Case Report

Ulcerative colitis in a Nigerian child: a case report

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ABSTRACT

Background: Ulcerative colitis (UC) is an idiopathic chronic inflammatory disorder characterized by unpredictable exacerbations and remissions. It is thought to be rare in Africans especially amongst paediatric age group and there has been only one presumed paediatric case report from Nigeria. Case Presentation: We report the case of a 13.5 year old boy who presented with typical symptoms and in whom the diagnosis of UC was confirmed by endoscopic examination and histology. He responded well to treatment with sulfasalazine tablets. Conclusion: This plus an earlier report suggest that UC may not be as rare in Nigeria as it was previously thought to be perhaps due to under-diagnosis caused by a lack of diagnostic facilities. A high index of suspicion is required in diagnosis and epidemiologic studies are required to determine its actual prevalence.

Key words: Ulcerative colitis, Nigerian, Child

INTRODUCTION

Ulcerative colitis is an idiopathic chronic inflammatory disorder localized to the colon and characterized by unpredictable exacerbations and remissions.1 It is a life-long disease.1,2 The exact aetiology of ulcerative colitis is unknown but interactions between genetic and environmental factors may be involved.1,2 The incidence of ulcerative colitis varies between countries.1,3 It is commoner in the western world and northern hemisphere than in Asia and the Far East and in Whites than in Black or Hispanics.1,3

Helmholtz reported the first paediatric case in 1923, and although several paediatric cases have been reported since then, very few have been reported from Africa and only one from Nigeria.4-6 This may be attributable to the purported rarity of the disease in Africans and children.1,2,7,8 The case reported here is the second from Nigeria and it illustrates that the apparent rarity of UC in African children may be due to a low index of suspicion and limited diagnostic resource.6,8

CASE REPORT

E.O, a thirteen and half year old boy, was referred after multiple hospital consultations to the Paediatric Gastroenterology Unit of Irrua Specialist Teaching Hospital, Irrua with complaints of abdominal pain of one year, passage of watery stools of nine months, weight loss of six months and fever of three months.

His problems started with the development of abdominal pain which was generalised and crampy, waxed and waned and was more marked in the epigastrium and left flank. There was no known relieving or aggravating factor. The stools were watery, contained mucus and streaks of blood occasionally and passed 4 times daily. He had no tenesmus. There was no associated nausea, vomiting, jaundice, oral thrush, night sweats, anthralgia or visual challenges. He had no history of sexual exposure. He was the 3rd of 4 children of the mother and the 9th of 14 children of the father who had 3 wives, and was the village king and had tertiary level of education.

There had been no period of respite since the onset of passage of watery stools and he had lost weight as evidenced by loosening of previously tight fitting clothes and progressive weight reduction in his medical records. He also had fever without chills or rigors. His source of drinking water was a bore hole and sewage disposal was by water closet. He was on a regular adult diet with the other house hold members but he patronised food vendors.

He had been treated with mixed magnesium trisilicate and antibiotics for the abdominal pain and dysentery without respite. He had no history of blood transfusion but was admitted four months prior to the onset of illness for cellulitis of the right leg. He was pulled out of boarding house due to the illness. He had no history of sexual exposure. He was the 3rd of 4 children of the mother and the 9th of 14 children of the father who had 3 wives, and was the village king and had tertiary level of education.
education. His mother sold kitchen utensils in a shop. No other household member had a similar illness.

Examination showed a pre-pubertal boy who was small for age with a weight of 32.9kg (68.5% of expected weight for age and the weight of a 10-year-old boy); the height of 147.5cm (92.2% of expected) and BMI = 15.1Kg/m². The skin was lustreless and with a generalised variegated rash; and some hyperpigmented and other healing ulcers. He was pale but anicteric and had no digital clubbing, peripheral lymphadenopathy, pedal oedema or evidence of uveitis. There was no tenderness, organomegaly or ascites on examination of the abdomen. Normal bowel sounds and no rectal abnormality was found on examination. All the other systems were also normal examinations.

