INFLAMMATORY BOWEL DISEASE AT KORLE-BU TEACHING HOSPITAL: CASE REPORTS

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INTRODUCTION

Inflammatory bowel disease is regarded as uncommon or even rare in Sub-Saharan Africa. Four cases documented at The Korle Bu Teaching Hospital between June 1997 and May 1998 are described and the literature briefly reviewed.

Case 1

A forty eight year old male presented to the clinic with a three-week history of bloody and mucoid diarrhoea initially associated with fever, chills and vomiting. He had been treated with cotrimoxazole, metronidazole, and co-amoxiclav with temporary relief but relapsed about four days prior to admission. He experienced abdominal cramps before bowel movement but these settled later. The bloody mucoid stools became frankly bloody. He was passing stools up to ten times per day, had lost weight, was lethargic, and anorexic. He had self medicated with chloroquine. The patient gave a past history of a similar episode lasting for three weeks about thirty years ago. He denied travel outside the country, and had not been hospitalised before. He was admitted to a medical unit.

On admission, he looked unwell and thin (weight 45kg). The abdomen was uniformly tender and tympanitic the bowel sounds were active. There was no organomegaly. Moderate pitting pedal oedema was present; he had no finger clubbing. The rest of systemic examination was unremarkable. Haemoglobin was 11.9gm/dl; erythrocyte sedimentation rate (ESR): 80mm fall; Leucocyte count: 8 x 10^9 with normal differentials; stool examinations showed presence of red blood cells but no parasites, ova or cysts, and repeated stool cultures were negative. There were no malaria parasites in blood film; serological tests for HIV were negative; he was mildly hypokalaemic (serum potassium 2.7 meq/l). Straight abdominal X-ray did not reveal any significant dilatation or fluid levels.

Flexible sigmoidoscopy revealed a grossly inflamed rectal and distal sigmoid colon with friable and bleeding surface; there was no normal looking mucosa but no ulcers were seen. Mucosal biopsies were taken for histology.

The patient was started on Sulphasalazine tablets while the histology was pending. The diarrhoea gradually improved and after several days the stools became semi-formed and blood-free. The histopathology report was "Sections show severely inflamed colonic mucosal fragments with crypt distortion and moderate goblet cell depletion. A fragment of granulation tissue showing inflammation is present indicating mucosal ulceration. No granulomas seen. Appearances are consistent with ulcerative colitis".

The patient was discharged home after twenty-five days to continue on Sulphasalazine 500mg thrice daily, oral iron and folic acid. Two months later he had normal stool habits, his weight had increased to 54kg, haemoglobin was 13.5gm/dl, ESR 20 mm fall, and electrolytes normal. He has since been lost to follow up, after six months.

Case 2

An 18 year old male was referred from a surgical unit where he was being treated for rectal prolapse. He gave an 8 year history of persistent diarrhoea sometimes with abdominal cramps. Stool frequency was six to ten times a day. The stools were initially loose, bulky, and bloody but later became watery and without blood. The abdominal cramps preceding the diarrhoea were worse at night but there was no vomiting. His appetite was described as good. He had been taken to several clinics and hospitals and had usually been given intravenous fluids and various tablets. He lived with his parents and there was no significant family or past history of diarrhoea or any other illness. He stopped school at primary level.

The patient was severely stunted (height 137 cm) and underweight (18 kg). His hair texture was poor, had no axillary or pubic hair; his external genitalia were infantile but his body proportion was normal. He had normal intellect. Abdominal examination revealed diffuse mild tenderness but normal bowel sounds. He had an easily reducible rectal prolapse

on rectal examination the faeces in the rectum was watery but not bloody or mucoid.

Laboratory investigations on admission were as follow: haemoglobin was 11gm/dl; leucocyte count was normal; erythrocyte sedimentation rate 15 mm fall/hr; serum amylase moderately elevated (500u/l); a repeat haemoglobin a month later was reported as 8.1gm/dl. Stool examination revealed leucocytes but no parasites, ova or cysts; stool occult blood was negative so was stool culture. Liver function tests and basic renal function tests including electrolytes were normal. Upper abdominal ultrasound, barium meal and follow through were normal. Barium enema was not entirely satisfactory but showed featureless non-haustrated transverse, descending and sigmoid colon suggestive of chronic ulcerative colitis.

Upper gastrointestinal endoscopy was normal but limited colonoscopy revealed several mucosal ulcers, inflamed and friable mucosa, rigid tube-like descending and sigmoid colon consistent with ulcerative colitis. Multiple biopsies were taken for histology, which was reported as: Sections of colonic biopsies show ulcer slough in association with a dense chronic inflammatory infiltrate with crypt distortion and goblet cell depletion. There is no evidence of cryptitis. The features are those of a non-specific ulcerating inflammatory bowel disease.

