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Case Report

An Overview on Human Enteric Parasites

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Abstract

Human enteric protozoan parasites, including Giardia sp., Entamoeba sp., and Cryptosporidium sp., are significant causes of gastrointestinal diseases transmitted through contaminated food or water. These parasites are endemic in many regions, particularly in developing countries with inadequate sanitation and limited access to clean water. Giardiasis, amoebiasis, and cryptosporidiosis remain major public health challenges due to their high prevalence and the substantial morbidity they cause. This article provides a detailed overview of these infections, including their etiology, epidemiology, pathophysiology, diagnosis, and treatment.

Keywords: Enteric Parasites; Protozoan Parasites; Gastrointestinal Disease; Giardiasis; Amoebiasis; Cryptosporidiosis

Giardiasis

Giardia lamblia (also known as G. intestinalis or G. duodenalis) is the protozoan parasite responsible for giardiasis, a widespread and impactful gastrointestinal infection. This infection primarily occurs through the ingestion of cysts, which are resilient to various environmental factors, including the acidic conditions of the stomach. These cysts can be present in contaminated water sources, food items, or can be transmitted through direct person-to-person contact. Upon reaching the small intestine, specifically the duodenum and jejunum, the cysts undergo excystation, releasing trophozoites. These trophozoites then attach themselves firmly to the mucosal surface of the intestinal wall. This attachment disrupts the normal nutrient absorption processes, leading to a range of gastrointestinal symptoms. The disruption in nutrient absorption thus caused is a primary factor contributing to the clinical manifestations of giardiasis, which can include diarrhoea, abdominal cramps, and malabsorption [1].

Epidemiology

Giardiasis is recognized as one of the most common parasitic infections on a global scale, with an estimated 280 million people affected each year. This widespread prevalence is particularly noted in regions where sanitation infrastructure is poor and access to clean, safe drinking water is limited. Such conditions are prevalent in many developing countries, making giardiasis a significant public health concern in these areas. In the Indian subcontinent, the prevalence rates of giardiasis can vary widely depending on the region and specific population groups. For example, studies conducted in India have indicated that prevalence rates can be as high as 4.4% among children under the age of five in rural areas, reflecting the challenges of maintaining clean water and proper sanitation in less developed settings. In contrast, urban areas in Bangladesh have reported even higher prevalence rates, with figures reaching up to 6.6% among similar age groups. These higher rates are often observed in densely populated and low-income areas where the risk of water contamination is significantly elevated. The correlation between high population density, economic challenges, and the prevalence of giardiasis underscores the importance of improving water quality and sanitation facilities to combat the spread of this infection [2,3].

Pathophysiology

After ingestion, Giardia cysts undergo excystation in the small intestine, a process that releases the active trophozoite form of the parasite. These trophozoites utilize their ventral adhesive discs to firmly attach themselves to the intestinal mucosa. This attachment significantly disrupts the epithelial barrier, which is critical for maintaining nutrient absorption ability. The resulting epithelial damage manifests as villous atrophy, where the villi in the small intestine become blunted and less effective at absorbing nutrients. Additionally, crypt hyperplasia, characterized by the enlargement and proliferation of the crypt cells in the intestinal lining occurs, further compromising nutrient uptake.

The pathogenic mechanisms of Giardia are multifaceted. They include the mechanical blockage of nutrient absorption due to the physical presence of the trophozoites covering the intestinal

surface. Additionally, Giardia exerts direct cytotoxic effects, damaging the epithelial cells, and induces host immune responses that lead to inflammation and further epithelial injury. These combined effects disrupt normal digestive processes and lead to a range of gastrointestinal symptoms. Common clinical manifestations of giardiasis include diarrhoea, abdominal cramps, bloating, and nausea. Malabsorption of nutrients due to the damaged intestinal lining can also occur, often leading to significant weight loss and nutritional deficiencies in affected individuals. These symptoms highlight the substantial impact that Giardia infection can have on the health and well-being of individuals, particularly in areas with high rates of infection [1].

