

Case Series

Ulcerative Colitis in Burkina Faso: A Report of Six Cases and Literature Review

Rectocolite hémorragique au Burkina Faso : à propos de six cas et revue de la littérature

Guingané Alice Nanelin¹, Koefi Hassane 1er Jumeau², Compaoré Ines Nomawende², Elom Eklo Koukou², Sombié Roger².

HIGHLIGHTS OF THE STUDY

What is already known on this topic

Ulcerative colitis considered rare in black Africans. Few data are available form Burkina Faso concerning this chronic ulcerative inflammatory disease affecting the rectum and colon

What question this study addressed

Clinical pattern of ulcerative colitis in a black African population

What this study adds to our knowledge

Ulcerative colitis is rare in Burkina Faso; it is usually severe and associated with a significant deterioration in the quality of life. How this is relevant to practice, policy or further research. Further studies with more patients are needed.

- · Service d'hépatogastroentérologie, CHU Bogodogo, Burkina Faso
- · Service d'hépatogastroentérologie, CHU Yalgado Ouédraogo, Burkina Faso

Cooresponding author: Dr Alice Nanelin Guingané: aliceguingane@yahoo.fr, 01 BP 3479 Ouagadougou 01 Burkina Faso. Phone:00226 73838374

Keywords: Ulcerative colitis, diarrhoea, colonoscopy, anorectoscopy, Ouagadougou.

Mots clés : Rectocolite hémorragique, diarrhée, coloscopie, anorectoscopie, Ouagadougou.

ABSTRACT

Introduction. Ulcerative colitis (UC) is a chronic ulcerative inflammatory disease affecting the rectum and colon. It is the most common cryptogenic inflammatory bowel disease and it is considered rare in black Africans. The aim of our work was to describe the clinical pattern of ulcerative colitis at the University Hospital Center Yalgado Ouédraogo (CHU-YO). Patients and methods. This was a descriptive, retrospective and cross-sectional study from April 1, 2006 to May 31, 2019. All patients with a clinical profile suggestive of UC were included. Results. Six patients were included (4 women and 2 men); their mean age was 32.6 years. For five patients, the signs evolved for less than 5 years. Bloody diarrhoea or bloody mucoid diarrhoea associated with abdominal pain were the most common symptoms. Abdominal examination was normal in half of the cases. One patient had isolated rectal localisation while four patients had recto-colic involvement. According to Truelove and Witts score, four patients had severe flares. Histological analysis found association of lesions that was strongly suggestive of ulcerative colitis. The clinical course of patients during treatment was generally favourable. Conclusion. Although rare, ulcerative colitis is found in Burkina Faso. Diagnosis and management are difficult in our practice.

RÉSUMÉ

Introduction. La rectocolite hémorragique, considérée comme rare chez les noirs Africains, est une maladie inflammatoire ulcérative chronique affectant le rectum et le côlon. Il s'agit de la maladie inflammatoire intestinale cryptogénique (MICI) la plus fréquente. L'objectif de notre travail était de décrire la rectocolite hémorragique au Centre Hospitalier Universitaire Yalgado Ouédraogo (CHU-YO). Patients et méthodes. Il s'agissait d'une étude descriptive, rétrospective et transversale du 1er avril 2006 au 31 mai 2019. Tous les patients ayant un profil clinique évocateur de RCH ont été retenus. Résultats. Six patients ont été inclus (4 femmes et 2 hommes) ; leur âge moyen était de 32,6 ans. Dans plus cinq cas sur six, les signes avaient évolué depuis moins de 5 ans. Une diarrhée sanglante ou muco-sanglante, associée à des douleurs abdominales, était le signe le plus important. L'examen abdominal était normal dans la moitié des cas. Une localisation rectale isolée a été observée dans un cas et une atteinte recto-colique chez quatre patients. Se basant sur le score de Truelove et Witts d'évaluation de la sévérité de la poussée, les poussées étaient sévères chez quatre patients. L'examen histologique a retrouvé une association de lésions fortement évocatrice de colite ulcéreuse. Le suivi pendant le traitement a été généralement favorable. Conclusion. Bien que rare, la rectocolite hémorragique existe chez les noirs Africains du Burkina Faso. Son diagnostic et sa prise en charge sont difficiles dans notre pratique.



