Colon cancer in Crohn’s disease: A case report.

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Abstract

Introduction
There is recognized increased risk for colorectal cancer in patients with inflammatory bowel disease, particularly in long-standing and extensive ulcerative colitis. This paper reports a case of colon cancer in Crohn’s disease.

Case report
A 25-year-old male patient was admitted with the complaints of abdominal pain, hematochezia and weight loss. Patient has been submitted to a total colectomy due to bowel obstruction.

Conclusion
The majority of patients with who develop intestinal cancer have in common the following items: cancer installs many years after the first symptoms.

Introduction
Crohn first described colorectal cancer (CRC) in association with inflammatory bowel disease (IBD) in 1925 and colorectal cancer still accounts for 10%-15% of deaths in patients with IBD1. The dysplasia-cancer sequence is a useful concept but it is more complex in IBD-CRC.

There is not always a clear, stepwise transition from normal, through low and high grade dysplasia, to cancer. However, low grade dysplasia can clearly progress to more advanced lesions and there is varying evidence as to the size of this risk2. Due to the low incidence of this neoplasm, we present a case recently treated at this institution.

Case report
A 25 years old male patient, diagnosed as Crohn disease 15 years ago, presented with abdominal pain, hematochezia and weight loss. Patient was receiving a treatment protocol with infliximab 350mg every 8 weeks and azathioprine 50mg/day for 3 years. The patient denies previous other surgeries or any other comorbidities.

On admission, a patient’s general condition was regular, with normal weight rate.

Physical examination revealed a mildly tender right abdominal mass.

Patient has undergone a colonoscopy in other service three years before and it showed clearly hyperemia in distal ilium, and pancolitis with flat erosions recovered by fibrin. There were 2 sessile polyps on transverse colon, one measuring 20mm and the other 8mm. The histopathological exam showed a case of tubular adenomas.

Other complementary exams showed haemoglobin 10.1g/dl, hematocrit 30%, leukocytes 9430 cells/ml, PCR 235,6, CEA 1.1, negative P-ANCA.

After undergoing another colonoscopy (Figure 1), the result was vegetative, ulcerated and estenosant lesion in transverse colon and light enanthematic pancolitis.

The total abdomen computerized tomography was realized based on the results of the colonoscopy, and it showed a colon segmental parietal thickness in the hepatic flexure region and ascendant colon.

It also showed mucinous adenocarcinoma of ascendant colon. It also showed adenocarcinoma metastasis in regional lymph nodes. (1/83)

Immunohistochemistry exam showed negative immunoperoxidase to MLH1 and PMS2 repair enzyme markers, favouring the association to microsatellites instability phenotype.

Patient was discharged seven days after surgery and is being observed in ambulatory appointments without signs of recurrence disease.

Discussion
The risk of colorectal cancer is increased in patients with ulcerative colitis (UC) and Crohn’s disease (CD). A Swedish population-based study estimated that the risk of colorectal cancer increased in patients with ulcerative colitis by 11% compared with the general population. In patients with Crohn’s disease, the risk of colorectal cancer is approximately 20% higher than in the general population.

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cancer in IBD was 93. The trend over the last 40 years has generally been of a decline in incidence of colorectal cancer. It is not clear if a gender difference exists in the risk. In another population-based study, males had a 60% higher risk of colorectal cancer than females though the effect of sex was seen only after ten years of follow-up. These gender differences could be explained by differences in the extent of inflammation or patient behaviour leading to differences in medication or surveillance.

Weedon et al. reported colorectal cancer in 8 of 449 patients with Crohn’s disease, or about 1.2% (i.e., an estimated 20 times greater risk than a control). Similarly, Gyde et al. described an approximately 4-fold increased risk in patients with Crohn’s disease.

CRC associated with UC is most common in the rectum and sigmoid colon. In contrast, CRC associated with CD is evenly distributed between the right colon and rectosigmoid. Cancer always occurs in areas involved in chronic inflammation. Synchronous tumours are much more common in IBD than in sporadic CRC (12 versus 3 to 5 percent).

It is generally accepted that CRC in IBD is preceded by dysplasia. Thus, dysplastic epithelium may be a marker for coexisting malignancy, and provides the rationale for surveillance. Dysplastic areas are often difficult to recognize on endoscopy. They may appear as flat or only slightly elevated above the level of the mucosa. The criteria for dysplasia stress the uniform clonal nature of dysplastic changes, which affects all parts of the crypt and surface epithelium. In contrast, regenerative changes are usually most prominent at the bases of the crypts and show evidence of surface maturation.

The genetic alterations found in ulcerative colitis associated colorectal cancer involve many of the same targets found in sporadic colorectal tumours and include multiple sites of allelic deletion, microsatellite instabilities, and mutations of APC, p53, Ki-ras as well as MSH2 and other genes. The progression of dysplasia to carcinoma is generally accompanied by an accumulation of these mutations and the similarities in the biology of colorectal cancer associated with ulcerative colitis and sporadic colorectal cancer appear to outweigh their difference.

The aim of any screening or surveillance program must be to identify early lesions to enable treatment and prevention before the development of invasive cancer.

Conclusion:
Confirmed risk factors for IBD-CRC are duration, severity and extent of colitis and a family history of CRC. Evidence-based guidelines advise surveillance colonoscopy for patients with colitis 8 to 10 years after diagnosis, with the interval for further surveillance guided by risk factors other tools that might predict later cancer development in Crohn’s disease, employing molecular or genetically-based markers are still desperately needed and should be aggressively pursued.

Consent
Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

References:

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