Diagnosis

Giardiasis is diagnosed primarily through the examination of stool samples. This is done by a variety of methods, including microscopic identification of cysts or trophozoites, antigen detection assays, and PCR. Microscopy is a traditional diagnostic approach that involves examining stool samples under a microscope to directly visualize Giardia cysts or trophozoites. However, this method can be labour-intensive and typically requires the collection of multiple stool samples over several days to increase diagnostic sensitivity, as the shedding of cysts can be intermittent.

Antigen detection assays, such as Enzyme-Linked Immunosorbent Assays (ELISA), have become popular due to their higher sensitivity and specificity compared to microscopy. These assays detect specific Giardia antigens in stool samples, allowing for a more reliable and quicker diagnosis. The advantage of antigen detection methods is that they can identify infections even when cysts or trophozoites are not visibly present in the sample.

Nucleic acid amplification tests, particularly Polymerase Chain Reaction (PCR), offer the highest accuracy in diagnosing giardiasis. PCR methods amplify specific Giardia DNA sequences, enabling the detection of low-level infections that might be missed by microscopy or antigen detection. Additionally, PCR can differentiate between Giardia and other similar protozoan parasites, providing a precise and definitive diagnosis. This high level of accuracy is crucial in both clinical and epidemiological settings, where understanding the exact cause of infection is essential for effective treatment and control measures [1].

Treatment

The primary treatment for giardiasis includes antiprotozoal agents such as metronidazole, tinidazole, or nitazoxanide. Metronidazole is the most commonly used, typically administered for 5-7 days. Tinidazole and nitazoxanide are alternatives with shorter treatment courses and similar efficacy. Supportive care with rehydration therapy is essential, especially in severe cases with significant fluid loss. Prophylactic measures, such as boiling or filtering drinking water, improving sanitation, and promoting good personal hygiene, are crucial in preventing giardiasis [1].

Amoebiasis

Entamoeba histolytica is the protozoan responsible for causing amoebiasis, a significant parasitic infection acquired primarily through the ingestion of cysts present in contaminated food or water. These cysts can survive the acidic environment of the stomach and, after reaching the more favourable conditions of the small intestine, they undergo excystation, releasing the active trophozoite form of the parasite. The trophozoites then invade the intestinal mucosa, causing extensive tissue damage and ulceration. This invasion disrupts the integrity of the intestinal lining, leading to a range of gastrointestinal symptoms.

The pathogenic activity of E. histolytica is not confined to the intestines. In severe cases, the trophozoites can migrate beyond the intestinal lining, through the bloodstream to extraintestinal sites, most commonly the liver. This can result in the formation of liver abscesses, a serious and potentially life-threatening condition. The process of invasion and tissue destruction by E. histolytica involves several mechanisms, including the production of proteolytic enzymes that degrade host tissues and the induction of host inflammatory responses that further contribute to tissue damage.

The clinical manifestations of amoebiasis can vary from mild diarrhoea to severe dysentery, characterized by abdominal pain, cramping, and the presence of blood and mucus in the stool. Extraintestinal manifestations, such as liver abscesses, can present with symptoms like fever, right upper quadrant pain, and hepatomegaly. These severe forms of the disease highlight the potential for E. histolytica to cause significant morbidity and necessitate prompt diagnosis and treatment to prevent complications [4].

Epidemiology

Amoebiasis is endemic in tropical and subtropical regions, where environmental and socioeconomic conditions contribute to its widespread prevalence. The Indian subcontinent shows significant rates of infection. Various studies have reported prevalence rates ranging from 4.5% to 7.4% in both urban and rural settings across the region. For instance, in Bangladesh, a study found a prevalence of 7.4% among children presenting with diarrhoea, highlighting the impact of amoebiasis on paediatric health in this area. Similarly, in Nepal, a prevalence rate of 6.2% was observed among school-aged children, indicating substantial infection rates within this demographic group.

