

Ekanem EE
Ikobah JM
Ngim OE
Okpara HC

Ulcerative colitis in a Nigerian child: case report

DOI:<http://dx.doi.org/10.4314/njp.v43i4.11>

Accepted: 2nd June 2016

Ekanem EE (✉)
Ikobah JM, Ngim OE
Okpara HC
Department of Paediatrics,
University of Calabar/University of
Calabar Teaching Hospital, Calabar,
Cross River State, Nigeria
Email:
emmanuelekanem@unical.edu.ng

Abstract: Ulcerative colitis (UC) is a chronic re-occurring inflammatory disease affecting mainly the colon. It is more prevalent in the United Kingdom, North America Scandinavia and less common in southern Europe, Asia and Africa. Commonly, UC is suspected in patients presenting with bloody diarrhoea, tenesmus, abdominal pain, and, when severe, weight loss, fatigue, and vomiting. Perhaps one child so far with UC has been reported south-west geo-political zone of Nigeria. We here report the first case of ulcerative colitis in a child in south-south Nigeria.

The objective of this report was to highlight the occurrence of ulcerative colitis in a Nigerian child in the setting of poor socio-psychological/economic background coupled with difficulty in investigating patient to arrive at a

diagnosis. An 11 year old Nigerian male child who was referred to the University of Calabar Teaching Hospital with bleeding per rectum, abdominal pain and swelling, bilateral leg swelling and weight loss for seven months prior to presentation. Examination and investigations done including colonoscopy, histology of biopsy, faecal calprotectin and pANCA confirmed ulcerative colitis. Ulcerative colitis though rare in Africa may have been missed in some children due to mis-conception and lack of diagnostic facilities/expertise. We may begin to see more of this with increasing interest in the subspecialty of paediatric gastroenterology and presence of diagnostic facilities.

Key word: Ulcerative Colitis, Diagnostic difficulties, Nigerian child

Introduction

Ulcerative colitis (UC) is a chronic re-occurring inflammatory condition of the colon, extending continuously from the rectum proximally to a varying degree. Most children with ulcerative colitis present between the ages of 10 and 18 years.^{1,2} The incidence of pediatric-onset UC, forms roughly 15% to 20% of patients of all ages with UC, ranging between 1 and 4 of 100,000/year in most North American and European regions.¹⁻³ Children and adults develop similar symptoms however children often present with more extensive disease.⁴ Childhood-onset UC is extensive in 60% to 80% of all cases, twice as often as in adults⁴ with 82% of children at first presentation have a pancolitis compared to 48% of adults.⁵ The extent of the disease is associated with disease severity, therefore, most pediatric-onset UC have a worse disease course.⁶ The pathogenesis of ulcerative colitis is unknown. A widely accepted hypothesis suggests that, in the genetically susceptible individual, a combination of host and environmental factors lead to the initiation and perpetuation of an abnormal intestinal immune response, resulting in Ulcerative colitis. Typically, UC is suspected in a patient presenting with bloody diarrhoea,

tenesmus, abdominal pain, and, when symptoms become severe, weight loss, fatigue, and vomiting. Children have unique age-related considerations, such as growth, puberty, nutrition, and bone mineral density (BMD) accretion during adolescence, as well as differing psychosocial needs and development.^{7,8}

Ulcerative colitis is thought to be rare in sub-Saharan Africa, however, in Nigeria, ulcerative colitis has been reported in adults.^{9,10} It was also reported in a seven year old female child in south-west Nigeria.¹¹ We report a confirmed case of ulcerative colitis in an 11 year old child in south-south Nigeria.

Case report

This was an 11 year old adolescent male who presented to the University of Calabar Teaching Hospital, Calabar, Cross River State, Nigeria with history of bleeding per rectum, abdominal pain, abdominal swelling and weight loss of seven months duration. Patient's stool was mixed with fresh blood. Abdominal swelling was insidious in onset and gradually increased over time. There was associated history of abdominal pain which was dull and continuous in nature. There was a positive history of

vomiting and easy satiety. There was a positive history of weight loss and fever. Leg swelling occurred at same time as abdominal swelling. There was history of dyspnoea on exertion, no orthopnoea or paroxysmal nocturnal dyspnoea. He was given herbal preparation as enema in a herbal home. He also received treatment from a secondary health facility from where he was referred to the UCTH. Patient is the second child in a family of two. Parents are separated and patient lives with the grandfather, a peasant farmer.

