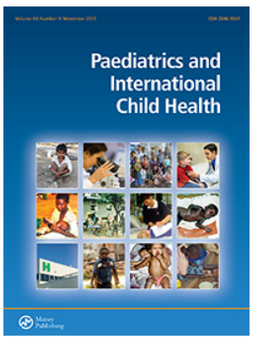

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Amoebic colitis presenting with hypo-albuminaemia in an eight-month-old breastfed girl

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ABSTRACT

Entamoeba histolytica is a protozoan parasite that affects a large proportion of the world's population and causes amoebic dysentery and extra-intestinal disease. Many individuals remain asymptomatic during colonisation; in 10% of individuals, the parasite breaks through the mucosal barrier and leads to invasive disease. An eight-month-old girl who was evaluated for hypo-albuminaemia and was diagnosed with amoebic colitis is reported. To the best of our knowledge, this is the first report of hypo-albuminaemia owing to amoebic colitis in any age group.

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Amoebic colitis; hypo-albuminaemia

Introduction

Entamoeba histolytica is a protozoan that is common throughout the world, especially in low- and middle-income countries [1,2]. Transmission is usually by consumption of food and water contaminated with amoebic cysts or directly by faecal–oral transmission. In many individuals, the parasite remains silent during colonisation, but in around 10% of patients it breaks through the immune barrier and causes dysentery and extra-intestinal disease [3]. Clinical onset is usually subacute and symptoms range from mild, non-bloody diarrhoea to fulminant amoebic colitis with a mortality rate of up to 40%. The course of the disease depends on the host's immunity, which acts as a barrier against the parasite [4,5]. An eight-month-old girl who was investigated for hypo-albuminaemia was diagnosed with amoebiasis is reported. Previously reported presentations of *E. histolytica* infection are amoebic colitis, liver abscess and pulmonary, cardiac and brain involvement. Hypo-albuminaemia in parasitic infections has been reported previously for *Giardia lamblia, Strongyloides stercoralis* and *Blastocystis hominis* [6,7]. However, hypo-albuminaemia owing to amoebic colitis has not been reported before in any age group.

Case report

A formerly healthy and partially breastfed six-month-old girl was admitted to hospital with vomiting three or four times and diarrhoea five or six times a day for three weeks. Her mother was on a normal diet and there was no known disease in the infant's past medical history. The diarrhoea was predominantly watery and no blood was observed in the stool. No-one else in the family had the same symptoms. The patient was evaluated for gastro-enteritis and only fluid replacement was given for mild dehydration. After four days in hospital she was discharged. The diarrhoea improved, although the patient continued to vomit and was diagnosed with gastro-oesophageal reflux disease. No medical treatment was given for the proposed reflux. By the second week of follow-up, she had lost weight and had bilateral oedema of the feet. On her second admission after three weeks of follow-up, the patient had mild dehydration and laboratory tests revealed hypo-albuminaemia [albumin 11.7 g/L (35–52)] and hyponatraemia (sodium 128 mmol/L). She was referred to Ihsan Dogramaci Children's Hospital for further investigation two months after her initial admission.

Physical examination including abdominal and anal examination was normal except for bilateral oedema of the feet. Complete blood count test, CRP and erythrocyte sedimentation rate were normal. Serum albumin was 12.5 g/L, total protein 30.5 g/L (51–73) and urinalysis was normal with no proteinuria. Liver enzymes and renal function tests were normal. The patient was investigated for possible causes of hypo-albuminaemia. Sweat chloride and faecal elastase tests were normal, and tests for stool ova and parasites were negative. Serum IgG was 79.3 mg/dL (220–900) and IgM 10.4 mg/dL (46–304)

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which were low for her age. Lymphocyte subsets and antibody responses were normal excluding combined immunodeficiency, and also there was no history of recurrent infections or fever. The vitamin B_{12} level was 49 pq/mL (126–590) and replacement therapy was commenced. An abdominal ultrasonography demonstrated dilation with fluid in the bowel segments. Upper gastro-intestinal endoscopy and rectosigmoidoscopy were performed. Except for mild antral hyperaemia, the endoscopic appearances were normal. Histolopathological examination of the colon demonstrated *E. histolytica* along with mild inflammation. On the 5th day of admission, metronidazole was commenced in a dose of 30 mg/kg/day for 10 days. Following treatment, the albumin level increased markedly and was within the normal range (37.8 g/L) on Day 20 after commencing therapy; the oedema resolved, as did the other symptoms. At the last six-month follow-up visit, she was well with normal serum albumin and total protein levels.

Discussion

E. histolytica may exist in two forms: the cyst stage and a trophozoite stage. Cysts can remain viable in the environment for weeks to months, and ingestion of a single cyst is sufficient to cause disease. Infection occurs following the ingestion of the cysts (usually through contaminated food or water) and after excysted, the trophozoite form causes invasive disease.

The vast majority of infested people remain asymptomatic and in only around 10% the trophozoite breaks through the immune barrier and causes dysentery and extra-intestinal disease [3]. The course of the disease depends on the parasite's virulence and the host's immunity which acts as a barrier against the parasite [4,5]. The trophozoite causes colitis by different mechanisms, including production of *E. histolytica* cystein proteases, the killing of neutrophils with apoptotic cell death, phagocytosis and a recently found mechanism termed amoebic trogocytosis where the trophozoites ingest different parts of the host cells [2]. Clinical onset of the disease is usually subacute and symptoms range from mild non-bloody diarrhoea to fulminant amoebic colitis with a high mortality rate of up to 40%. Although this patient's presenting symptoms were not specific for amoebiasis, it rarely presents with chronic diarrhoea accompanied by weight loss or non-specific gastro-intestinal symptoms with abdominal pain and the presentation may also mimic inflammatory bowel disease. Diagnostic tools for amoebiasis include direct microscopic investigation, antigen detection and histological examination of the bowel through colonoscopy [3,8]. Rapid diagnostic tests are highly sensitive and there are also specific screening methods in high-income countries therefore direct microscopic examination of stool can only be used as a complementary technique when other diagnostic methods are unavailable [9]. In this case, the diagnosis was made by histological examination of the biopsy specimens and a second test to confirm the diagnosis was not required. All *E. histolytica* infections should be treated owing to the risk of invasive disease and also the risk of spreading the infection to the other family members. Metronidazole, ornidazole and tinidazole are the treatment options with high cure rates [10–12]. In this patient, the main symptom was bilateral oedema of the feet owing to hypo-albuminaemia and the probable reason for hypo-albuminaemia was protein-losing enteropathy (PLE). PLE is defined as excessive protein loss due to the impaired integrity of intestinal mucosal barrier and clinical findings differ according to the underlying diseases. The causes of PLE in childhood include mucosal injury owing to inflammatory bowel disease, parasitic, bacterial and viral gut infections and non-ulcerative diseases, including hypertrophic gastropathies, eosinophilic gastro-enteritis, food-induced enteropathies, coeliac disease and lymphatic abnormalities such as intestinal lymphangiectasia [13]. No other cause of the hypo-albuminaemia apart from *E. histolytica* infection was found. As far as we are aware, hypo-albuminaemia owing to *E. histolytica* infection has not been reported previously in children or adults.

In conclusion, *E. histolytica* colitis should be considered as a cause of PLE and hypo-albuminaemia.

Disclosure statement

No potential conflict of interest was reported by the authors.

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