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Ulcerative colitis complicated by colon cancer in a young adult

Mabika B[1*], Nguizi Ogoula Sidonie S[1], Obame Ngwa C[2], Obone Mezui L[2], Eyi Nguema A[2].
Département d'Anatomie-Pathologie. Université des Sciences de la Santé
Département de Médecine Interne. Service de Gastro-entérologie. Centre Hospitalier de Libreville.

***Corresponding Author:**
Mabika B
Département d'Anatomie-Pathologie. Université des Sciences de la Santé

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Abstract

Ulcerative colitis is an ulcerative hemorrhagic disease of the colon rare in black Africa. It exclusively affects the mucosa and the submucosa. Its etiology is unknown, the diagnosis is made on a set of clinical and histological arguments. Its evolution in spurts, can lead to various complications, one of the most serious of which is colorectal cancer.

The observation we report here is striking because it is a rare disease in Africa at this age, the course of which is complicated by the occurrence of invasive moderately differentiated glandular carcinoma.

Keywords: Ulcerative colitis - Glandular carcinoma - Black Africa.

INTRODUCTION

According to the literature, the maximum frequency of glandular carcinoma of the colon is between 60 and 79 years [1]. When colorectal cancer occurs in young people, look for ulcerative colitis or polyposis. The worldwide distribution of colorectal carcinomas shows high rates in developed countries while their occurrence is less in Africa and South America [2].

We report the case of a young patient who died of colon cancer with multiple metastases on ulcerative colitis (UC).

OBSERVATION

Mr. A.N.M. Gabonese, 21, single without children, was admitted to the Gastroenterology department of the Libreville Hospital Center on 06/14/07 with a picture of chronic diarrhea accompanied by a deterioration in general condition. The examination revealed the persistence for 5 months of a diarrhea made up of mucous faecal stools, mixed with blood and sometimes pure rectal bleeding. It was accompanied by diffuse abdominal pain, maximum in the iliac fossa and the left flank, intense or even disabling. There were profuse cold night-time sweats, and gradual weight loss despite the retention of appetite. In the antecedents, there were already similar diarrheal episodes since childhood, not labeled and progressing by flares (at a rate of two to three per year), then typhoid fever treated two months ago with ofloxacin (2cp / d for 10 d). The examination on arrival noted a deterioration of the general condition (loss of 6 kg in six months), an overall dehydration, bilateral centimetric painless mobile axillary lymphadenopathy. This clinical picture suggested inflammatory bowel disease or intestinal tuberculosis.

The hepatorenal, pancreatic, coprological tests and the Blood Formula Count (CBC) were normal, the tuberculin intradermal reaction (IDR) was negative. The Sedimentation Rate (ESR) was 33/65 mm, carcinoembryonic antigens (CEA) greater than 200 ng / ml, bilharzial serology positive at 1/320 for subtype H, related to a serological scar. The other serologies (amoebic, HIV, HBsAg, ACHVC) were negative.

Abdominal ultrasound revealed multinodular hepatomegaly in favor of a metastatic liver. The

abdominal scan also suspected an appearance of ulcerative colitis. Chest x-ray revealed rounded left hilar opacity consistent with lymphadenopathy. Colonoscopy described a mucous membrane, fragile congestive, bleeding on contact with the tube, with swollen, pseudo-polypoid, sometimes ulcerated areas. At 20 cm from the anal margin, there was a narrowing of the lumen.

The biopsies performed were fixed with 10% buffered formalin. All material collected was included. After embedding in paraffin, the blocks were cut into thin sections 4 µ thick. All sections were stained with eosin hematoxylin. Reading under a light microscope showed inflammatory colitis characterized by ulcerated lesions with inflammatory fibrin leukocyte and pseudo-polypoid clumps. The sometimes-dedifferentiated glands had a variable caliber. Cryptic micro-abscesses were identified in places. In some fragments, the lesion was carcinomatous, consisting of neoplastic glands whose cells exhibited cytonuclear atypia. Finally, we noted a fairly high mitotic activity and the absence of parasites (Figure 1, 2 and 3).