On examination he was acute on chronically ill looking, conscious, wasted, small for age, a febrile, severely pale, jaundiced with bilateral pitting pedal oedema. Respiratory rate was 32 cycles/minute, chest was clinically clear, pulse rate was 88/minute, blood pressure was 100/60 mmHg, heart sound was S1 and S2. Patient had a grossly distended abdomen with tenderness over the right and left hypochondria, liver was enlarged by 8 cm below the right costal margin firm, tender, nodular, with poorly defined edge. The spleen was 6 cm enlarged below the left costal margin, firm, smooth, non tender. Fluid thrill was demonstrable. Rectal examination revealed good anal hygiene, anal tags present at 4 o'clock, 7 o'clock and 11 o'clock positions, normal sphincteric tone, no palpable rectal masses. Examining finger stained with faeces mixed with blood. An initial diagnosis of chronic liver disease was made.

Results

Packed cell volume ranged between eight percent and 19%, patient was transfused four times while on admission. Full blood count showed total white cell count of 5.8%, erythrocyte sedimentation rate of 55 mm/hr (Westergren method), neutrophil of 67%, lymphocyte of 33%, evidence of anisocytosis, microcytosis, hypochromasia and poikilocytosis. Liver function test done showed total bilirubin of 58 $\mu\text{mol/l}$ (normal range of 0.0-17.0), direct bilirubin of 45.5 $\mu\text{mol/l}$ (normal range of 0.0-6.8), indirect bilirubin of 12.8 $\mu\text{mol/l}$ (normal range of 0.0-10.3), alanine aminotransferase/SGPT of 41.0 U/L (normal range of 0-40), aspartate transferase/SGOT of 80.0 U/L (normal range of 0-37), alkaline phosphatase of 753 U/L (normal range of 245-770), total protein of 57 g/L (normal range of 55.0-82.0), albumin of 34 g/L (normal range 37.0-53.0), globulin of 23 g/L (normal range 15.0-36.0), GGTP 243.4 U/L (normal range 10.0-71.0). C-reactive protein level was within normal limit. Lipid profile showed total cholesterol 3.6 mmol/l (normal range 3.6-5.2), HDL Cholesterol 1.6 mmol/l (normal range of 0.9-1.5), LDL Cholesterol 0.7 mmol/l (normal range of 1.9-3.5), triglyceride 2.9 mmol/l (normal range of 0.6-1.7). Initial prothrombin time was 30.5 seconds (prothrombin control 15 second), international normalized ratio (INR) of 2.13. A repeat done after vitamin K administration showed prothrombin time was 23.6 second (prothrombin control 15 second), international normalized ratio of 1.65. Stool microscopy, culture and sensitivity result was normal except for pres-

ence of 5-6 RBC per hpf. Echo-cardiography result was normal. Hepatitis B and C screening was negative and HIV test was also negative. Summary of findings of abdominal ultrasound scan using a 3.5 MHz curvilinear probe includes mild enlarged and coarse looking liver with caudate lobe enlargement, minimal ascites and splenomegaly, gall bladder enlargement and increase in wall thickness but no convincing evidence of portal hypertension.

C-reactive protein assay was within normal limit. Liver biopsy was not done due to the deranged INR. Faecal calprotectin result was 155.57 mg/kg faeces (biological reference interval <25 mg). Peri-nuclear anti-neutrophil cytoplasmic antibody (p-ANCA) was positive. Upper and lower GI endoscopy was done and samples taken for histology and cytology. Both the faecal calprotectin and p-ANCA assay were done in India as we could not find a local laboratory that could perform the test. Upper GI endoscopic findings were normal with no evidence of varices. Colonoscopy showed extensive haemorrhagic mucosa of the large bowel all the way to the transverse colon (Fig. 1). Lesions were worse in the rectosigmoid area. No ulcers, polyps, diverticular or tumour were seen and no haemorrhoids. Cytology was negative for malignancy and histology result showed chronic non-specific colitis (Fig. 2). The activity of UC using the paediatric ulcerative colitis activity index (PUCAI) for the index patient was a score of 45 giving him a moderate disease at diagnosis.

