

Case Report

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Inflammatory bowel disease in Nigerians: Still a rare diagnosis?

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Abstract

Inflammatory bowel disease (IBD) has been thought to have a low incidence among subSaharan Africans mainly because of the sporadic cases reported from the continent in comparison with the larger numbers reported from North America and Western European countries. Is this difference based on real demographic susceptibilities or a reflection of lower level of healthcare delivery? Three cases of ulcerative colitis and one case of Crohn's disease diagnosed in a tertiary institution in northern Nigeria in the span of three years are reported. Their presentation coincided with the creation of the Gastrointestinal Surgery Unit of our hospital and with it the availability of endoscopic diagnostic procedures. All four patients were indigenous Nigerians. Our findings suggest that IBD may be more common in this part of the world than previously thought. With an increased awareness of the disease in our population, a greater utilization of modern medicine as against alternative medicine and with wider availability of diagnostic tools in our hospitals, it is our guess that more cases may be found in the future to dispel the belief that Africans are somewhat immune to this affliction.

Keywords: Inflammatory bowel disease, awareness, diagnosis, Nigerians

Résumé

Inflammatoire maladie de l'intestin (MII) a été considérés comme ayant une faible incidence chez les africains subsaharienne principalement en raison des cas sporadiques signalés depuis le continent en comparaison avec les plus grands nombres rapportés du Nord Pays d'Amérique et d'Europe occidentale. Cette différence repose sur les susceptibilités réel démographiques ou une section refl de niveau inférieur de la prestation des soins de santé ? Trois cas de colite ulcéreuse et un cas de maladie de Crohn diagnostiquée chez un établissement de soins tertiaires dans le nord du Nigéria en l'espace de trois ans sont signalés. Leur présentation a coïncidé avec la création de l'unité de chirurgie gastro-intestinales de notre hôpital et avec elle la disponibilité de diagnostic endoscopique procédures. Tous les quatre patients étaient nigériens autochtones. Nos constatations suggèrent que l'IBD peut être plus commun dans ce partie du monde qu'on ne le pensait auparavant. Avec une prise de conscience accrue de la maladie dans notre population, une plus grande l'utilisation de la médecine moderne à l'encontre de médecine alternative et avec la plus grande disponibilité des outils de diagnostic dans notre hôpitaux, c'est notre deviner qu'on peuvent trouver plus de cas à l'avenir pour dissiper la conviction que les africains sont quelque peu immunitaire à cette affl iction.

Mots clés: Maladies inflammatoires de l'intestin, de sensibilisation, de diagnostic, nigériens

Introduction

The inflammatory bowel disease (IBD) is characterized by nonspecific chronic relapsing

inflammation of the intestine and extra-intestinal manifestations of unknown aetiology and partly understood pathogenesis. Two clinical forms are recognized, ulcerative colitis (UC) and Crohn's

disease (CD). However, 10% of patients with colitis may initially be indeterminate, most of whom have been observed to progress to full-blown UC.^[1,2]

Earlier reports from sub-Saharan Africa indicated the rarity of IBD among black Africans and from Nigeria, presence of the disease has been documented from reports of isolated cases.^[3-6] At Ahmadu Bello University Teaching Hospital, Zaria, only one diagnosis had been reported in the literature in over four decades of existence^[7] but within a three-year period after opening of the Gastrointestinal Surgery Unit; three cases of UC and one case of CD have been confirmed. We present these patients to illustrate some of the challenges of managing this disease in our environment.

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Case Reports

Case 1

A 28-year old Nigerian man presented to our surgical outpatient with a 10 year history of bloody diarrhea, stool frequency of 5 to 6 times daily, tenesmus, lower abdominal pain and weight loss. He had sought treatment in several private and public hospitals to no avail. He underwent HIV screening and stool tests on numerous occasions, the results of which were negative for HIV and there was absence of pathogens in his stools. He had no family history of chronic diarrhea. Physical examination findings were unremarkable. He had normal haemogram, electrolytes and urea, serum protein, and liver function tests. Repeated stool examinations did not isolate pathogenic microorganisms. Barium enema showed loss of haustrations, shortening, straightening, luminal narrowing and smoothed mucosa from rectum to transverse colon. Colonoscopy showed mucosal congestion and edema, scattered bleeding spots and ulceration from rectum to midascending colon [Figure 1]. Histopathologic features were consistent with UC. He was placed on prednisolone and sulfasalazine, with reduction of stool frequency to twice or less daily. He is yet to report for follow up colonoscopy 20 months after diagnosis, having relocated to Abuja, 250 kilometres away.

Case 2

A 27 year old Nigerian man presented with a 5 year history of bloodstained mucoid diarrhea (frequency, up to 10 times daily), colicky hypogastric pain, tenesmus, weight loss and body weakness. Several times he had sought orthodox and alternative medical solutions for his symptoms without improvement. He had no family history of similar symptoms and had not been previously hospitalized.