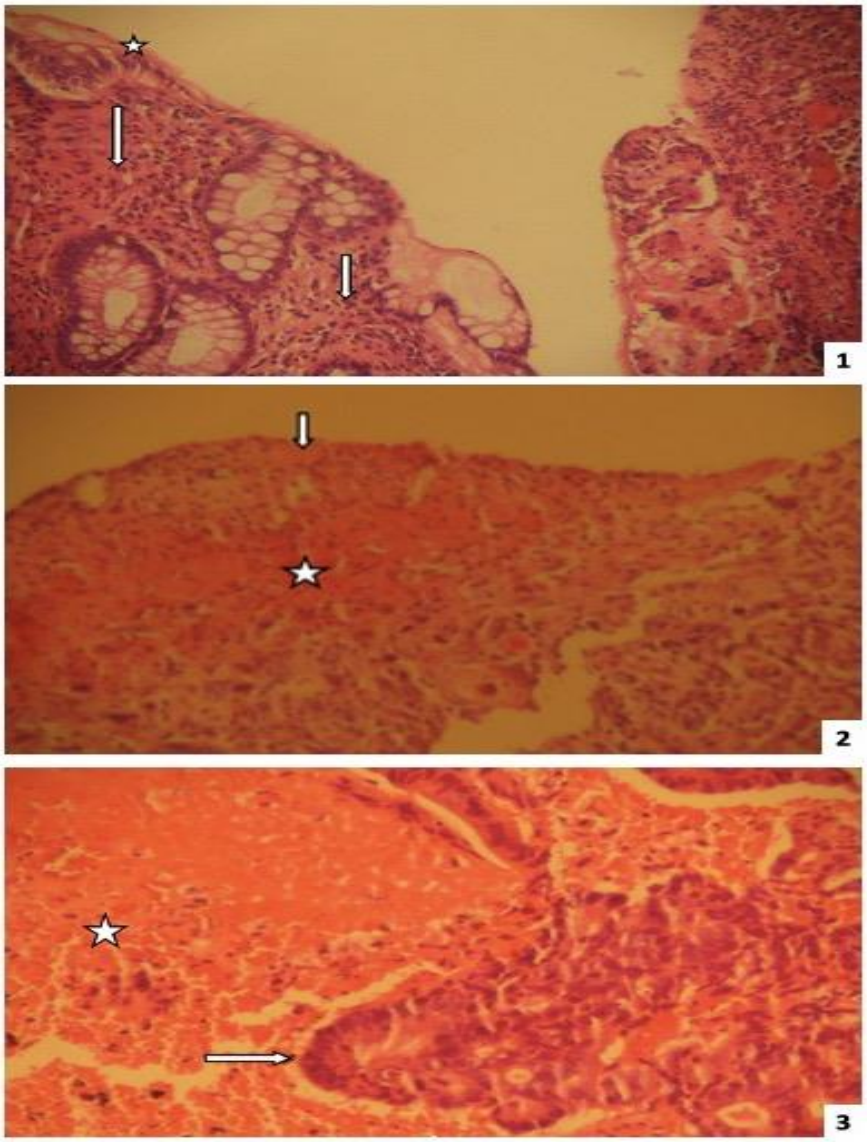

Figure: **1)-**Very inflammatory colonic mucosa, hyposecreting glands (star) and chorion in mononuclear inflammatory elements (arrow). X 300; HE. **2)-**Ulcerated colonic mucosa, abraded coating (arrow) and chorion rich in polymorphic inflammatory elements (star). X300; HE. **3)**- Epitheliomatous mass (arrow) neighboring with fibrinoleukocytic necrosis (star).

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This histological description demonstrated a glandular carcinoma on ulcerative colitis. The axillary lymph node biopsy concluded that a moderately to poorly differentiated carcinoma metastasized. We concluded that the diagnosis was glandular carcinoma, with stage IV metastases, according to the TNM classification, complicating ulcerative colitis.

The therapeutic management was mainly symptomatic (analgesics, intestinal dressings, rehydration and food intake rich in calories and protein). The evolution was mainly marked by the persistence of abdominal pain. One month after discharge from hospital, the patient died in an array of severe generalized pain, with cachexia.

DISCUSSION

The neoplastic degeneration that can complicate UC has been established for several decades. The latency between the discovery of UC and neoplastic transformation varies from one study to another, probably due to the risk factors involved and a recruitment bias [3]. UC occurs at all ages, frequently in young adults between the ages of 15 and 35, with an upsurge in the 5th decade [4]. In the study by Kissi [5] carried out in black Africa, the average age of patients was 37 years, with extremes ranging from 23 to 58 years. Before 8 to 10 years of development of UC, the risk of developing CRC is not higher than in the general population [6]. In a recent meta-analysis, this risk was estimated at 2% after 10 years of disease progression [7]. So, the CRC on UC would appear on average around 45-50 years, because of the age of the patient at the discovery of the disease, and the latency before the neoplastic transformation. In our case, however, the patient was only 21 years old. If the etiology of UC still remains unknown, the very early appearance of colon cancer on UC can be explained here on the one hand by the various predictive factors of cancer (age of colitis, colonic extent of colitis). and on the other hand, by the absence of protective factors (chronic treatment with 5-ASA, aspirin, NSAIDs, corticosteroids, tobacco consumption), which could considerably delay the onset of colon cancer.