The packed cell volume was 25.5%, white cell count 7,800 cells/mm³ with differentials of neutrophils 58.4%, lymphocytes 30.1% and eosinophils, monocytes and basophils 11.5%. The blood film examination showed anisocytosis, poikilocytosis, microcytosis and hypochromasia. The erythrocytes sedimentation rate was 130mm/hr. The retroviral screen was negative. Microscopically, the stool was brown watery and mucoid but not blood stained. No parasite, or ova or cysts was seen on microscopy, but there were over 20 pus cells/HPF. Culture of the stool yielded *E. coli*, which was sensitivity to chloramphenicol, gentamicin, ceftriaxone and ofloxacin. Urinalysis was normal.

The provisional diagnosis was immunosuppression, with the differential of paediatric HIV infection, food allergy, milk diarrhoea and inflammatory bowel disease were made. He was managed as an outpatient with oral rehydration salts – solution, 200mg of zinc per orum twice daily for two weeks, 200mg of cefixime per orum daily for a week and 400mg of albendazole statin per orum. Milk consumption was also stopped. However, the symptoms failed to resolve after the two months of management and the differential diagnosis of inflammatory bowel disease had to be given further consideration. Endoscopy was then carried out and showed moderately inflamed mucosa involving the entire colon, with bleeding ulcerated patches, which were more severe proximally, absence of normal intervening mucosa and small quantities of mucus in the lumen (figure 1). There were no masses, stenosis, deformities or internal haemorrhoids.

The diagnosis of UC was confirmed from the histology of biopsy samples taken during endoscopy (figure 2) which showed an intense lymphoplasmacytic inflammation of the lamina propria with basal plasmacytosis. There is marked glandular distortion characterized by dilated, branched and irregular crypts (figure 3). Also seen was a focus of crypt abscess. A conclusion of ulcerative colitis, moderately active disease was made.

*Figure 1. Moderately inflamed mucosa involving the colon*

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*Figure 2. Medium power photomicrograph showing marked lamina propria lymphoplasmacytic infiltrates and extensive glandular distortion. (H&E) x 100*

*Figure 3. High power photomicrograph showing cryptitis and crypt abscess. (H&E) x 400.*

Following confirmation of the diagnosis, he was treated with sulphasalazine [5-aminosalicylic acid (5-ASA)] given orally at 60mg/kg/day after counselling of the patient, his mother and eldest sister about the disease and treatment protocol. Approximately six months after the commencement of
treatment, both abdominal pain and fever have resolved and he has ceased to lose weight. However, bowel opening is still three to four daily with occasional watery stools but no streaks of blood and he feels better.

**DISCUSSION**

Diarrhoea with blood, mucus and/ or pus in the stool with urgency and nocturnal bowel movements are the typical presenting features of UC. Other complains include crampy lower left quadrant abdominal pain especially with bowel movements, anorexia, weight loss and growth failure. The extracolonic manifestations such as uveitis, pleuritis were absent. The characteristic endoscopic features of UC were present as were also the typical histopathologic features. Using the Paediatric Ulcerative Colitis Activity Index (PUCAI), this child had a moderate disease activity index of 50 at diagnosis, which had reduced to mild disease activity of 10 by six months on treatment. This demonstrates improvement of his health objectively.

There is no medical cure for UC. The treatment in children is aimed at achieving the best possible clinical and laboratory control of the disease with minimal adverse effects and permitting function as normally as possible. This patient responded to treatment with Sulfasalazine, the mainstay of treatment for UC. Moderate to severe exacerbation of UC are managed with steroids to induce remission, but the long term effects of steroids limits prolonged use. Other medical treatment include the use of immunomodulators such as azathioprine, 6-mercaptopurine for steroid-dependent or refractory disease and cyclosporine for fulminant colitis. Colectomy is reserved for intractable disease, complications of therapy, and fulminant disease unresponsive to medical management. Psychosocial support is also important as part of the management.

**CONCLUSION**

This case taken together with the previous report indicate that UC may not be so rare in Nigeria contrary to the earlier perceptions. This case also illustrates the necessity of a high index of suspicion in the diagnosis of UC in children.

**REFERENCES**