Squamous cell carcinoma in Barrett’s esophagus

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Summary
Barrett’s esophagus (BE), consequence of chronic gastro-esophageal reflux disease (GERD), is a premalignant condition, capable of turning into adenocarcinoma (ACa). However, the presence of squamous cell carcinoma (SCa) coexisting with Barrett’s metaplasia is reported in some papers. The aim of this paper is to present 17 patients involving synchronous BE and SCa.

Key words. Barrett’s esophagus, adenocarcinoma, squamous carcinoma.

Carcinoma epidermoide en esófago de Barrett

Resumen
El esófago de Barrett, consecuencia del refluo gastroesofágico crónico, es una condición premaligna capaz de desarrollar adenocarcinoma. Sin embargo, la presencia de carcinoma epidermoide, coexistiendo con metaplasia de Barrett, fue reportado en algunas publicaciones. El propósito de este trabajo es presentar 17 pacientes con esófago de Barrett y carcinoma epidermoide sincrónico.

Palabras claves. Esófago de Barrett, adenocarcinoma, carcinoma epidermoide.

Abreviaturas
BE: Barrett’s Esophagus
C&M: Prague criteria
GM: gastric metaplasia
IM: intestinal (specialized) metaplasia
GERD: gastro-esophageal reflux disease
ACa: adenocarcinoma
SCa: squamous carcinoma
Mx: máximo
Mn: mínimo

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Barrett’s esophagus (BE), according with the Montreal Consensus criteria,1 is defined as columnar metaplasia lining the distal esophagus, with specialized intestinal metaplasia, with goblet cells (IM), or gastric metaplasia, and with cardial type or fundic-oxyntic type mucosa (GM).

It is a pre-malignant condition with an increased risk of adenocarcinoma (ACa). Only IM develops ACa. No cancer was found in other types of columnar mucosa.2-4 All the same, non-goblet columnar metaplasia of the esophagus could progress to cancer, but the magnitude of risk is unknown.2

However, we found some papers in which squamous or adenosquamous carcinoma develops jointly with Barrett’s mucosa instead of ACa.5-23

Case report
All patients were diagnosed by means of upper endoscopy, and multiple biopsies were performed in the Barrett’s mucosa and all visible lesions. The appearance and measurement of the metaplasia were classified according to C&M Prague Criteria.2, 24 Patients examined previously to the existence of these criteria were reviewed and reclassified according to the present nomenclature. At least two experienced gastrointestinal pathologists evaluated all biopsies in order to avoid interobserver variation.5, 25

From January 1982 to January 2013, 1,424 BE were diagnosed. IM was found in 501 of them and GM in 923. ACa developed in 67 patients and squamous cell carcinoma (SCa) in 17. Two patients had simultaneously both types of cancer.
In Table 1 we show: date of diagnosis, patient identification with the record number, sex, age, Prague C&M criteria, location and gross appearance. Thirteen patients were male. The average age was 58.23 years old (range 32 to 81 years old). Among women, the average age was 73.25 years old (range 67 to 83 years old).

According to Prague C&M criteria, the appearance was tongue-shaped (M) in 3 patients and circumferential (C) in 14. Regarding the length, 7 BE were short (less than 3 cm) and 10 long (3 cm or more). 9 of the SCa were located in the middle esophagus with a free space of malignant tissue reaching columnar metaplasia. In 2 of them ACa in Barrett's mucosa was synchronous with SCa, in the remaining cases. Neither tumor nor displasia were found in Barrett's columnar metaplasia, while the other 8 SCa had evolved to distal esophagus nearby columnar metaplasia. There were three types of gross appearance in the endoscopy: mass, ulcerative or infiltrative. Varying strictures were present in all cases.