INTRODUCTION

Inflammatory bowel disease (IBD) results from chronic, intermittent or continuous inflammation of part of the digestive tract. Its causes are unknown. IBD typically includes ulcerative colitis, Crohn's disease and unclassified IBD [1]. Ulcerative colitis has been defined as a long-term disease affecting the colon in part or in whole, usually in its surface and almost always at its end, evolving by successive flare-ups interspersed with remissions and characterized by a muco-haemorrhagic syndrome during flares [2, 3]. It affects mostly the adolescent and the young adult. Both conditions evolve into a chronic state characterized by progressive flare-ups associated with complications.

Very few studies concerning UC were conducted in Africa, particularly in black Africans. People with UC have an increased risk of developing colorectal cancer (CRC). The global risk of developing CRC is 4.81 greater for UC as compare to the general population. The aim of this work was to study the diagnostic, therapeutic and prognostic aspects of UC at the Yalgado Ouédraogo University Hospital in Ouagadougou.

PATIENTS AND METHODS

This was a descriptive, retrospective and cross-sectional study from April 1, 2006 to May 31, 2019. It was conducted at the gastroenterology unit of the Yalgado Ouédraogo University Hospital. All patients were recruited from medical consultation records and admission records of patients treated for UC between April 1, 2006 and May 31, 2019 in the gastroenterology unit of the Yalgado Ouédraogo University Hospital (CHU-YO). The criteria of inclusion were as follows: a clinical picture suggestive of UC, organic lesions on colonoscopy and/or rectoscopy suggestive of UC, histologic findings of biopsies suggestive of UC.

The diagnosis of UC was made with the following clinical signs (bloody mucoid diarrhea or isolated rectorrhagia either acute, subacute or chronic), biological (decrease in haemoglobin levels and increase in erythrocyte sedimentation rate), parasitological stool analysis (absence of parasites or entero-pathogenic germs in repeated analyses of stools and endoscopic signs (rectal or rectocolic lesions of various degrees: erythema, congestion, mucosal friability or spontaneous haemorrhage, ulcerations; or the combination of mucosal abnormalities suggestive of UC).

Confirmations was made by histological analysis showing: erosions, ulcerations, oedema, haemorrhage, inflammatory infiltrate and lymphoid hyperplasia.

RESULTS

Six patients were included (04 females and 02 males). One patient had a high socio-economic level (observation 6), two had a medium socio-economic level (observation 3 and 5) and three patients had a low level or had no responsibility (1, 2 and 4). The mean age was 32.6 years and the age range from 24 to 39 years. Table 1 summarizes the socio-professional characteristics.

Table 1: Socio-professional characteristics									
Patients	Age /Sex	Level of Profession Ma study stat							
1	24 /F	Secondary school	Student	Single					
2	37/F	None	Housewife	Married					
3	39/M	University	Teacher	Married					
4	26/M	University	University Student	Single					
5	32/F	University	Sales agent	Single					
6	38/f	University	Medical doctor	Single					

Regarding the onset of the disease, in more than 3/4 of the cases, the signs had been evolving for less than 5 years. Specifically for 3 patients, duration was less than 1 year, two others between 1 and 5 years and finally only one had more than 5 years of evolution. The psychological factors that were found are summarized in Table 2.