In rural parts of India, the prevalence of amoebiasis has been documented at 5.8%, demonstrating that the infection is widespread across different demographics and geographic areas. These figures point at the endemic nature of amoebiasis in regions where sanitation and access to clean water are often inadequate. The high prevalence rates in both urban and rural settings reflect the persistent challenges in controlling the transmission of Entamoeba histolytica, which is facilitated by contaminated water sources and poor hygiene practices. This widespread prevalence necessitates ongoing public health efforts to improve water quality, sanitation, and health education to reduce the burden of amoebiasis in these regions [5,6].

Pathophysiology

Trophozoites of Entamoeba histolytica adhere to and invade the colonic epithelium, resulting in the formation of characteristic flask-shaped ulcers. This causes extensive cell death and tissue destruction, which manifests clinically as dysentery, characterized by bloody diarrhoea and severe abdominal pain. The damage to the intestinal mucosa results from the parasite's production of proteolytic enzymes and toxins, which degrade host tissues and provoke a significant inflammatory response.

In cases of invasive amoebiasis, the trophozoites can breach

the intestinal barrier and enter the portal circulation, leading to extraintestinal complications.

The most notable of these complications is the development of liver abscesses. When the trophozoites reach the liver, they can form abscesses filled with necrotic tissue and parasitic debris. These liver abscesses present clinically with symptoms such as right upper quadrant pain, fever, and hepatomegaly. The presence of these abscesses can lead to significant morbidity if not promptly diagnosed and treated. The pathogenesis of liver abscesses involves the destruction of hepatic tissue, leading to the formation of pus-filled cavities.

The ability of E. histolytica to cause both intestinal and extraintestinal disease relays the importance of early detection and effective treatment to prevent serious complications. Understanding the mechanisms of tissue invasion and immune evasion employed by the parasite is crucial for developing targeted therapies and improving outcomes for affected individuals [4].

Diagnosis

Diagnosis of amoebiasis involves stool microscopy, serological tests, and antigen detection assays. Stool microscopy is a fundamental diagnostic tool that allows for the identification of cysts and trophozoites of Entamoeba histolytica in stool samples. However, this method requires meticulous examination and often necessitates the analysis of multiple stool samples to increase diagnostic accuracy. Despite its utility, microscopy alone may not always provide a definitive diagnosis due to the potential for variability in sample quality and the need for skilled workers.

Serological tests, such as enzyme-linked immunosorbent assays (ELISA), are employed to detect antibodies produced against E. histolytica. These tests are particularly valuable for diagnosing invasive forms of amoebiasis, such as amoebic liver abscess, rather than solely intestinal infections. ELISA assays offer a higher sensitivity for detecting the presence of antibodies in the bloodstream, which can be indicative of more severe or systemic involvement of the disease.

PCR-based methods represent a highly accurate and advanced diagnostic approach. It allows for a precise detection of E. histolytica DNA, which aids in distinguishing between pathogenic E. histolytica and non-pathogenic Entamoeba species, such as E. dispar and E. moshkovskii. This high level of specificity and sensitivity makes PCR a valuable tool for confirming the diagnosis of amoebiasis and ensuring the appropriate treatment strategy is employed [4].

Treatment

The treatment for amoebiasis typically involves a two-phase approach: treating the invasive disease with metronidazole or tinidazole, followed by a luminal agent such as paromomycin to eliminate intraluminal cysts. Metronidazole is administered for 7-10 days, and paromomycin for an additional 7-10 days to ensure complete eradication. In cases of liver abscesses, drainage may be necessary if the abscess is large or unresponsive to drug therapy [4].

Cryptosporidiosis

Cryptosporidium species, notably Cryptosporidium parvum and Cryptosporidium hominis, are the primary pathogens responsible for causing cryptosporidiosis. The infection process typically starts with the ingestion of oocysts. These oocysts are notably resistant to many common disinfectants, making them difficult to eliminate from contaminated water sources and surfaces.

