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An Ultra-rare Hereditary Disease of 28-year Old Pregnant Patient with Malignant Alteration

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Abstract

Familial adenomatous polyposis (FAP) is an autosomal dominant disorder characterized by the development of hundreds to thousands of colorectal adenomatous polyps and the inevitable occurrence of colorectal carcinoma, if the colon is not removed. In a 28-year old patient, following diagnostic criteria, have been established more than 500 colorectal adenomas and family history of FAP.

Keywords: Colorectal; Carcinoma; Adenocarcinoma; Rectal carcinoma; Antibodies

Introduction

Having in the mind that the colorectal carcinoma is the most frequent carcinoma of the digestive system, and that often arises from adenoma by Marson's "adenoma-carcinoma sequence" theory, we have undertaken the following study: morphological, histochemical and immunohistochemical examinations of one hundred adenomas of various size and macroscopically pattern in FAP, complicated by rectal mucinous adenocarcinoma.

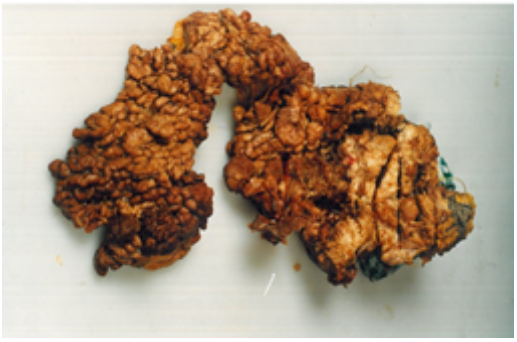

Figure 1: Hundreds of adenomas and rectal carcinoma.

Material and Methods

Total colectomy was induced by FAP, associated with rectal carcinoma. Numerous hundreds of tubulars, tubulo-villous and villous adenomas, size from 5 to 35 mm, as well as 10 biopsies of the cancerous rectal tissue, together with 10 regional lymph nodes, were fixed in 10% formaldehyde and processed in auto technicon. Paraffin sections of 2 microns were stained with; classic H&E method, histochemical PAS, HID-AB, pH2.5 methods as well as immunohistochemical ABC method, by using antibodies to; Ki-67 (mitotic activity), p53 suppressor activity and CEA (marker for colonic adenocarcinoma) [1-5].

Figure 2: Large size of villous adenomas – arrows.

Figure 3: Various size and villous structure of adenomas.

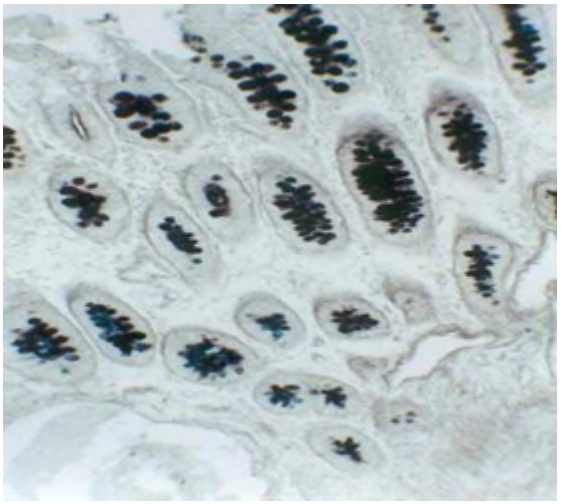

Figure 4: Normal colonic mucosa with normal secretio of sulfo–mucin.

Results

Most polyps were small, sessile and lobulated, "carpeting" the lining of the whole large bowel and numbering hundreds (Figures 1 and 2). Scattered larger sessile and pedunculated polyps were much less common, but the largest was surrounded by mucinous infiltrative rectal carcinoma (Figure 2). Histopathological, wide spectrum of size, structure and dysplasia of adenomas was obsreved (Figures 3-5). Malignant alteration of tubular adenoma with mucinous secretion was also seen (Figures 6 and 7). Strong mucinous secretion was found in both villous adenoma and mucinous adenocarcinoma (Figures 8 and 9).