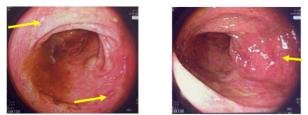
Table 2: summary of psychological factors in our patients							
	Patients						
	1	2	3	4	5	6	
Psycho-pathologic factors							
Learning difficulties	Х			Х			
Family constraints	Х						
Emotional shock	Х			Х		Х	
Constraints related to disease				Х			
Others							

The presenting complaints were: bloody diarrhea or bloody mucoid diarrhea in 04 cases sometimes associated with abdominal pain; weight loss in 01 case and mucoid diarrhea in 01 case.

On physical examination, one patient (number 3) had an altered general state, one patient (number 1) had an infectious syndrome and clinical anemia was found in one patient (number 2). Abdominal examination was normal in half of the cases. Only one patient (number 1) presented during her admission a toxi-infectious condition requiring emergency resuscitation measures. No patient had an extradigestive manifestation of UC.

Colonoscopy and rectosigmoidoscopy showed the topography of the lesions as follows: pancolitis in 1 case (observation 3); rectocolitis in 4 cases (patients 1, 2, 4 and 5); isolated rectal involvement in 1 case (observation 6).

The elementary lesions found with colonoscopy (observations 1, 2, 3, 4 and 5) and rectosigmoidoscopy (observation 6) were: a spontaneous hemorrhagic mucosa on touch in 05 cases; erosions in 01 cases; ulcerations in 02 cases; a pathological mucosa characterized by erythema, congestion, exudate and granular appearance in almost all cases.



Endoscopic appearance of ulcerative colitis: yellow arrows showing superficial erosions and ulcerations

Copyright © 2022: The Authors. This is an open access article published by HSD under the CC BY NC ND 4.0 license



Ulcerative colitis was associated with: peptic esophagitis in 1 case; hiatal hernia in 1 case; gastroesophageal reflux disease in 1 case; erythematous pangastropathy in 1 case; rectal sessile polyp in 1 case; double ulcer in 1 case and mycotic colitis in 1 case.

Histology found lesions strongly suggestive of ulcerative colitis with an inflammatory infiltrate in all our cases. Table 3 summarizes the anatomopathological lesions.

Anatomopathological lesions		2	3	4	5	6
Erosions and /or mucosal ulcers		×	×	×	×	×
Crypt abscess				\times		\times
Glandular atrophy						
Inflammatory infiltrates		\times	\times	\times	\times	\times
Vascular disorders		×	\times	×	\times	×

The diagnosis of ulcerative colitis in our study was made in front of several clinical, biological, endoscopic arguments, and more or less confirmed by histology with histological reports strongly suggestive of this condition. These enable us to classify our 06 patients according to the modified TRUELOVE and WITTS criteria (mild, moderate, severe activity). In four cases (1, 2, 3 and 5) the flare was severe, moderate in one case (observation 4) and mild in one case (observation 6).

Patient 1: six stools per day, temperature at 38 ° C, a heart rate at 92 beats / min, haemoglobin level, at 9.6 g / dl, ESR not available which corresponds to a severe flare-up.

Patient 2: seven stools per day, temperature at 37.5 $^{\circ}$ C, heart rate at 90 beats / min, haemoglobin level at 8.1g / dl corresponding to a severe flare.

Patient 3: seven to nine stools per day, temperature at 37.8, haemoglobin level at 9.5g / dl, heart rate at 88 beats / min, ESR not available which corresponds to a severe flare-up.

Patient 4: four to five stools per day, temperatures at 37.4 $^{\circ}$ C, heart rate at 90 beats / min, haemoglobin rate at 13.4 g / dl, ESR not available which corresponds to a moderate flare-up.

Patient 5: ten stools per day, temperature: $36.5 \circ C$, heart rate: 76 beats / min, haemoglobin level: 10.4g / dl, ESR not available which corresponds to a severe flare-up.

Patient 6: three stools per day, temperature at $37 \circ C$ a heart rate at 80 beats / min, ESR not available which corresponds to a mild flare.

Regarding treatment, three patients (cases 1, 4 and 6) received treatment with mesalazine or 5-ASA once diagnosed. Azathioprine was used in 3 patients (observations 3, 4 and 5) as soon as the diagnosis was known.