The patient was started on sulphasalazine but he was admitted to the emergency department a few days later with upper abdominal pain the profuse diarrhoea and died after a few days. A post mortem report described an extremely emaciated body, dehydrated and pale. The small intestine was grossly normal; the large intestine contained scant fluid mucoid faecal matter. The entire extent of the colonic nucosa showed nodular elevations with ulcerated foci and a covering of fibrinoid exudate. Mesenteric lymph nodes were enlarged, discrete and firm. Changes consistent with ulcerative colitis, severe dehydration and anaemia.

Case 3
This patient, 28 years old, was referred with a diagnosis of hepatitis. He had been passing mucoid, bloody diarrhoea and vomiting for about nine days. The diarrhoea, according to him, was sometimes as often as twenty times a day, vomiting up to fifteen times a day. He had taken anti-malarial medication, herbal preparations, cotrimoxazole and metronidazole without effect. He had lost weight from 60kg to 53 kg at the time of admission. He looked ill and

slightly icteric but febrile. There was diffuse abdominal tenderness but the liver and spleen were not enlarged; there was no peripheral lymphadenopathy. His initial haemoglobin on admission was 15.3 gm/dl; ESR 3 mm/hr; liver function tests were moderately abnormal i.e. total bilirubin 292 mcmol/l (6-8) direct bilirubin 251 mcmol/l (0 – 9) total protein: 50gm/l (60-86); albumin: 28gm/l (38-44); aspartate aminotransferase; 112u/l (0-12); alanine aminotransferase: 98u/l (0-12); alkaline phosphatase: 533 u/l (60-170). Stool examination showed abundant pus cells and red blood cells; blood cultures were negative. Hepatitis B surface antigen, HIV serology, and Widal serology were all negative. Repeat haemoglobin and ESR during admission were 12.1 gm/dl and 15 mm fall respectively.

The patient was initially treated as bacillary dysentery with cotrimoxazole but he continued to pass frequent mucoid bloody stools, accompanied by abdominal pains for over two weeks of admission; he also developed marked pitting pedal oedema. A flexible sigmoidoscopy was performed on the sixteenth day of admission. This revealed grossly inflamed and haemorrhagic mucosa with contact bleeding changes consistent with inflammatory bowel disease, probably ulcerative colitis. Multiple biopsies were taken for histology and the patient was started on sulphasalazine. After four days the abdominal pains and distention diminished and diarrhoea reduced to three times daily. The histological report suggested granulomatous colitis either Crohn's or abdominal Koch's.

The patient gradually improved, the stools became semi-formed and of normal frequency (2-3 times daily), the pedal oedema regressed. He was discharged home after nearly six weeks in hospital. Colonoscopy was performed one month later. The colonic mucosal inflammation had improved considerably; however the terminal ileum was not visualized. No cobblestone changes, linear ulcers or skip lesions were seen. Repeat multiple mucosal biopsies submitted for histology were reported as chronic inflammatory disease more in keeping with Crohn's colitis. The description was: 'colonic biopsies show crypt distortion and shortening, focal cryptitis but no crypt abscesses and mucin depletion in a moderately inflamed lamina propria with increased eosinophils'. A barium enema later did not show any abnormal changes in the colon or terminal ileum. At his last review six months later he was well and had normal stools, had gained back his weight (60kg), and the pedal oedema had gone. The liver function tests had improved although the

alkaline phosphatase was still markedly elevated (1479u/l).

Case 4

This is a 15 year old student who, since 1995, has had recurrent episodes of bloody diarrhoea sometimes six times a day. He had lower abdominal cramps relieved after bowel evacuation. Several stool examinations had sometimes revealed intestinal flagellates. Various antibiotics and metronidazole had been given on several occasions. He was again admitted in March 1998. His haemoglobin had progressively fallen, over the years, from 11 gm/dl to 5.6 gm/dl in June 1997. On the current admission, his haemoglobin was 6.8gm/dl and was transfused, bringing his haemoglobin to 8.6 gm/dl; ESR was 60mm/hr. A limited colonoscopy to mid-descending colon, revealed proctitis, poorly distending sigmoid and descending colon with granular mucosal surface but no ulcers, changes compatible with inflammatory bowel disease. Multiple biopsies were taken and the patient was started on sulphasalazine. His symptoms improved after about one week, and the patient was discharged after ten days. The histology report described changes as follows: multiple colonic biopsies show crypt distortion but no cryptitis or crypt abscesses in a patchily inflamed lamina distortion but no cryptitis or crypt abscesses in a patchcily inflamed lamina propria. Lymphoid aggregate and occasional granuloma is seen. Features are those of an inflammatory bowel disease and in keeping with Crohn's colitis. He now has normal bowel habit on maintenance dose of sulphasalazine, and haematinics. His current haemoglobin is 10gm/dl.