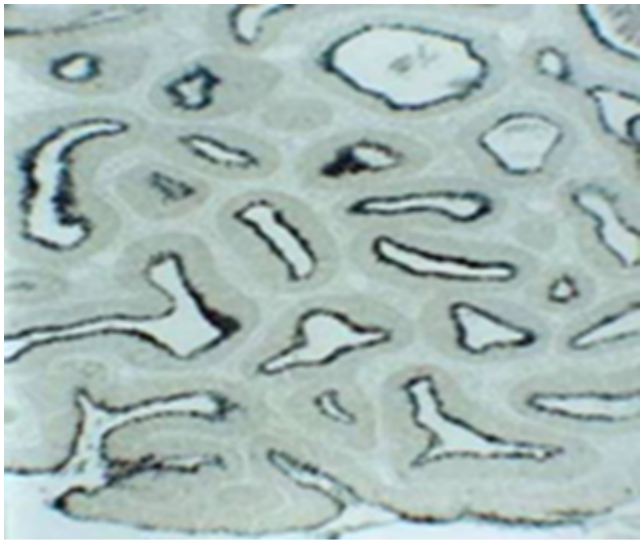

Figure 5: Adenoma tubulo-villosum cum dysplasio gr. II et cum hiposrcretio sulfo-mucin.

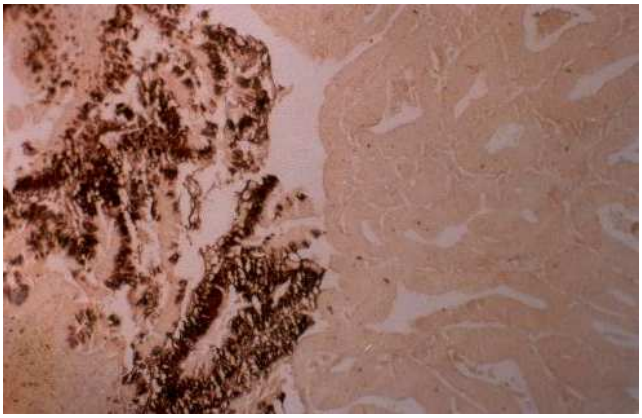

Figure 6: Adenoma-tubullare cum alteratio maligna et cum asecretio (arrow).

High expression of proliferative nuclear activity (Ki-67 antigen) was also observed (Figures 10 and 11). Strong p53 nuclear activity of the suppressor gene in adenocarcinoma has also been found (Figure 12) [5-7].

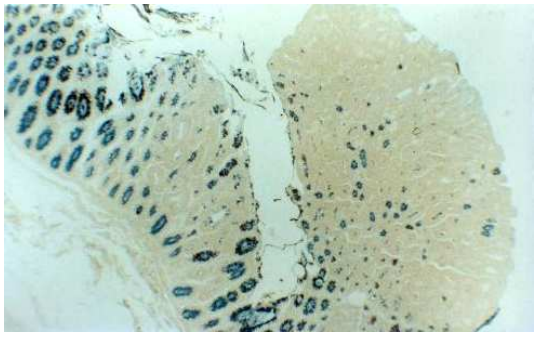

Figure 7: Micro adenoma collonis cum hypo-asecretio (arrow).

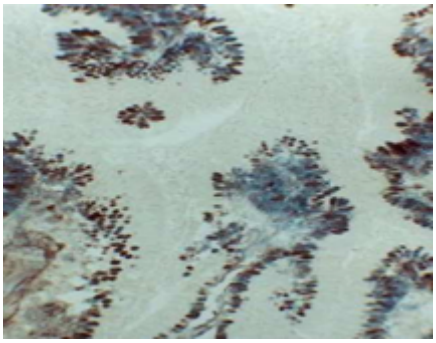

Figure 8: Adenoma-vilosum cum hypersecretion, sulfo- mucin.

