Original Article

Nonspecific colitis a forgotten entity in Central Sudan

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Abstract

Background: Nonspecific colitis is an inflammatory Bowel Disease (IBD) that runs a clinically benign course. Histopathologically it is characterized by superficial mucosal erosions with lymphocytic infiltration in the lamina propria with no granuloma.

Aim of this study: is to describe the clinical presentation of Non-Specific Colitis (NSC), and to evaluate its response to 5-aminosalicylic acid (5-ASA) in Sudanese patients.

Setting and Design: This study is a prospective cohort. A total of 26 patients presenting with bloody, mucoid diarrhoea, lower abdominal pain or colonic mass were included in the study.

Methods and Materials: A total of 26 patients presenting with bloody mucoid diarrhoea, lower abdominal pain with or without colonic mass were included in the study. All patients underwent colonoscopy. *Statistical analysis*: A descriptive statistical analysis was done using SPSS.

Results: 19 (%) of patients were males and 7 were females with male to female ratio of 3:1. The majority of patients 16 (61.6%) had bloody diarrhea while 18 (69.2%) opened their bowel 4-6 times a day. The majority 10 (38.8%) had rectum and sigmoid involvement. All patients showed a good response to 5-ASA.

Conclusions: This study showed that NSC has clinical features that are very much similar to mild UC with a rather good clinical response to oral 5-ASA.

Keywords: Nonspecific colitis, indeterminate colitis, 5-aminosaclyic acid.

The term indeterminate colitis (IC) is an interim, or preliminary descriptive term used by pathologists for cases of inflammatory bowel disease (IBD) in which a definite diagnosis of ulcerative colitis (UC) or Crohn's disease (CD) cannot be established. Most cases are due to fulminant ulcerative colitis, a condition in which the classic pathologic features of UC are often obscured and may overlap with CD[1]. The measurement of perinuclearantineutrophic cytoplasmic antibodies (P-ANCA) and anti-saccharomyces cervisiae (ASCA) antibodies has been suggested as a method to differentiate UC form CD [2,3].The sensitivity of these tests is only 40-60%, limiting their usefulness in IC[3].

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The aim of this study is to describe the clinical presentation of NSC, and to evaluate its response to 5-aminosalicylic acid (5-ASA) in Sudanese patients.

Methods

Thestudy was conducted in Gezira Centre for Gastrointestinal Endoscopy and Laparoscopic Surgery in Gezira State Central Sudan, in the period from 2006 to 2007. A total of 33 patients presenting with bloody mucoid diarrhoea, lower abdominal pain with or without colonic mass were included in the study. After explaining the purpose of the study, a written informed consent was obtained. A protocol including a detailed history, physical examination, full blood count, ultrasonography and stool examination was performed. All patients underwent colonoscopy using videocolonscope Olympus EVIS EXERA, CV-145 type. The biopsies were examined for histopathology. Colonic biopsies showing features of nonspecific colitis (NSC) were included in the study

whilethose showing features of UC on IC were excluded. NSC was defined as superficial mucosal erosion with lymphocytic cell infiltration to the lamina propria with no granulomatous features.

All patients received 5ASA (mesalaziane, Asacol, Becam) 800mg, three times per day for a period of 6 weeks. A follow up was done after 6 weeks for each patient including: colonoscopy, full blood count, and stool examination. Descriptive statistical analysis was done using SPSS version 10.

Results

The study consists of 26 patients. 19 (73.1%) were males and females were 7 (26.9%) with mean age 42.5 ±17.7 with age range between (13-80 years), as shown in table 1

Table (1) Age range distribution of the patients

Age range	Number N=26
11-20	01(3.8%)
21-30	05(19.2%)
31-40	10(38.5%)
41-50	04(15.4%)
51-60	01(3.8%)
>61	05(19.2%)

The mean (±SD)of the haemoglobin at presentation was 110 ±19.1gm/l range 9=13gm/dl. Platelet mean count was $457X10^6$ /L ± 12910^6 /L, stool examination revealed that 28/33 (84.8%) of patients had blood, while 5/26 (19.2%) had *Giardia* cysts.

