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UNUSUAL PRESENTATION OF CHRONIC GIARDIASIS

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Abstract : We report an 11 year old girl who was a previously healthy girl, presented with five month history of sudden and significant weight loss. On examination was found to be severely emaciated, hypotensive, with edema secondary to hypoalbuminemia,severe dyselectrolytemia and atrial brillation. After an extensive evaluation for the cause of clinical state all her symptoms were attributed to chronic Giardiasis. She improved completely and regained her original weight on effective treatment of this parasite.

Keyword : Hypoalbuminemia , dyselectrolytemia , chronic giardiasis , atrial fibrillation

CASE REPORT

Eleven year old girl presented to the pediatric casualty with history of significant loss of weight over the past five months. She has been studying in a boarding school and except for occasional pasty stools sometimes there was no history of ill health. There was no history of decreased intake of food, fever, and contact with tuberculosis. She did not give any history of being depressed, having a poor body image or induced vomiting. There was no past significant medical history. Child was developmentally normal and immunized up to date. On examination she was alert and responsive. She had atrial fibrillation and low blood pressure (BP 80/55 mm hg) and looked severely emaciated and had flaky skin. Her weight was 26.5kg against a expected of 36 kgs (<3rd percentile as per CDC growth charts). She was pale and had pedal edema present .There was no icterus or generalized lymphadenopathy. Rest of her systemic examination done was normal. The differentials of malabsorption syndromes, inflammatory bowel disease, tuberculosis, protein losing enteropathy, immunodeficiency, anorexia nervosa and addison's disease were considered. Initial blood investigations showed a macrocytic anemia with a hemoglobin of 4.5 gm/dl with normal white cell counts, though the levels of vitamin B12 and folate were within normal limits. She had severe hyponatremia (Na-114 mg/dl),hypokalemia(K-1.7 mg/dl), hypocalcemia (Ca-4.6 mg/dl),hypophosphatemia(P-1.4 mg/dl), hypomagnesmia (Mg-1.21 mg/dl) , hypoalbuminemia (1.0 gm/dl). The arterial blood gas showed metabolic alkalosis with a pH of 7.45 with bicarbonate of 32.7. She was immediately resuscitated with fluids, started on hydrocortisone (after taking her cortisol level) and inotropes. She was started on broad spectrum antibiotics after blood culture. She also required recurrent albumin transfusions.Her atrial fibrillation was attributed to the severe dyselectrolytemia (ECHO done was normal) and her electrolytes were corrected over a period of 7 days but required oral supplementation for a total period of 2 months. She required 4 mmol/kg of potassium for prolonged period. Her cardiovascular parameters gradually returned to normal. She was extensively evaluated for a possible malabsorption state, stool examination showed Giardia lamblia cysts and few fat globules. Stool occult blood , anti-TTG ,xylose test and cultures were negative.

Upper GI scopy and colonoscopy were done which were non contributary(Minimal chronic duodenitis with mild chronic atrophic gastritis. CT abdomen was also normal except for minimal ascites. Infectious causes like TB and HIV were ruled out and immunodeficiency states (Immunoglobulin levels were normal) were ruled out. Renal causes for her dyselectrolytemia were evaluated. Differentials of Bartter syndrome, Gittleman syndrome and Pseudobartters in view of hypokalemia, hypomagnesemia and metabolic alkalosis.Renin levels done were normal (0.1 ng/ml/hr) but aldos terone level was low(<10).The possibility of primary hypomagnesemia(due to defect in absorption of magnesium) with hypocalcemia secondary to in PTH unresponsiveness due to hypomagnesemia was considered. The PTH level done was normal. In a case report by Woodard et al (1) hypocalcemia, hypomagnesemia and hypoalbuminemia was seen in a 2 month old infant who presented with diarrhea and seizures. Hypoalbuminemia could be because of decreased protein absorption and decreased albumin production.Also the possibility of increased tubular losses secondary to chronic diarrhea was considered(Both urine spot Na and K were elevated) and the metabolic alkalosis was attributed to probable contraction alkalosis secondary to chronic diarrhea. Sweat chloride done was normal (30 mmol/L) which ruled out pseudo-barter syndrome. In view of severe dyselectrolytemia paediatric endocrinology consult was obtained,who suggested the posssiblity of polyendocrinopathy syndrome,though the investigations done were within normal limits. Her electrolytes were repeated after discontinuing all IV correction for 48hrs, which was in normal limits.

She was also seen by child and adolescent psychiatrist and eating disorder was ruled out (Not fulfilling DSM IV criteria).

She was treated with a course of Metronidazole dose Inj.Flagyl 350mg iv tid x 7days followed by Tab.Flagyl 200mg tid x 7. She was nutritionally rehabilitated with high protein and high carbohydrate diet with electrolyte supplementation. During the hospitalization she developed nutritional rehabilitation syndrome

With worsening hypophosphatemia, which was managed by increasing the supplementation. Slowly she improved and we were able to discharge her on oral potassium, calcium, phosphate and vitamin D supplementation. On follow up her stool still showed Giardiasis and she received 2 more courses of Metronidazole. Her electrolytes and other metabolic parameters normalized on subsequent visits without any other intervention. She was followed up for 3 months and she gained 11 kgs (weight after 3 months was 37.4 kgs) and did not have any other problems.