Fig 1

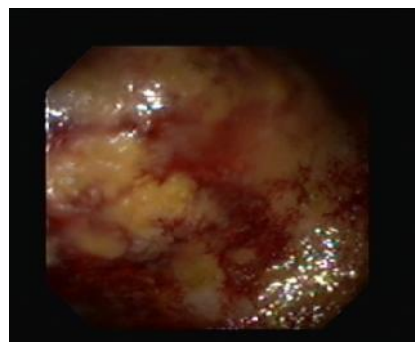
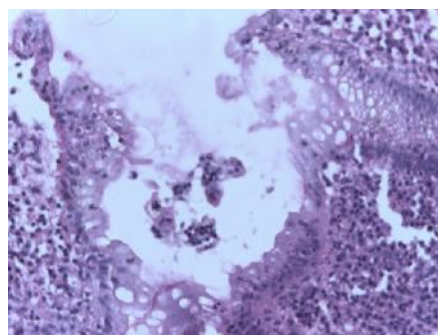


Fig 2



Discussion

UC is known to occur globally though less common in Africa and especially sub-Saharan Africa. This could be due to lack of diagnostic expertise and the fact that there is still a high burden of infective diseases to battle with

and the thought of UC commonly seen as a westernized illness is far from it. However, with increasing interest in gastroenterology as a sub-specialization and improving expertise, few cases has been reported in adults in Nigeria^{9,10} Ghana¹² South Africa,¹³ Uganda.¹⁴ UC has also been reported in a seven year old Nigerian child but without the current diagnostic investigations of endoscopy, faecal calprotectin and P-ANCA to confirm this hence our patient is the first confirmed case of UC in a paediatric patient in Nigeria.¹¹

As in other reported cases in Africa, the diagnosis of UC in our index patient was not thought of initially at first presentation. Patient was treated for dysentery and with persistence of passage of bloody diarrhoea, deranged liver function test and the need for recurrent blood transfusion, patient had a colonoscopy with biopsy done and UC was confirmed. This therefore underscores the need for a high index of suspicion even in African children with chronic bloody diarrhoea, weight loss and recurrent anaemia. Cabrera-Abreu¹⁴ showed a diagnostic sensitivity and specificity of 90.8% and 80% respectively in patients with signs suggestive of IBD and existence of anaemia or thrombocytosis. In children with UC, blood loss could occur in 84%, diarrhoea in 74% and abdominal pain in 62% of patients.¹⁵ Weight loss is less common in UC (35%) than CD (58%).¹⁵ Our patient presented with all these symptoms. Extra-intestinal manifestations of IBD may be present in 25% to 35% of children.¹⁵ Our patient presented with perianal disease (skin tags) and hepatic disease. Hepatic abnormalities in children with ulcerative colitis have been well described.¹⁶ While these are typically identified after the ulcerative colitis diagnosis, they may also precede the gastrointestinal symptoms.¹⁶ Transient elevations of alanine aminotransferase (ALT) occur in 12% of children with ulcerative colitis and appear to be related to medications or disease activity. Persistent ALT elevations suggest the presence of primary sclerosing cholangitis (PSC) or autoimmune chronic hepatitis.¹⁷ Among children with ulcerative colitis, 3.5% develop sclerosing cholangitis and less than one percent develop chronic hepatitis. The diagnosis of PSC may be suspected based on symptoms of chronic fatigue, anorexia, pruritus or jaundice, although many children may be asymptomatic. The diagnosis of PSC may be established through a combination of cholangiography and liver biopsy.¹⁷ Our patient could not have a liver biopsy done due to deranged INR and cholangiography was far reached.

Presently, there is no permanent medical cure for UC. The general goals of treatment in children are to control symptoms of the disease with minimal adverse effects of the medicines used and to achieve normal functioning of the patient. The intensity of treatment is dependent on the severity of the disease. Patients with moderate ulcerative colitis are usually treated on an outpatient basis. However our patient was admitted on account of severe anaemia, poor socio-economic background and dysfunctional home setting. After initial stabilization, he was started on pediatric medical regimen with low residue diet and prednisolone. Two days into commencement of therapy, blood in stool stopped, appetite improved and patient became more ambulatory. However compliance to medication was poor due to the dysfunctional home setting. This was also reported as affecting treatment success in the first reported case in Nigeria¹¹

Patient has been lost to follow-up despite repeated phone calls. This is commonly the case in patients with chronic illnesses and even worse in children who have to rely on adult caregiver. This has been reported in Nigerian adults^{9,10} with UC and in the Nigerian child with UC.¹¹ Late presentation to hospital is a major problem in sub-Saharan Africa. Patients patronize alternative medical practitioners as was the case in our patient before presenting to the hospital as a last resort. Delay or failure of diagnosis of IBD may also result from lack of awareness, denial of the presence of IBD by our physicians and limited facilities for most of the key investigations in our environment.¹⁸⁻²⁰ Paediatric endoscopy only started recently in Nigeria.^{19,20} Perhaps with increasing use of endoscopic diagnostic facilities, more cases of IBD in children will be discovered.