There was no remarkable finding on clinical examination except bloodstained loose feces. Stool examination did not isolate any pathogen and HIV screening tests were negative. He had iron deficiency anemia, mild leukocytosis, mild hypokalemia and



Figure 1: Colonoscopic view of the lower and upper rectum, sigmoid and descending colon of Case I



Figure 2(a): Barium enema of Case II



Figure 2(b): Histopathology of Case II: lymphoplasmic infiltration of mucosa with pseudopolyp formation in UC

normal serum proteins. Barium enema showed loss of colonic haustrations up to transverse colon, luminal narrowing, shortening of transverse colon and smoothening of mucosal margin [Figure 2a]. Sigmoidoscopy revealed edematous hemorrhagic rectal mucosa, pseudopolyps, copious blood-stained mucous discharge and contact bleeding. Biopsy confirmed chronic inflammation with lymphoplasmic infiltration and pseudopolyps consistent with UC [Figure 2b]. He was immediately commenced on prednisolone and within 4 weeks, his symptoms had abated and stool frequency decreased to three times daily with no blood. He has been lost to follow-up.

Case 3

A 36 year old Nigerian woman presented to the surgical outpatient with diarrhea of 5 year duration (56 stool frequency per day), hematochezia, tenesmus, and lower abdominal pain. She is a mother of four children but her last four pregnancies ended in miscarriages following exacerbation of symptoms. She neither smoked cigarettes nor drank alcohol. On examination, she appeared well preserved with no remarkable findings. Barium enema showed granular mucosal appearance of the colorectum, loss of haustral markings, and shortening of segments giving the so called 'pipe stem' appearance. On colonoscopy, there were colorectal mucosal abnormalities characterized by edema, congestion, ulceration and bleeding most severe in the rectum and extending proximally. Biopsy result confirmed focally ulcerated epithelium with moderate to intense inflammation composed of lymphoplasmic cells, histiocytes, eosinophils, occasional neutrophils and moderate to severe dysplasia of the lining and glandular epithelia, consistent with IBD probably UC with dysplasia. Her symptoms have been controlled with prednisolone and sulfasalazine but she has so far refused consent for surgery recommended for the severe dysplasia.

Case 4

A 17 year old Nigerian woman presented with history of rectal bleeding of 12 years duration, accompanying two to three bowel motions daily. She had multiple blood transfusions and was yet to commence menstruation. On clinical examination, she had two anal ulcers, one fistula-in-ano and scattered mucosal elevations in the rectum. Her stool examination was normal. Other laboratory parameters were within normal range. Double-contrast barium enema showed cobblestone appearance. Colonoscopy showed coarse mucous membrane with edematous, congested, ulcerated and hemorrhagic polypoid lesions from the rectum to the descending colon. Biopsy confirmed nonspecific colitis with lymphoplasmic infiltration. A diagnosis of IBD, probably Crohn's colitis, was

made. The patient's family consented to limited surgery without stoma. She was therefore offered low anterior resection with colorectal anastomosis. At surgery, the rectum, left colon, and pelvic mesocolon were grossly thickened and vascular, with normal appearance of transverse colon, right colon and small bowel. Biopsy from the colon confirmed transmural chronic inflammation and fibrosis and the final diagnosis was CD. She came for one followup visit and has not been seen since then.

Discussion

Samuel Wilks of London is credited with the earliest documentations of UC in 1859 and 1875 while Crohn, Ginsberg and Oppenheimer published the landmark article on terminal ileitis in 1932, later renamed CD.^[8]

IBD has been more common in the highly developed countries of Northern Europe, Western Europe and North America, with prevalence of 30 to 200/10⁵ for UC, 1.2 to 106/10⁵ for CD and incidence of 0.5 to 24.5/10⁵ UC and 0.1 to 16/10⁵ CD.^[9] In developing countries, the extent of disease burden is largely unknown because of inadequate epidemiological data.^[10]

Current hypothesis indicates that the genesis of IBD may be polygenic and multifactorial, the chronic inflammation resulting from dysfunctional gut immune system in response to genetically determined failure to process certain commensal antigens. Whether the patient develops CD or UC is dependent on the defective genes and immune pathways involved; CD is mediated by Th1 and Th17 Tcells, while UC results from Th2 and NK T-cell pathway.^[11,12]

The role of the genetic system in IBD etiopathogenesis became clearer in the last decade with the discovery in 2001 of the CARD15/NOD2 (Caspase-Recruitment Domain 15/Nucleotide Oligomerization Domain 2) gene on chromosome 16q12, associated with increased risk of ileal CD. This locus has been named IBD1. Other loci implicated include, IBD2 (12q13 linked to UC), IBD3 (6p2123, UC, CD), IBD4 (14q11-12, CD), IBD5 (5q3133, CD), IBD6 (19p13q13, CD, UC), and IBD7, (1p36, CD, UC)^[12,13] Preceding the genetic linkage studies were observations that clearly implicated the genetic system in the etiology of IBD. A family history is present in 5 to 20% of cases, with first degree relative risk of 20 to 50 folds for CD and 10 to 20 for UC; twin studies showed higher monozygotic concordance while Jews are 2 to 9 times more likely to develop IBD than other ethnic neighbors.^[1,9]