DISCUSSION

Giardia is the most prevalent human intestinal parasite in the world (2). Infection is more common in worsening hypophosphatemia, which was managed by increasing the supplementation. Slowly she improved and we were able to discharge her on oral potassium, calcium, phosphate and vitamin D supplementation. On follow up her stool still showed Giardiasis and she received 2 more courses of Metronidazole. Her electrolytes and other metabolic parameters normalized on subsequent visits without any other intervention. She was followed up for 3 months and she gained 11 kgs (weight after 3 months was 37.4 kgs) and did not have any other problems.

DISCUSSION

Giardia is the most prevalent human intestinal parasite in the world (2). Infection is more common in children than in adults. The etiological agent, Giardia duodenalis (syn. G. intestinalis , G. lamblia) is a flagellated, binucleated protozoan parasite which infects a wide array of mammalian hosts . Human giardiasis is distributed worldwide, with rates of detection between 2-5% in the developed world and 20-30% in the developing nations such as India(3) The symptoms of giardiasis include abdominal cramps, nausea, and acute or chronic diarrhea, with malabsorption and failure of children to thrive occurring in both sub-clinical and symptomatic disease (4).There is significant variation in the outcome of Giardia infections. Most infections are self-limiting, although re-infection is common in endemic areas and chro nic infections also occur. Moreover, some individuals suffer from severe cramps, nausea and diarrhea while others escape these overt symptoms.

The manifestations may wax and wane over many months. Malabsorption may be responsible for the significant weight loss that can occur in giardiasis. Malabsorption and maldigestion mainly result from a diffuse shortening of epithelial microvilli. This enterocytic injury is mediated by host T lymphocytes activated by disruption of epithelial tight junctions and giardia-induced enterocyte apoptosis(2). Even in cases of otherwise asymptomatic infection, malabsorption of fats, sugars, carbohydrates and vitamins may occur .This can lead to hypoalbuminemia ,dyselectrolytemia and deficiencies of various vitamins.

Acquired lactose intolerance occurs in up to 40 percent of patients; clinically, this manifests with exacerbation in intestinal symptoms following ingestion of dairy products (5). Recovery can take many weeks, even after clearance of the parasite. In a study done by Sullivan et al (6)in Gambian children with chronic diarrhea, 14 out of 31 children had giardiasis compared with only four of 33 healthy age and sex matched control children. Twenty three children with chronic diarrhea were reinvestigated after treatment with metronidazole; giardia was found in 11 of them. These results show that giardia is highly prevalent in children with chronic diarrhoea and malnutrition and that the infection does not respond to standard therapeutic measures. In a study done by Farthing et al(7) on Guatemalan children showed duration of Giardia episodes and their association with diarrhea appeared to be the most important factors associated with growth disturbance. In a similar case reported by Thomas et al (8) ,a 13 year old with suspected anorexia nervosa was subsequently found to have chronic giardiasis. Also in a case reported by Gunasekaran et al (9) an adolescent with systemic features suggestive of inflammatory bowel disease was found to have Giardiasis and the symptoms completely resolved with treatment with metronidazole . Symptom recurrence, including abdominal symptoms and fatigue, can result from re-infection, treatment failure, disturbances in the gut mucosa or post-infection Syndromes In the treatment of Giardiasis the greatest clinical experience is with the nitroimidazole drugs, i.e., metronidazole, tinidazole, and ornidazole, which are highly effective. A 5- to 7-day course of metronidazole can be expected to cure over 90% of individuals, and a single dose of tinidazole or ornidazole will cure a similar number(11). The published rates of cure vary for different regimens, but they are frequently reported to be 90%.Nevertheless, some individuals experience treatment failure, despite having received successive courses of treatment that have been documented to result in a cure for most patients. There are 6 potential causes of treatment failures: reinfection, inadequate drug levels, immunosuppression, resistance to the drug, sequestration in the gallbladder or pancreatic ducts, and unknown reasons.In a case series done by Theodore et al(12) , 6 of the patients were treated with metronidazole ortinidazole in combination with quinacrine, and this regimen resulted in cure for 5 of the 6 patients. Metronidazole and quinacrine are more effective than furazolidone, but furazolidone has the advantage of a liquid formulation that makes administration to children easier(13). The presence of macrocytic anaemia(MCV-122 and Hb-4.5 gm/dl) with normal B12 and folate could not be explained.Other causes such as hypothyroidism and autoimmune hemolytic anaemia were excluded.The case was discussed was discussed with clinical haematologist, who suggested that the MCV might be erroneously high on account of the increased rouleaux formation.The other possibility of vitamin E responsive macrocytic anaemia was also considered,since the level of vitamin E were not done,this could not be confirmed(14).

CONCLUSION:

Chronic giardiasis is a rare and enigmatic disease that presents with many symptoms similar to chronic gastrointes-tinal disorders (e.g. IBD and celiac disease) and anorexia nervosa and be considered as a differential for these diseases. Severe dyslectrolytemia and hypoalbuminemia can also be present with seen in chronic giardiasis.

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