Clinically, the questioning did not highlight either the familial notion of cryptogenetic inflammatory disease of the colon, nor of a history of colon cancer, which may also be due not only to the very limited life expectancy in our patients. regions, but above all because of the extreme scarcity of diagnostic resources at CRC. Indeed, if UC appears in young adults, CRC appears even later [8].

The seniority of the UC is also a factor favoring the development of the CRC. Endoscopic exploration with biopsy of the colonic mucosa, which made it possible to establish the diagnosis of CRC on UC, was not carried out until the age of 21 years. Conversely, the history of the disease clearly reveals those digestive disorders, especially diarrhea, had already existed since childhood. The usual additional biological investigations (KAOP, stool culture, glycemia, retroviral, amoebic, bilharzian serologies) had not revealed any anomaly capable of explaining this symptomatology. Apart from the abdominal ultrasound which was normal, the other morphological explorations (barium enema, abdominal CT scan) had suspected UC which was confirmed by histological examination of the biopsy specimen. We can therefore imagine that this chronic enteritis from which our patient suffered for years, and which will certainly not have attracted the attention of the doctors who previously took charge of this patient, (hence the instantaneous and late diagnosis of complicated UC CRC), was most likely the clinical manifestation of his UC. In this logic, our patient had to present his UC for well over 10 years, duration of evolution long enough to be compatible with a high risk of CRC [9]**.** In the retrospective study by Goudet [10] on 86 patients, there was a clear predominance of 72% of men, against 28% of women. It also emerges that the average duration of evolution at the time of the surgical intervention for CRC was 19.20 years. These data are verified in our case where the patient is male and where the symptomatology that led to the diagnosis has evolved since childhood, therefore for around 20 years.

The extent of colitis is classified into 3 categories: rectosigmoiditis, left colitis, extensive colitis. The relative risk of cancer is all the higher as the colitis is more widespread: 1.7 in case of rectosigmoiditis, 2.8 in case of left colitis, 14.8 in case of pancolitis [7]. The colonoscopy performed in our patient could only explore the last 20 centimeters of the colon, because of the narrowing of the lumen of the colon secondary to the tumor. One can say a posteriori having regard to the very long evolution of the clinical manifestations, the severity of the functional signs, and the frequency of the crises, that the colonic attack was very probably extensive, from where the cancerous complication.

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The severity of colonic inflammation, the existence of inflammatory pseudo-polyp, non-tumor lesions and non-precursors of cancer, double the risk of CRC [11], even if the risk of cancer persists in the event of inactive or not very active colitis. On average, our patient had 2-3 seizures per year. Between these episodes, the relaxation of digestive signs was not perfect. It should be noted the difficulty in distinguishing in these manifestations, those due to UC, and those that could be attributed to the innumerable etiologies responsible for digestive disorders in our environment (such as infectious factors) and which our patient could not escape. Moreover, we do not know the impact of this very rich infectious pathology in our regions on the development of UC. In the analysis of rectal emissions, mucous and bloody matter appears, and at times pure rectal bleeding, proof of the fragility and intensity of the lesions of the mucous membrane of the colon. During the lower digestive endoscopy, a notable macroscopic change was observed with a fragile congestive mucosa bleeding on contact, then swollen areas, pseudo-polypoid sometimes ulcerated. Histologically, the classic appearance of UC was described with significant lesions, including inflammation. This severity of the lesions, which we also find, is one of the factors favoring colonic carcinology on UC.

CONCLUSION

UC (chronic inflammatory bowel disease) is rare in our regions. Of unknown etiology which does not allow neither to prevent it nor to treat it radically, it is endowed with multiple complications, the fear of which remains the neoplastic transformation, favored by the extent of the colitis, the severity of the inflammation, the duration of UC, the concept of familial colon cancer, and association with sclerosing cholangitis. As soon as it is diagnosed, colonoscopic monitoring is necessary beyond 8 years of evolution, becoming even more regular over time, despite treatment with 5ASA which would prevent cancerization.

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