Clinically, the majority of patients 18/26 (69.2 %), opened their bowel 4-6 times a day and 8/26 (30.8 %) opened their bowel at night. Sixteen (61.6 %) patients had blood in their stools; and six (23.0%) had lower abdominal pain. Fresh rectal bleeding and iliac fossa mass had the same frequency of two cases for each (7.7%) those masses were detected during ultrasonography, but they were not seen during colonoscopy.

The mean duration of symptoms was 22.5 ± 28.573 range 6 months -10 years.

Colonoscopy showed that the rectum and the sigmoid were involved in 10/26 (38.5%) of cases. Rectum alone was involved in 11.5% of cases (3/26), while six cases (23.0%) showed an equal involvement of rectum, ascending and transverse colon. The rest of the patients had sporadic lesions as shown in table 2.

Table (2) the presenting different level of lesion in the study subjects (N=26)

Level of lesion	Number (%)
Rect. and sigm	10(38.5%)
T-colon	03(11.5%)
Rectum and Ascend. colon	01(03.8%)
T- colon and Ascend. colon	03(11.5%)
Sigm.	01(03.8%)
Rect. &sigm. and T-colon	01(03.8%)
From rect. and Ascend. colon	01(03 .8%)
Rect, sigm & Ascend.colon	01(03.8%)
Rect, Ascend.and T-colon	01(03.8%)
Rect.	03(11.5%)
Splenic texture	01(03.8%)
Total	26(100%).

Rect.=Rectum, Sigm.= sigmoid

The histopathology had shown superficial mucosal erosions with lymphocytic cell infiltration, in the lamina propria with no granuloma or neoplasia seen (nonspecific colitis) Figure (3).

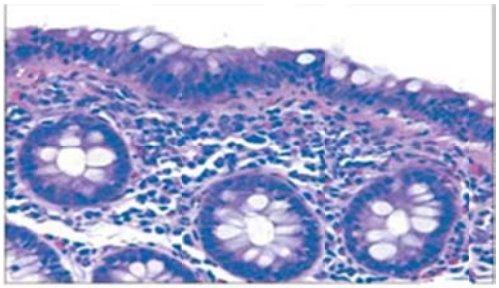

Figure (3): Shows chronic nonspecific inflammation in the lamina propria. No granulomas or crypt abscess seen

Outcome: Regarding the response to 5-aminosacylic acid, 26/26 (100%) patients had a good response with good mucosal healing. All patients showed decrease in the motions' frequency, the amount of blood and mucus in

stool. No patients developed hypersensitivity or other obvious toxicity from 5-ASA in this study.

Discussion

There are no randomized clinical trials of medical therapy for IC. Assumptions have been made that patients with IC have a clinical course similar to UC and therefore medical therapy follows the approach one would use for UC. Therapy include 5-ASA suppositories and or topical steroids for proctitis and proctosigmoiditis[4].

In 1978, Price introduced the term IC to refer to a subgroup of approximately 10-15% of IBS cases in which there was difficulty in distinguishing between UC and CD because the features of the typical severe UC were replaced by deep ulcer, relative rectal sparing and trasmural inflammation[5]. Typical CD fissuring type ulcerations were seen in 13% of cases of IC but the other histological feature of these IC cases reported so atypical that a diagnosis of CD could not be justified. The presence of scattered mononuclear inflammatory cells in the muscularis propria adjacent to ulceration was regarded as a non-specific response and is not a discriminating feature between UC and CD. This nonspecific response could be due to a disease in mucosa caused by an organism that was not identified on routine stool culture[6].

The evaluation of IC is done by histological examination of colonic specimens. This was shown to carry inter-observer variation[7]. Another modality of evaluations is the use of upper GIT endoscopy to link gastro-duodenitis caused by Helicobactorplori to both UC and CD especially in children [8-13].

There is no clear definition of nonspecific colitis (NSC) in the literature. It is a term that is similar to indeterminate colitis (IC), in that it is between ulcerative colitis (UC) and CD[1].