Upon ingestion, the oocysts travel through the digestive system and eventually release sporozoites in the small intestine, which penetrate the epithelial cells of the intestinal lining. The invasion of these cells leads to cellular damage and contributes to significant fluid loss. This cellular disruption and fluid loss can result in symptoms such as severe diarrhoea and abdominal pain, which are characteristic of cryptosporidiosis [7].

Epidemiology

Cryptosporidiosis is a major global health concern, particularly as a leading cause of diarrheal disease. It poses a significant risk to vulnerable populations, including young children under the age of five, and immunocompromised individuals. The Indian subcontinent, in particular, experiences notable prevalence rates of this infection, ranging from 5% to 10%. The rates tend to be higher during the rainy season, likely due to increased environmental contamination and the proliferation of the parasite in water sources.

In India, a detailed study conducted in Vellore found that the prevalence of cryptosporidiosis among children under five years old was 6.1%. Similarly, in Karachi, Pakistan, research indicated that 7.3% of children with diarrhoea were infected with the parasite. These figures underscore the considerable impact of cryptosporidiosis on young children in the region [8,9,10,].

Additionally, a study conducted in Kathmandu, Nepal, reported an even higher prevalence of 8.2% among children. This finding further highlights the widespread nature of the infection across the Indian subcontinent and underscores the need for effective public health interventions and preventive measures [11].

Pathophysiology

After ingestion, Cryptosporidium oocysts undergo excystation in the small intestine, releasing sporozoites. These sporozoites then penetrate and invade the epithelial cells lining the intestinal mucosa. Within these cells, the parasite undergoes replication, which induces complications like villous atrophy and crypt hyperplasia. Villous atrophy refers to the degradation of the intestinal villi, which are essential for nutrient absorption, while crypt hyperplasia involves the excessive proliferation of the intestinal crypts, leading to an imbalance in the normal cellular architecture of the gut.

These cellular disruptions severely disrupt intestinal absorption and secretion, resulting in symptoms such as watery diarrhoea, malabsorption of nutrients, and disturbances in electrolyte balance. This exacerbates the severity of diarrhoea and contributes to dehydration and nutritional deficiencies.

In immunocompromised individuals, such as those with HIV/AIDS, cryptosporidiosis can present with increased severity. The infection may become chronic and particularly severe, potentially leading to life-threatening complications. This is due to the body's reduced ability to effectively clear the parasite, which allows the infection to persist and cause more significant damage to the intestinal lining [7].

Diagnosis

Diagnosis of cryptosporidiosis involves detecting oocysts in

stool samples through microscopic examination using acid-fast staining, immunofluorescence, antigen detection assays, and PCR. Immunofluorescent assays and enzyme immunoassays are preferred for their sensitivity and specificity. PCR-based methods offer the highest diagnostic accuracy and can detect low levels of infection, which is particularly useful in epidemiological studies and outbreak investigations [7].

Treatment

Nitazoxanide is the only FDA-approved drug for treating cryptosporidiosis, although its efficacy varies, especially in immunocompromised patients. Supportive care with rehydration therapy is crucial, particularly in severe cases with significant fluid loss. For HIV-infected individuals, initiating or optimizing antiretroviral therapy (ART) is essential as it helps restore immune function, which can reduce the severity and duration of cryptosporidiosis. Preventive measures, such as avoiding contaminated water and practicing good hygiene, are critical in high-risk areas [7].

Conclusion

Giardiasis, amoebiasis, and cryptosporidiosis are significant public health concerns, particularly in regions with poor water and sanitation infrastructure. Understanding their mode of infection, diagnosis, and treatment is essential for effective management and prevention. Continued efforts to improve water quality, sanitation, and public health education are vital in reducing the burden of these diseases worldwide.

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