The combination of general and/or rectal corticotherapy permitted relapse amendment in five patients (observations 1, 2, 3, 4 and 5). One patient was managed with sulfasalazine (observation 2). Following the initial therapy, azathioprine was administered in three patients (observations 1, 3 and 5) as maintenance treatment, mesalazine (observation 6) in one patient, sulfasalazine (observation 2) in one patient and adalimumab which is an anti-TNF in one patient (observation 4). No complication requiring surgery was encountered in our study despite being a definitive treatment. The prizes of the drugs used for the treatment were:

- Mesalazine 1g suppository (Pentasa® 1g) box / 15: 41,45\$;
- Mesalazine 1g (Pentasa® 1g) tablet box / 60: 82,5\$;
- Azathioprine 500 mg (Imurel® 500mg) tablet box / 100: 41,8\$;
- Sulfasalazine 500 mg (Salazopyrine® 500 mg B) tablet box/100: 20,82\$
- Prednisolone 20 mg (Prednisone® 20 mg) tablet box /20: 4,3\$
- Adalimumab 40 mg (Himura® 40) box / 2 pen for injection: 699\$.

The mean follow-up period was 18.6 months, ranging from 4 months to 48 months. Remission was observed after initiation of treatment in five patients (patients 1, 2, 4, 5 and 6). One patient (patient 3) was refractory to treatment and was lost to follow-up.

As complications related to corticosteroid therapy, we had a case of diabetes (patient 4) and pedal oedema associated with many discomforts which led to the cessation of corticosteroids. Two patients (patients 1 and 3) required admission at least once during their follow-up. Figure 1 shows the endoscopic appearance

DISCUSSION

UC is a chronic inflammatory bowel disease of unknown aetiology; it is considered rare in Africa. Clinicians rarely think of it in the course of digestive diseases, which are usually considered as parasitic and bacterial infections with a clear predominance of intestinal functional disorders.

Very few studies have addressed UC in Africa, and particularly in black Africans. Since the first 4 cases published in 1964 by Billinghurst [4, 5] in Kampala, other cases in France in an Ivorian woman [6], Kenya [7], Senegal [8, 9, 10, 11], Nigeria [12], Ivory Coast [13], and Zimbabwe [14] have enriched the documentation on this subject. In Burkina Faso, the three studies on UC in 1999, 2010 and 2016 were reported respectively by Zanré [15], Bougouma [16] and Ouattara [17]. This small number of studies could be explained by the apparent scarcity of UC on one hand and by the lack of diagnostic tools and therapeutic difficulties in Burkina Faso on the other hand. Epidemiology of UC is poorly understood in Burkina Faso because epidemiological studies face many difficulties that prevent the recruitment of all patients, namely: the difficulty of diagnosis based on non-specific criteria and the difficulty for most patients to be able to carry out all the necessary investigations to establish the diagnosis because of their high cost and the absence of a social security system. Endoscopic examinations including colonoscopy and anorectoscopy cost between 30,4\$ and 60,79\$ in our context while the minimum wage is 46,63\$ in Burkina Faso. In addition, the number of cases is probably underestimated because of the similarity of its signs with infectious colitis. We can agree with the other authors that UC is rare in Africa, but its incidence is



sometimes underestimated due to coexistence with other intestinal diseases especially parasitic diseases.

The disease was observed in 04 women and 02 men, thus a sex ratio of 0.5. This female predominance is found in the majority of studies without being a risk factor. All our patients were young (< 40 years old), therefore confirming the classic notion of a peak around 30 years. In a study in Maghreb, Alyoune found that 62.8% of patients were aged between 20 and 42 years [18]. However, in some studies, such as those of Wrigth [19] and Molodecky [20] a second peak was found between 60 and 70 years. This disease affects young people, lasts a lifetime and is associated with deterioration in the quality of life [21, 22].