DISCUSSION

Two of the cases described had been symptomatic for several months and had multiple empirical treatments at various clinics. Although infective, amoebic and parasitic diarrhoeas are very common in our environment patients who continue to have frequent bloody or non-bloody diarrhoea and those whose stool microscopy reveals red cells as well as pus cells warrant further investigation with either sigmoidoscopy with biopsy or barium enema. Sigmoidoscopy with biopsy is preferable because histology may reveal changes of inflammatory bowel disease when gross appearance of the colonic mucosa may be normal. The presence of intestinal flagellates or ova may be a red-herring, as in case 4; it is unusual for these to cause severe and bloody diarrhoea. Equally patients who develop progressive anaemia, high ESR, or weight loss as a result of diarrhoea need further investigation. It is important to exclude post amoebic and bacillary and dys-

enteric and tuberculous colitis, the latter especially in granulomatous colitis. When available, polymerase chain reaction (PCR) may aid diagnosis of tuberculous colitis. These disorders were all considered during investigations.

Case 2 became severely stunted as a result of inflammatory disease starting before adolescence, his quality of life and that of his parents became disrupted as a result of his intractable diarrhoea. He died as a result of delayed diagnosis and management.

The haemoglobin levels of patients 1 and 3 were relatively high initially (11.9 gm/dl and 15.3 gm/dl respectively) despite passing very frequent bloody diarrhoea probably because of the acute onset, the short period of the diarrhoea and dehydration causing haemo-concerntration. Although anaemia and elevated ESR are important findings either or both may be normal. In one study from South Africa the only laboratory finding that correlated with the severity of inflammatory bowel disease was serum albumin level[3].

In patient 2, apart from the initial bloody diarrhoea, his subsequent attacks of diarrhoea were not frankly bloody perhaps as a result of chronic healing with fibrosis of the colonic ulcers. The report of the colonic biopsies was of non-specific ulcerating inflammatory bowel disease; however the gross post-mortem description in addition to the radiological and colonoscopic findings were compatible with chronic ulcerative colitis. The histology of the colon post-mortem has not been available yet.

The clinical presentation of case 3 was unusual by it's acute and severe onset with abnormal liver function tests; thus diagnosis of acute hepatitis and bacillary dysentery were made initially but he failed to respond to treatment. His response to sulphasalazine therapy was rapid and impressive. His alkaline phosphatase remained high; in fact rising. Abnormal liver function tests, including high alkaline phosphatase, may occur in inflammatory bowel disease as a non-specific reaction or as a result of hepatic lesions such as pericholangitis, sclerosing cholangitis, chronic active hepatitis. This patient has unfortunately defaulted.

Except in case 2 who died, the other patients responded favourably to sulphasalazine, which is available in Ghana. Newer drugs are available and widely used in many advanced countries. These include mesalazine, slow release mesalazine and osalazine. These are reputed to have fewer side

effects and are as effective as sulphasalazine. In severe and potentially fatal attacks, steroids are used to induce remission. Steroid and mesalazine retention enemas are also available.

Review of the literature suggests that inflammatory bowel disease is uncommon in sub-Saharan Africa. However, several reports have come from South Africa[1-4] and a few case reports from other parts of Africa. Seven and two cases of Crohn's disease were reported from Ethiopia and Dakar respectively[5;6]. Muguti[7] reported seven cases of ulcerative proctocolitis in black Zimbabweans over an eight year period. In another survey involving 75 mission hospitals in 24 sub-Saharan African countries, of 12,859 cases of bloody diarrhoea 1914 had typhoid but only 22 cases of inflammatory bowel disease was recorded; however it was pointed out that histological support was least available in West Africa[8].

It appears that severe complications or malignant change were uncommon in African patients. O'Keefe et al[1,2] reviewed 114 patients with ulcerative colitis and 82 patients with Crohn's disease in Cape Town, South Africa, between 1970 to 1979; there was one case of carcinoma of colon among the ulcerative colitis group and one Crohn's related death. Segal[4] also reported on 46 patients in a Soweto hospital, South Africa and noted that there was a relative lack of complications leading to surgery despite severe disease and extensive colonic involvement. There was a long delay between onset of symptoms and diagnosis. Kelly et al[9] reviewed the records of patients with ulcerative colitis attending St. Bartholomew's Hospital, London. Of the 166 patients, only four females were of African or Caribbean origin. Three of these had sclerosing cholangitis compared with four of 162 patients of European or Asian origin.

Patients with inflammatory bowel disease need long term follow up and regular endoscopic or radiological surveillance in order to detect any complications early. Most patients tend to default once they feel better. Clinicians must consider inflammatory bowel disease in cases of chronic or recurrent diarrhoea especially if bloody since effective management is available.

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