Conclusion

Ulcerative colitis is reported in a sub-Saharan African child. Though this appears rare, mis-diagnosis and misconception of its rarity in African children may have accounted for lack of reports on this inflammatory disease in Africa.

Conflict of interest: None

Funding: None

Reference

1. Mendeloff AI, Calkins BM. The epidemiology of idiopathic inflammatory bowel disease. In Kirsner JB, Shorter RG, eds. *Inflammatory Bowel Disease. Philadelphia: Lea &Febiger, 1988: 3-34.*
2. Benchimol EI, Fortinsky KJ, Gozdya P, Meta Van den Heuvel, Johan Van Limbergen, Griffiths AM. Epidemiology of pediatric inflammatory bowel disease: a systematic review of international trends. *Inflamm Bowel Dis 2011; 17:423-439.*
3. Chong SK, Blackshaw AJ, Morson BC, Williams CB, Walker-Smith JA. Prospective study of colitis in infancy and early childhood. *J PediatrGastroenterolNutr1986; 5(3): 352-358.*
4. Gryboski JD. Ulcerative colitis in children 10 years old or younger. *J PediatrGastroenterolNutr1993; 17: 24-31.*

5. Mamula P, Telega GW, Markowitz JE, Brown KA, Russo PA, Piccoli DA et al. Inflammatory bowel disease in children 5 years of age and younger. *Am J Gastroenterol* 2002 Aug; 97(8): 2005–2010
6. Langholz E, Munkholm P, Krasilnikoff PA, Binder V. Inflammatory bowel diseases with onset in childhood. Clinical features, morbidity, and mortality in a regional cohort. *Scand J Gastroenterol* 1997 Feb; 32(2): 139–147.
7. Van Limbergen J, Russell RK, Drummond HE, Aldhous MC, Round NK, Nimmo ER et al. Definition of phenotypic characteristics of childhood-onset inflammatory bowel disease. *Gastroenterol* 2008 Oct; 135(4): 1114-22
8. Fiocchi C. Inflammatory bowel disease: etiology and pathogenesis. *Gastroenterol* 1998; 115: 182–205.
9. Ukwenya AY, Ahmed A, Odigie VI, Mohammed A. Inflammatory bowel disease in Nigerians: Still a rare diagnosis? *Annals of Afr Med* 2011; 10(2):175-179
10. Alatishe OI, Otegbayo JA, Nwosu MN, Lawal OO, Ola SO, Anyanwu NC et al. Characteristics of inflammatory bowel disease in three tertiary health centres in sothern Nigeria. *West Afr J Med*. 2012 Jan-Mar; 31(1):28-31
11. Senbanjo IO, Oshikoya KA, Onyekwere CA, Abdulkareem FB, Njokanma OF. Ulcerative colitis in a Nigerian girl: A case report. *BMC Research Notes* 2012 5:564.
12. Nkrumah KN. Inflammatory Bowel Disease at the Korle Bu Teaching Hospital, Accra. *Ghana Med J*. 2008 Mar; 42(1):38-41
13. Segal I, Tim LO, Hamilton DG, Walker AR. The rarity of ulcerative colitis in South African blacks. *Am J Gastroenterol*. 1980 Oct; 74 (4):332-336
14. Billingham JR, Welchman JM. Idiopathic ulcerative colitis in African: a report of four cases. *Brit.med.J*. 1966;1:211-213
15. Sandhu Bk, Fell JME, Beattle RM, Mitton SG. BSPGHAN Guidelines for the management of inflammatory bowel disease in children in the United Kingdom. 2008 Oct pp 1-36.
16. Hyams J, Markowitz J, Treem W. Characterization of hepatic abnormalities in children with inflammatory bowel disease. *Inflamm Bowel Dis* 1995; 1: 27.
17. Roberts EA. Primary sclerosing cholangitis in children. *J GastroenterolHepatol* 1999; 14: 588–593.
18. Ekwunife CN, Nweke IG, Achusi IB, Ekwunife CU. Ulcerative colitis prone to delayed diagnosis in a Nigerian population: Case series. *Ann Med Health Sci Res*. 2015 Jul-Aug; 5(4):311-313
19. Ngim OE, Ikobah JM, Ukpabio I, Bassey GE, Ekanem EE. Paediatric Endoscopy in Calabar, an emerging trend-Challenges and prospects: A report of two cases. *IOSR J. Dental and Medical Sciences*. 2014 ;13(1):28-30
20. Ikobah JM, Ngim OE, Adeniyi F, Ekanem EE, Abiodun P. Paediatric Endoscopy in Nigeria-humble beginning. *Nig J Paed*. 2015; 42 (2):147-150