Aubry [8] in Dakar reported a duration of evolution of 1 to 4 years before diagnosis, which is close to our study, as well as Segal [23] which reported an average of 3 years. Corinne in France found a median of 2 months, and that the delays in diagnosis had been significantly reduced between 1988 and 2008 [24]. The interval between the onset of diagnostic signs is longer in developing countries than in developed countries during UC probably because of limited equipment and difficulties in accessing specialized care for the general population in developing countries. Self-medication, often linked to the confusion made by patients with other pathologies more frequent in our settings could also explain this diagnostic delay. Indeed, dysentery or haematochezia are often related to intestinal amoebiasis or hemorrhoidal disease and not to the possibility of UC especially, since this condition is unknown and considered rare in Africans.

In more than 3/4 of cases, the signs had been evolving for less than 5 years. People with UC have an increased risk of developing colorectal cancer (CRC). The global risk of developing CRC is 4.81 greater for UC as compare to the general population [25].

Four patients had a university level and Bougouma found nine patients (45%) with a university-level, ten (50%) with secondary school level and only one to primary (5%) [16]. In Burkina Faso, it would appear that high level of education is a factor influencing the need for specialized medical care [26]. In many cases, certain psychological disturbances more or less related to certain life events could be the basis of the onset or at least contribute to aggravating the flares of ulcerative colitis [16].

Regarding aetiopathogenic factors, intestinal amoebiasis is a cosmopolitan parasitosis, endemic in the intertropical area and sporadic in temperate countries. In Africa the simultaneous presence of UC and colonic amoebiasis has already been reported [27]. Inflammation and abrasion of the intestinal mucosa caused by UC may facilitate deep penetration of E. coli. histolytica from abscesses "in shirt buttons". This association is a source of diagnostic difficulty of UC in the tropics, both of which causes bloody diarrhea. The diagnosis of UC is often made after a well-conducted antiamoebic treatment. In our study, 4 patients (patients #1, 2, 4 and 5) had parasitological stool analysis done, they were all negative and only one (patient 4) received antiamoebic treatment during his episode of dysentery. In tropical areas, it is necessary to think of colonic amoebiasis for any bloody mucoid diarrhea. Parasitological stool analysis should be repeated in patients. Samples of mucous during rectoscopy with anatomopathological analysis of biopsies allow the diagnosis of amoebosis. When the course of treatment under antiamoebian is unfavorable, it is necessary to think of UC, be it revealed or aggravated by colonic amoebosis. Most publications point out that the major manifestations of UC in blacks are comparable to those in whites, but occurrence of extra-digestive complications in the former is more frequent. Blacks presents with advanced stages of the disease at the time of diagnosis [12]. The lesions described in our study are similar to those described in the literature at the different stages of UC. Our results are comparable to those of other authors [16, 15, 11].

One patient (patient 2) was pregnant at the time of diagnosis. Some studies have correlated pregnancy with inflammatory bowel disease. Therefore, the level of activity of the disease at the time of conception is also a major prognostic factor because it determines the risk of flare during pregnancy. If conception occurs in the active phase of the disease, it will remain active during pregnancy in 60 to 70% of patients, and 2/3 of them will worsen [28]. The risk of flare in pregnancy is estimated at 20 to 25% if IBD is not active at the time of conception, but at least 50% if active. The risk of relapse is greater in the 1st trimester, probably because of the spontaneous interruption of treatment by many patients [29].

Histology in our patients found lesions which association is strongly suggestive of ulcerative colitis. These results are consistent with those described in the literature [18, 30, 11]. The absence of amoeba in histological samples is an argument against amoebic colitis in our study, just as the absence of lymphoepitheloid granuloma is an argument against Crohn's disease. Some South African authors acknowledge that intestinal tuberculosis rarely simulates UC clinically and radiologically, even though colonic tuberculosis in its dysenteric form sometimes misleads the clinician [31]. Histology here remains the key to diagnosis.

