CASE REPORT

Collision adenocarcinoma – carcinoid tumor of the colon. Case report and review of the literature

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Abstract
Collision tumors are extremely rare and raise interesting problems related to histogenesis. In this paper we report a case of collision tumor of the colon and review the literature related to this subject.

Keywords: collision tumor, adenocarcinoma–carcinoid, colon.

Introduction
Carcinomas of the gastrointestinal tract are occasionally accompanied by neuroendocrine neoplasms.

These lesions have been roughly divided into four categories [1–5]: composite tumors; collision tumors; mixed tumors; amphicrine tumors.

In the composite tumors two types of tissue exist within the same tumor, being intermingled with each other in a similar proportion.

In the collision tumors the two elements are adjacent to one another without intermixture of individual cell types (“side by side” pattern).

Mixed tumors are further classified into carcinomas with interspersed endocrine cells and carcinoids with interspersed non-endocrine epithelial cells. Finally, in amphicrine tumors endocrine and non-endocrine features are present in the same cell.

It should be, however, mentioned that in some papers mixed and composite tumors are discussed under the same heading.

Among these groups collision tumors are extremely rare and raise interesting problems related to histogenesis. In this paper we report a case of collision tumor of the colon and review the literature related to this subject.

Patient and methods
A 34-year-old male presented with a tumor of the sigmoid colon, which apparently has invaded the posterior wall of the bladder.

A rectosigmoid resection was performed and the surgical specimen was submitted to the Pathology Department.

Several pieces of the tumor were fixed in 10% formalin, embedded in paraffin and cut at 5 µm.

The histological sections were stained with Hematoxylin–Eosin (HE), and with Grimelius argyrophil silver technique.

Immunohistochemistry was performed for neuron specific enolase (NSE) and chromogranin A (Dako reagents), using the manufacturer’s indications.

Results
The gross inspection of the specimen revealed an exophytic tumoral mass measuring 6/5/5 cm, involving the whole thickness of the colonic wall.

The microscopic examination of Hematoxylin–Eosin stained slides showed two different aspects. One was a moderately differentiated tubular adenocarcinoma, composed of closely packed glands lined by cuboidal epithelium with round or oval vesicular nuclei (Figure 1).

In the lower part of the adenocarcinomatous area, but completely separated of it by a conjunctive band, there was another component (Figure 2).

These cells showed argyrophilic on Grimelius technique (Figure 4), and were positive for neuron specific enolase (Figure 5) and chromogranin A (Figure 6).

This arrangement was diagnosed as a collision tumor of the colon, composed of an adenocarcinoma and a carcinoid tumor, as no intermingling was noted at the interface of the components. Both components invaded the intestinal wall, with serosal involvement, maintaining their individuality; there was no bladder tumoral infiltration.

Lymphatic permeation was present, but no venous invasion was observed; three regional lymph nodes presented adenocarcinoma metastases.
Discussions

Both collision [5–8] and composite tumors [9–16] are more frequent in the stomach. In the colon composite tumors were mentioned in several publications [7–23]. Collision tumors of the colon [23, 24] and esophagus [25] are, however, exceptional. Finally, adenocarcinoma and carcinoid tumors may appear in different segments of the gastrointestinal tract as separate, concurrent tumors [26–29].

The histogenesis of these “combined tumors” has not yet been definitely established. Corsi A and Bosman C [7] who have studied a collision type tumor suggest a double and independent origin of the two components.

Other authors believe, however, that both components of the tumor result from the multidirectional differentiation of a unique cell [4, 15, 30], although the identity of this cell remains a subject of dispute.

The case that we have studied showed two histological different and topographically separate components. In this context, the data of Fukui H et al. [3] may be relevant. The authors examined a tumor extending across the pyloric ring, the gastric portion of which revealed adenocarcinoma, while the duodenal portion showed argyrophilic neuroendocrine carcinoma. Genetic analysis of the p53 gene mutations suggested that the neuroendocrine carcinoma of the duodenum may have developed from a phenotypic change of adenocarcinoma cells to endocrine cells during tumor progression.

The mechanisms of neuroendocrine differentiation of carcinoma cells are still unknown and it is questionable if this hypothesis can be applied to other situations.

New investigations are thus necessary in order to elucidate the pathogenesis of these tumors and the impact of their particular histological pattern on the prognosis of patients.

References

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Figure 1 – Area of adenocarcinoma with tubular pattern of growth (HE stain, ×25)

Figure 2 – The two neoplastic components separated by a fibrous band: carcinoid to the left and adenocarcinoma to the right (HE stain, ×10)

Figure 3 – Area of carcinoid with an acinar pattern of growth (HE stain, ×25)

Figure 4 – Grimelius argyrophilic stain revealed the presence of black granules within the cytoplasm of carcinoid cells (×100)

Figure 5 – Carcinoid cells were positive for neuron specific enolase (×25)

Figure 6 – The tumoral cells of the carcinoid component showed positivity for chromogranin A (×50)


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