (curcumin) extracts may represent effective and natural therapeutic alternatives with low side effects and without drug resistance in *Giardia* treated patient. Chai *et al.* (2021) in Korea reported that albendazole proved effective in treating giardiasis, with safe with few side effects; but, when used for prolong time (>14-28 days) or even only one time, liver toxicity and other side effects occurred with emerging drug resistance.

CDC (2021) reported that patients treated for giardiasis may continue to experience illness symptoms or have giardiasis positive results. In these cases, before switching therapies doctors must consider the following: Dehydration due to diarrhea can be a particular risk among pregnant women and can be life-threatening for infants. For this reason, rehydration was indicated for these groups.

Metwally *et al.* (2022) in an Egyptian single-center study reported patients, *H. pylori* gave >90% resistance to metronidazole & amoxicillin; modest resistance to erythromycin, azithromycin, &clarithromycin; and low resistance to moxifloxacin, & levofloxacin (≤20%). Dual resistance was high for amoxicillin/clarithromycin and amoxicillin/metronidazole, which preferred quinolones rather than clarithromycin or metronidazole as first-line treatment of *H. pylori.* This agreed with the low eradication rate for these two regimens in Egyptian clinical trials (Diab *et al,* 2018). Also, Liou *et al.* (2013) reported that *H. pylori* may be associated with extra-intestinal diseases, including immune thrombocytopenic purpura, refractory iron deficiency anemia, and vitamin B12 deficiency. Hassan *et al.* (2023) in Egypt reported that combined pre-biotic and probiotic supplementation gave promising anti-*Giardia* activity and restored the intestinal structures and modulate IgA response, apart from giving synergistic effects when added to Nitazoxanide.

Prevention: Cysts are resistant to chlorination, and iodine is disinfectant, but takes up to 8 hours before the waterconsumption. Filters must be available. Ensure filters must meet the National Safety Foundation (NSF) Standard 53 or NSF Standard 58 ratings for cyst reduction. Boiling water for 10 minutes killed *Giardia* cysts (Adeyemo *et al*, 2019).

Conclusion

Giardia duodenalis is a zoonotic protozoan parasite that can cause epidemic or sporadic diarrheal illness, with cysts and trophozoites. Cysts are the infectious form of the parasite; following cyst ingestion, trophozoites are released in the proximal small intestine. Trophozoites don't adhere to the small intestine, but move to large intestine where they revert to the infectious cysts that passed back into the environment in excreted stool.

Giardiasis high-risk groups include infants, young children, international adoptees, travelers, immunocompromised individuals, and patients with cystic fibrosis, especially in areas with poor sanitary conditions and limited water-treatment facilities.

Transmission of *Giardia* cysts to man may occur by three routes: waterborne, foodborne, or fecal-oral transmission. It is an important cause of diarrheal illness among hikers in wilderness areas who drink water that was not been adequately filtered, treated, or boiled. Giardiasis can also be transmitted via ingestion of raw or undercooked food contaminated with cysts or via food that is contaminated after cooking. Person-to-person transmission can occur where there is fecal incontinence and poor hygiene, such as childcare centers.

Clinical severity associated with giardiasis is variable. About half of exposed individual's clear infection without symptoms, about 15% of individuals shed cysts asymptomatically, and others 35 to 45% have symptomatic infection.

Symptoms include diarrhea, malaise, abdominal cramps, and weight loss. Malabsorption causes significant weight loss in chronic giardiasis. Acquired lactose in tolerance occurs in up to 40% of patients.

Diagnosis is by antigen detection assays, nucleic acid detection, and stool examination. Sometimes, antigen or nucleic acid detection tests are preferred.

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Explanation of figures

Fig. 1: Duodenal biopsy showed G*iardia lamblia* trophozoites (100x, H & E stain).

Fig. 2: Duodenal biopsy showed positive *Heliobacter pylori* section.