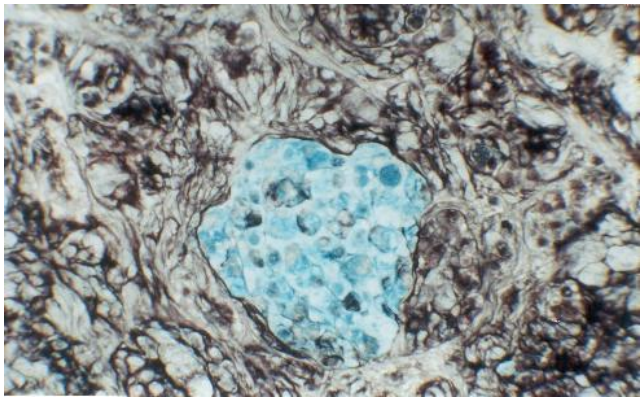

Figure 9: Adenocarcinoma mucinosum cum hypersecretion sialo-sulfo mucin.

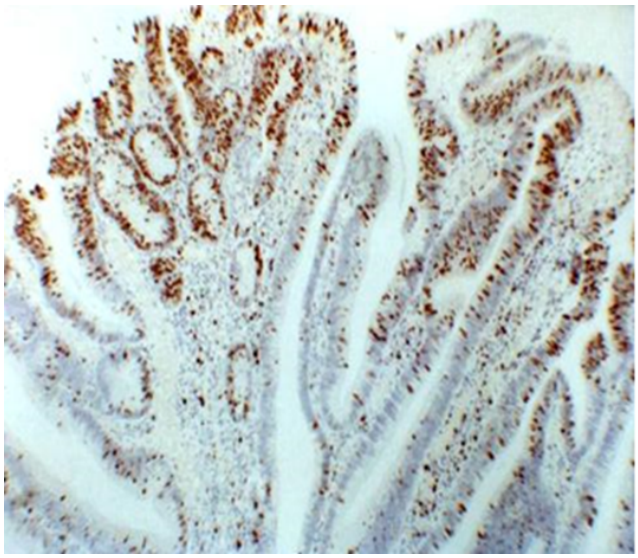

Figure 10: High mitotic superfitial Ki-67 nuclear activity.

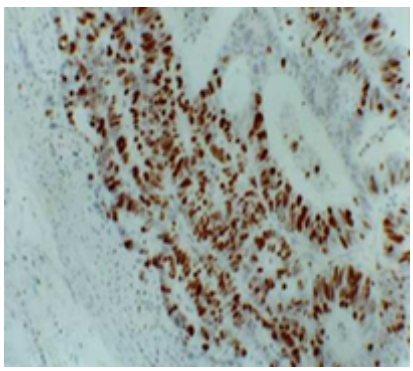

Figure 11: Diffuse mitotic activity in adenocarcinoma.

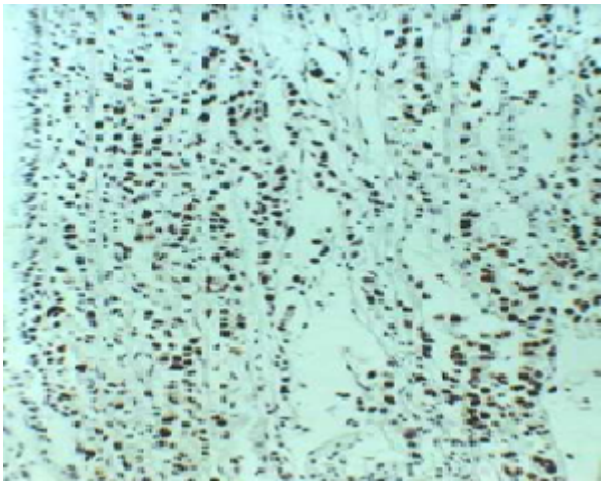

Figure 12: Rectal carcinoma with strong p53 activity.

Conclusion

We have confirmed the theory "adenoma-carcinoma sequence", showing that size, villous structure, severe dysplasia, as well as their number, represent the risk factors in colorectal carcinogenesis [7-9]. On the basis of the expression of normal HID and coexpression of small intestinal AB-mucin in both villous adenoma and surrounding mucinous adenocarcinoma, we have pointed out the direct connection between villous adenoma and mucinous colorectal adenocarcinoma. The discovery of the malignant alteration of the FAP in very young (28-years old) patient, could be explained by her pregnant state with decreased host immunity [10-14].

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