The disease is related to an interaction between genetic, immunological and environmental factors, observed mainly in developed countries. Although the aetiology of UC remains uncertain, it has become clear that it results from an inappropriate immune response against the intestinal milieu in a context of genetic predisposition.

The chronic activation of innate and acquired immune system by an unbalanced intestinal milieu leads to excessive production of proinflammatory molecules leading to inflammation that alters the structure and function of the colonic mucosa. In addition, there are abnormalities of the epithelial barrier characterized by depletion of calciform cells and a modification of mucus ineffectively protecting the epithelium from bacterial colonisation [32].

Inflammatory parameters may be normal in the case of mild or moderate UC. The blood test may reveal thrombocytosis secondary to the inflammatory state, anaemia indicating a severe or chronic active disease and leucocytosis. Patients with severe impairment have elevated C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), associated with anaemia and hypoalbuminemia. However, neither CRP nor ESR are



specific enough to differentiate UC from another cause of colitis. The determination of fecal calprotectin is useful in the initial phase in case of suspicion of UC. It also plays a key role in follow-up during treatment, having a good correlation with the endoscopic activity of the disease.

The danger in diagnosing ulcerative colitis lies in the frequency of parasitic colitis and tuberculous infection (dysenteric form of colonic tuberculosis).

Also, looking for a parasitic and / or infectious aetiology is necessary in the first place. Histologic examination of rectal and colic biopsies makes it possible in most of cases to establish the diagnosis.

The management of our patients was based on mesalazine used in 3 patients (patients 1, 4 and 6), azathioprine administered in 4 patients (1, 3, 4 and 5), sulfasalazine in one patient (observation 2) and adalimumab in one patient (patient 4). General and/or rectal corticosteroid therapy was used in five of our patients (observation 1, 2, 3, 4 and 5). In most studies on IBD and on UC specifically, management is well codified according to the severity of the pathology. It is made of anti-inflammatories, corticosteroids, immunosuppressants and anti-TNF as appropriate [33, 34, 35, 36]. Remission in our study was achieved after initiation of treatment in 5 patients (observations 1, 2, 4, 5 and 6), one patient (observation 3) was refractory to treatment.

During follow-up, complications related to corticosteroid therapy were investigated. We noted in this regard a case of diabetes (observation 4) during treatment and pedal oedema associated with many discomforts which led to the cessation of the corticosteroid.

Aubry and Segal noted that follow-up of patients after the initiation of treatment is difficult, as patients return to consultation only during acute episodes [8, 37].

The high cost and the non-availability of certain medications in pharmacies is another major difficulty in the adequate care of patients who fall back on traditional therapy, most often easy to afford. While the minimum wage is 46,63 \$ in Burkina Faso, the various products are still very expensive or not available. The question of fiber diet is also a point of concern for patients in our context because of the eating habits of Burkinabe especially based on cereals.

No complication requiring surgery was observed in our study despite being a radical treatment. The mean followup period of our patients was 18.6 months, ranging from 4 months to 48 months. Remission is often observed in the literature [30], but the course can be punctuated by complications. Sobel in South Africa reported one case of malignant degeneration [31]. Aubry at the main hospital in Dakar, reported a case of severe rectal dysplasia. One of his patients died of severe intestinal haemorrhage on the 20th day of his admission [8]. Risk factors include the duration of the disease (usually, CRC is not found until seven years of illness), the extent of colonic involvement, the coexistence of sclerosing cholangitis, and a positive family history for CRC [25].

After 10 years of evolution, the condition becomes relatively quiescent. Therefore, while the immediate prognosis is good for our patients, there is still uncertainty about the long term prognosis. The risk of colorectal cancer is estimated between 1 and 5% depending on the authors and occurs more frequently in case of ulcerative colitis evolving more than 10 years and in case of pancolitis evolving more than 10 years [11]. This study has a main limitation which is the low number of cases taken into account, however this disease being rare in black Africans, we nevertheless considered it interesting to present them.