A defective gene merely increases IBD risk and other factors contribute to the development of the disease. Two similar environmentally based hypotheses have been used to explain the gross geographic differences and trends in the incidence of IBD noticed worldwide. The hygiene hypothesis proposes that individuals born into less privileged environments and exposed to certain microbes early in life are at reduced risk of IBD, whereas failure of exposure to old friends predisposes to IBD, the old friends being microbes to which the intestinal immunogenetic systems have become conditioned as commensals during evolution.^[9,14]

Smoking has an interesting relationship with IBD, with incidence of CD increased in smokers (from thrombogenic and vasculitic effects) while nicotine seems to protect from UC.^[8,9,14] The risk of UC has also been shown to be lowered by previous appendectomy.^[14]

Each of the four patients presented had 5 years or more of symptoms before diagnosis, a problem common to other cases reported from sub-Saharan Africa.^[15,16] It is a rampant practice for our patients to patronize varieties of complementary and alternative medical practitioners for any chronic illness and come to the hospital only as the last option. Delay or failure of diagnosis may also result from lack of awareness and denial of the presence of IBD by our physicians. A patient presenting with chronic diarrhea, abdominal pain, and weight loss which are the hallmarks of uncomplicated IBD will be most likely investigated and managed for parasitic infestations such as *Entamoeba histolytica*, *Giardia*, *Schistosoma mansoni*, bacterial infections caused by *Shigella*, *Salmonella*, *Mycobacterium tuberculosis*, Lymphogranuloma, or HIV-related enteritis. In an environment endemic for tuberculosis, differentiating between Crohn's and tuberculous enteritis can be tasking.^[17,18] It is not unusual for our general practitioners to dispense with investigations and empirically treat diarrheal cases with antibiotics and amebicides. The relapsing nature of IBD and the benefits of antimicrobial therapy, though limited, give patients false sense of improvement and this may also contribute to diagnostic delay.

Our experience with diagnosis of IBD in these four patients coupled with the coincidence with the establishment of a dedicated Gastrointestinal Unit, with improved access to flexible gastrointestinal endoscopy and radiology, has drawn our attention to the possibility that the isolated cases reported from several centres may reflect underdiagnosis rather than the perceived rarity of the disease. This opinion is supported by reports of larger number of black Africans with IBD presenting at single centres

in South Africa and Ghana.^[15,16]

The presentation of UC in Cases 1 to 3 was typical and all had extensive colonic involvement consistent with several years of disease. Crohn's colitis was considered in Case 4 on account of significant anal involvement, discontinuous mucosal lesions, cobblestone appearance on radiologic and endoscopic findings and transmural disease histologically. Significant bleeding is not a common feature of CD and rectal involvement occurs in about 50%.^[2] On the contrary, bleeding, rectal involvement, restriction of inflammation to mucosa and submucosa with anal sparing are features of UC. Ideally, all patients suspected with IBD involving the colon should have endoscopic examination of upper gastrointestinal tract and small bowel (enteroclysis or capsule endoscopy) in search of lesions, the presence of which will favor a diagnosis of CD.

None of the four patients had extra-intestinal manifestations of IBD which may be present in as many instances as 10 to 30%.^[2,14] These lesions are seen in the skin (erythema nodosum and pyoderma gangrenosum), mouth (aphthous stomatitis), joints (peripheral arthralgia, arthritis and ankylosing spondylitis), eyes (iritis and uveitis), biliary tract (sclerosing cholangitis), kidneys (nephrotic syndrome) and pancreas (pancreatitis).

The risk of dysplasia coexisting with carcinoma at surgery in patients with UC is estimated at 16 to 43%, while progression to carcinoma may occur in up to half of the patients at 5 years.^[2,19] The risk of dysplasia and invasive cancer in patients with UC increases with the duration, anatomic extent and severity of colitis, family history of colorectal carcinoma and presence of sclerosing cholangitis. A young patient such as the one presented in Case 3 with UC and dysplasia should be a candidate for total proctocolectomy and ileal pouch anal anastomosis with anal mucosectomy. This procedure is curative. Pouchitis (7.33%), intestinal obstruction (27%) and anastomotic leak are known operative complications.^[20] Other complications for which a surgical operation may be indicated in UC include intractable symptoms, severe bleeding, toxic megacolon and perforation.

Follow-up has been a problem with these patients as it is with other chronic diseases in this part of the world. We have not been able to assess the UC patients for long-term drug responses and complications. Case 1 with over ten years of symptoms requires surveillance colonoscopy and we have been curious to know if limited surgery will be sufficient on the long run for Case 4.

Conclusion

The difficult routes to diagnosis encountered by these four patients indicate that underdiagnosis contributes to the 'rarity' of IBD in our population. A high index of suspicion coupled with availability of gastrointestinal endoscopic facilities in most parts of Nigeria will give true reflection of the prevalence.

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