The study showed that males are outnumbering the males (19 versus 7) and their age is clustering around 31 to 40 years (38.5%). Sixteen (61.6%) of the study group presented with bloody mucoid diarrhoea or a lower abdominal pain and (30.8.6%) 6/26 open their bowel at the night. These features are consistent with UC presentation[15].

During colonoscopy the rectum and sigmoid colon were found to be invariably in involved and in (38.5%) have tinny superficial ulcers that turned to have features of NSC in a pattern similar to colonic involvement in UC[16, 15].

5-ASA is usually used to maintain the remission after controlling the acute attack of UC colitis [17].In this study it was used as oral medication to treat cases of NSC. All patients showed a good response.

In conclusion NSC showed clinical features that are very much similar to mild UC with a fairly good clinical response to oral 5-ASA. A search for causative organisms should be made and a course of an oral corticosteriods followed by oral 5-ASA may be tried for treatment.

References

1. Odze RD. Pathology of indeterminate colitis. J ClinGastroenterol 2004; 38 (5 Suppl): S36-4
2. Dubinsky MC, Ofman JJ, Urman M et al. Clinical utility of serodiagnostic testing in suspected pediatric inflammatory bowel disease. Am J Gastroenterol. 2001;96(3):758-65.
3. Legnani PE, Kornbluth A. Difficult differential diagnoses in IBD: ileitis and indeterminate colitis. SeminGastrointest Dis. 2001; 12:211–22.
4. Burakoff R. Indeterminate colitis: clinical spectrum of disease. J ClinGastroenterol 2004; 38 (5 Suppl 1):S41-3
5. Price AB. Overlap in the spectrum of non-specific inflammatory bowel disease – 'colitis indeterminate'. J ClinPathol 1978; 31: 567–77.
6. Wells AD, McMillan I, Price AB, et al. Natural history of indeterminate colitis. Br J Surg1991; 78:179–81.
7. Swan NC, Geoghegan JG, O'Donoghue DP, et al. Fulminant colitis in inflammatory bowel disease: detailed pathologic and clinical analysis. Dis Colon Rectum 1998; 41(12):1511–1515
8. Sasaki M, Okada K, Koyama S, et al. Ulcerative colitis complicated by gastroduodenal lesions. J Gastroenterol 1996; 31:585–9.
9. Kaufman SS, Vanderhoof JA, Young R, et al. Gastroenteric inflammation in children with ulcerative colitis. Am J Gastroenterol 1997; 92(7):1209–12.
10. Valdez R, Appelman HD, Bronner MP, et al. Diffuse duodenitis associated with ulcerative colitis. Am J SurgPathol 2000; 24:1407–13.
11. Tobin JM, Sinha B, Ramani P, et al. Upper gastrointestinal mucosal disease in pediatric Crohn's

disease and ulcerative colitis: a blinded, controlled study. J PediatrGastroenterolNutr 2001; 32:443–8.
12. Honma J, Mitomi H, Murakami K, et al. Nodular duodenitis involving CD8+ cell infiltration in patients with ulcerative colitis. Hepatogastroenterology 2001; 48(42):1604–10
13. Kundhal PS, Stormon MO, Zachos M, et al.Gastral antral biopsy in the differentiation of pediatric colitides. Am J Gastroenterol 2003; 98(3):557–61.
14. Durno CA, Sherman P, Williams T, et al. Magnetic resonance imaging to distinguish the type and severity of pediatric inflammatory bowel diseases. J PediatrGastroenterolNutr 2000;30:170–4.
15. Collins P, Rodes J. Ulcerative colitis, diagnosis and management. BMJ 2006; 333:340-3
16. Rubin GP, Hungin AP, Kelly PJ, et al. Inflammatory bowel disease: epidemiology and management in an English general practice population Aliment. PharmacotTher 2000;14:1553-9
17. Howarth LJ, Wiskin AE, Griffiths DM, et al. Outcome of childhood ulcerative colitis at 2 years. Acta Paediatr 2007; 96(12):1790-3.