CONCLUSION

UC is rare in our settings. Its diagnosis and treatment are difficult. A thorough clinical examination of any bloody mucoid diarrhea that does not subside after a wellconducted or recurrent anti-amoebic treatment could allow clinicians to give themselves a better chance to diagnose this condition. Endoscopy and especially histology are essential for diagnosis. Reducing the costs associated with paraclinical explorations and medications could also improve the management of this disabling pathology in our context.

REFERENCES

- 1. Loftus EV. Clinical epidemiology of inflammatory bowel disease: Incidence, prevalence, and environmental influences. Gastroenterology mai 2004; 126(6):1504-17.
- 2. Kanti M, Patel-Lwangan S K A: Study of medical admissions to Mulago hospital Kampala. East Afr Med J 1971; 48 (2) 76-84.
- **3.** Mahid SS, Minor KS, Stromberg AJ et al. Active and passive smoking inchildhood is related to the development of inflammatory bowel disease. Inflamm Bowel Dis.avr 2007; 13(4):431-8.
- **4.** Bilinghurst J-R Ulcerative colitis in the African. Brit Med J 1964; 2:117-121.
- 5. Bilinghurst J-R, Welchman J-M Idiopathic ulcerative colitis in the African. One report of few cases.Brit Med J, 1966; 1:211-213.
- **6.** Carayon A, Réville JJ : Rectocolite hémorragique chez deux Africains, Bull Soc Med Afr Noire 1968 ; 13 : 484-489.
- 7. Aubry P, Klotz F, La rectocolite hémorragique ou colite ulcéreuse chez les sénégalais de race noire : à propos de 14 cas. Med Trop, 1984; 44 (3):269-278.
- 8. Awori N W, Rees P H, Causes of chronic diarrhea in Kenya and their relationship to ulcerative colitis. East Afr Med J, 1972; 49:604-613.
- **9.** Ball M, Ndiaye MF Rectocolite hémorragique associée à un pyoderma gangrenosum (à propos d'un cas observé à la clinique dermatologique du CHU de Dakar), Dakar Med 1982 ; 27 :72-76.
- **10.** Derrien J-P, Maydat L : Rectocolite chez l'Africain de race noire (à propos de 6 observations) Bull Soc Med Afr Noire langue 1970 ; 15 :282-288.
- **11.** Diouf M.L, Dia D, Thioubou A et al : Prévalence de la rectocolite hémorragique dans le centre d'endoscopie digestive de l'hôpital Aristide-Le-Dantec de Dakar. J. Afr. Hépatol. Gastroentérol. (2010) 4:97-102.
- **12.** Greenwood B M, Larsi E B: Ulcerative colitis in Africans. Brit Med J 1966; 1:13-20.
- **13.** Kissi H, Mahassadi A, Yao-Bathaix M, et al. La RCH en Côte-d'ivoire: réflexions à propos de 15 cas. Med Afr Noire 5209, 2005; 449–93.
- Kanti M, Patel-Lwangan S K A: Study of medical admissions to Mulago hospital Kampala. East Afr Med J 1971; 48 (2) 76-84.