**Tabla 1. Squamous cell carcinoma in Barrett's esophagus.**

<table>
<thead>
<tr>
<th>Date</th>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
<th>Prague C&amp;M</th>
<th>Esophageal location</th>
<th>Gross appearance</th>
</tr>
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<tbody>
<tr>
<td>7/8/08</td>
<td>470 AT</td>
<td>F</td>
<td>67</td>
<td>M1</td>
<td>Middle</td>
<td>Mass</td>
</tr>
<tr>
<td>3/10/98</td>
<td>158 AJ</td>
<td>M</td>
<td>52</td>
<td>C1</td>
<td>Middle</td>
<td>Ulcerative</td>
</tr>
<tr>
<td>15/5/99</td>
<td>125 BJ</td>
<td>F</td>
<td>72</td>
<td>C3</td>
<td>Distal</td>
<td>Ulcerative</td>
</tr>
<tr>
<td>23/4/97</td>
<td>168 GA</td>
<td>M</td>
<td>54</td>
<td>C1</td>
<td>Middle</td>
<td>Mass</td>
</tr>
<tr>
<td>22/5/97</td>
<td>38 GPH</td>
<td>M</td>
<td>64</td>
<td>M3</td>
<td>Middle</td>
<td>Mass</td>
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<tr>
<td>26/3/96</td>
<td>121 LF</td>
<td>M</td>
<td>58</td>
<td>C3</td>
<td>Distal</td>
<td>Mass</td>
</tr>
<tr>
<td>31/10/96</td>
<td>161 MJF</td>
<td>M</td>
<td>72</td>
<td>C1</td>
<td>Middle</td>
<td>Ulcerative</td>
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<tr>
<td>30/10/98</td>
<td>195 MV</td>
<td>M</td>
<td>76</td>
<td>C3</td>
<td>Middle + distal (SCa+ACa)</td>
<td>Mass</td>
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<tr>
<td>26/7/00</td>
<td>219 OF</td>
<td>M</td>
<td>63</td>
<td>C2</td>
<td>Distal</td>
<td>Mass</td>
</tr>
<tr>
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<td>145 OT</td>
<td>M</td>
<td>58</td>
<td>C2</td>
<td>Middle</td>
<td>Infiltrative</td>
</tr>
<tr>
<td>9/8/94</td>
<td>86 PH</td>
<td>M</td>
<td>63</td>
<td>M3</td>
<td>Distal</td>
<td>Infiltrative</td>
</tr>
<tr>
<td>8/2/94</td>
<td>81 LC</td>
<td>M</td>
<td>32</td>
<td>C9</td>
<td>Middle</td>
<td>Ulcerative</td>
</tr>
<tr>
<td>10/10/96</td>
<td>149 QD</td>
<td>F</td>
<td>83</td>
<td>C1</td>
<td>Middle + distal (SCa+ACa)</td>
<td>Mass</td>
</tr>
<tr>
<td>7/10/02</td>
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<td>M</td>
<td>59</td>
<td>C6</td>
<td>Distal</td>
<td>Mass</td>
</tr>
<tr>
<td>13/9/02</td>
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<td>M</td>
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<td>C3</td>
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<tr>
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<td>71</td>
<td>C6</td>
<td>Distal</td>
<td>Infiltrative</td>
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<tr>
<td>11/10/02</td>
<td>465 TB</td>
<td>M</td>
<td>81</td>
<td>C4</td>
<td>Distal</td>
<td>Infiltrative</td>
</tr>
</tbody>
</table>

**Discussion**

It is known that the natural history evolves from GERD to ACa through BE, but only occasional papers report that other types of cancer, mostly squamous or adenosquamous carcinoma, can appear related to BE.5, 23 This fact reminds us that BE is a mosaic of metaplasia, dysplasia and neoplasia, showing variable degrees of architectural and cell changes in the intestinal and gastric epithelium lining the esophagus.26 So, why would not it be possible that BE turn into SCa instead of ACa? We should not forget that BE is a consequence of long-term gastro-esophageal reflux disease.27 Various kinds of refluxed material cause different types of lesions, including ulcers, strictures, metaplasia, dysplasia, and cancer.5, 21, 28

ACa in BE develops on IM. Failure in detection of ACa in biopsies cannot be interpreted as absence of it because of the patchy appearance that it may adopt.26, 29 In our series of 923 GM, no cancer was found in gastric-fundic or cardial columnar mucosa. Both type of cancer (ACa and SCa) were developed in esophagus with IM.

**Referencias**