- **15.** Zanré L. Les maladies inflammatoires cryptogénétiques de l'intestin à propos de 3cas de maladie de crohn et 3 cas de rectocolite hémorragie Thèse Med Ouagadougou 1999 :144p.
- 16. Bougouma A, Sombié R, Goumbri-Lompo O et al. La rectocolite hémorragique chez le noir africain. Analyse de 20 cas au Burkina Faso. J. Afr. Hépatol. Gastroentérol. (2010) 4:147-151.
- **17.** Ouattara A. Rectocolite hémorragique : à propos d'un cas observe dans le département de pédiatrie du centre hospitalier universitaire yalgado Ouédraogo (chu-yo) thèse Med Ouagadougou 2014 ; 72p.
- Alyoune M, Biadiallah M Ch. Rectocolite hémorragique à propos de 70 cas.Epidémiologie et formes cliniques. Med Maghreb 1992, 34 :5-8.
- Wright J-P, O'Keefe E. The epidemiology of inflammatory bowel disease in Cape Town 1980-1986.S Afr Med J 1986; 70(1):10-5.
- **20.** Molodecky NA, Soon IS, Rabi DM et al. Increasing incidence and prevalence of the inflammatory bowel diseases with time, based on systematic review. Gastroenterology 2012; 142: 46-54.
- **21.** Love JR, Irvine EJ, Fedorak RN. Quality of life in inflammatory bowel disease. J Clin Gastroenterol. 1992 Jan; 14(1):15–9.
- **22.** Büsch K, Sonnenberg A, Bansback N. Impact of inflammatory bowel disease on disability. Curr Gastroenterol Rep. 2014 Oct; 16(10):414.
- **23.** Shivananda S, Lennard-Jones J, Logan R et al. Incidence of inflammatory bowel disease across Europe: is there a difference between north and south? Results of the European Collaborative Study on Inflammatory Bowel Disease (EC-IBD). Gut 1996; 39:690-7.
- **24.** Cosnes J, Carbonnel F, Beaugerie L et al. Effects of appendicectomy on the course of ulcerative colitis. Gut. déc 2002;51(6):803-7.
- 25. Brondolo V K, Maillard M, Delarive J et al. 2010. available from: https://www.revmed.ch/revue-medicalesuisse/2010/revue-medicale-suisse-233/les-maladies-

inflammatoires-chroniques-de-l-intestin-survival-kit-pour-internistes-et-generalistes#tab=tab-authors

- **26.** Demographic and Health and Multiple Indicator Survey (edsbf-micsiv). Ouagadougou: National Institute of Statistics and Demography (INSD); 2010. Available from: https:// dhsprogram .com/ pubs/ pdf/ pr9/ pr9 .pdf [cited 2022 Feb 4].
- **27.** Molinier S, Imert P, Guisset P et al. Amibiase hépatique et rectocolite hémorragique: une association fortuite Med Trop 1995; 55 (2): 183 184.
- **28.** Ungaro R, Mehandru S, Allen PB et al. Ulcerative colitis. Lancet 2017; 389:1756-70.
- **29.** Couve S, Seksik P : Maladies inflammatoires de l'intestin et procreation. Gastroenterol clin biol 2003; 27:318-625.
- **30.** Kalliolias GD, Ivashkiv LB. TNF biology, pathogenic mechanisms and emerging therapeutic strategies. Nat Rev Rheumatol. janv 2016;12(1):49-62.
- **31.** Sobel J, Schamoth L. Ulcerative colitis in South African Bantu. Gut 1970; 11: 760-763.
- **32.** Tysk C, Lindberg E, Jarnero G et al. Ulcerative colitis and Crohn's disease in an unselected population of monozygotic and dizygotic twins. A study of heritability and the influence of smoking. Gut1988; 29: 990-6.
- **33.** Carbonnel F. Prise en charge d'une rectocolite hémorragique sévère ou corticorésistante. Gastroenterol Clin Biol 2007; 31: 398-403.
- **34.** Derrien J-P, Maydat L : Rectocolite chez l'Africain de race noire (à propos de 6 observations) Bull Soc Med Afr Noire langue 1970 ; 15 :282-288.
- **35.** Monaco C, Nanchahal J, Taylor P et al Anti-TNF therapy: past, present and future. Int Immunol. janv 2015;27(1):55-62.
- **36.** Sahami S, Kooij IA, Meijer SL et al. The Link between the Appendix and Ulcerative Colitis: Clinical Relevance and Potential Immunological Mechanisms. Am J Gastroenterol. févr 2016;111(2):163-9.
- **37.** Segal I: Ulcerative colitis in a developing country of Africa: the Baragwanath experience of first 46 patients. Int J Colorect Dis 1988; 222-225.

