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Combined oesophageal adenocarcinoma and carcinoid in Barrett's oesophagitis: potential role of enterochromaffin-like cells in oesophageal malignancy

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Abstract

A case of combined adenocarcinoma and carcinoid tumour which occurred in a patient with a Barrett's oesophagus is presented. Its significance is discussed in relation to the potential role of enterochromaffin-like cells in the development of oesophageal malignancy, and the possibility is raised that long term treatment with omeprazole, commonly used in this condition, could promote this.

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True oesophageal carcinoid is extremely rare and only four cases have been reported in the world literature.¹⁻⁴ It is well recognised that lung tumours can show mixed differentiation and this is well illustrated in combined small cell and non-small cell carcinomas. A recent report⁵ suggests that the diffuse type of gastric carcinoma may be derived from enterochromaffin-like (ECL) cells—that is, the cells

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Figure 1 Superficially invasive moderately differentiated adenocarcinoma arising in an extensive area of glandular dysplasia in a Barrett's oesophagus. (Haematoxylin and eosin, low magnification.)

from which carcinoid tumours develop. We describe a case of combined adenocarcinoma and carcinoid tumour which occurred in Barrett's oesophagitis. This case provides further evidence that ECL cells may be important in the development of upper gastrointestinal malignancy.

Case report

A 57 year old Caucasian man with a 20 year history of heartburn resulting from reflux oesophagitis presented to his general practitioner with a three month history of lassitude and fatigue. He had no other symptoms. The only past history was of ankylosing spondylitis for 30 years. He had not smoked for 20 years but had smoked heavily before that. On examination there were no abnormal findings. A full blood count showed a normochromic normocytic anaemia with haemoglobin of 11·1 g/dl. Barium swallow showed a moderate sized hiatus hernia with normal oesophageal and gastric mucosa. He was referred for endoscopy which showed a Barrett's oesophagus with an ulcerated area at 35 cm. Biopsy indicated Barrett's oesophagitis with severe dysplasia and probable invasive adenocarcinoma. The results of chest radiography, abdominal computed tomography, and liver function tests were all normal and he was admitted for two stage oesophagectomy with colonic interposition.

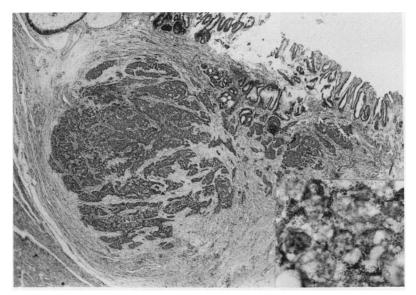
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The excised oesophagus was lined with abnormal granular mucosa with an area of superficial ulceration; microscopy of this showed superficially invasive moderately differentiated adenocarcinoma arising in an extensive area of glandular dysplasia in a Barrett's oesophagitis (fig 1). In addition there were several foci of carcinoid tumour in the same area (fig 2), some clearly within blood vessels. The resection lines and lymph nodes were free from carcinoma.

The patient made an uneventful recovery from surgery. No hydroxyindole acetic acid was detected in a urine sample six weeks later and a repeat thoracoabdominal computed tomographic scan showed no evidence of tumour elsewhere. Repeat liver function tests gave normal results.

Discussion

There is a well recognised association between Barrett's oesophagitis and oesophageal adenocarcinoma.⁶ The Mayo study⁷ followed up 85 patients with Barrett's oesophagitis for 20 years and identified two patients (2.5%) who developed adenocarcinoma. Other smaller studies, however, have suggested a rate of adenocarcinoma of up to 10%.⁸ Oesophageal carcinoids are extremely rare¹⁻⁴ and combined adenocarcinoma and carcinoid of the oesophagus has not been reported previously, although the carcinoid tumour reported by Cheng et al³ showed mucin production.



Submucosal carcinoid tumour showing a trabecular arrangement of cells typical of a foregut carcinoid. (Haematoxylin and eosin, low magnification.) The insert (bottom right) shows numerous intracytoplasmic neuroendocrine granules present in the carcinoid cells (black dots) (Grimelius method, high magnification).

Waldum et al 5 suggest that the diffuse form of gastric carcinoma may be derived from ECL cells. They propose that the occasional mixed tumours seen in the stomach,9 of which this is the equivalent in the oesophagus, could occur by proliferation of a more primitive cell than the committed ECL cell or be the result of paracrine effects of mediators released by the endocrine cells themselves. This raises the possibility that ECL cells could be important in the origin of other oesophageal adenocarcinomas. This is of particular relevance in Barrett's oesophagitis as omeprazole is a standard therapy for this condition and long term treatment with this drug causes ECL hyperplasia and development of carcinoid tumours in rats.10

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Suture granuloma simulating lung neoplasm occurring after segmentectomy

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Abstract

A suture granuloma was resected which developed after segmentectomy of a squamous cell carcinoma of the lung and radiologically mimicked a neoplasm. This report emphasises that although the appearance of the lesion may be typical for malignancy, the possibility of a benign suture granuloma should be considered, especially if the lesion appears shortly after surgery.

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The occurrence of a suture granuloma after resection of lung tissue is very rare compared with surgery in other organs. We present a case of a suture granuloma which developed three months after resection of a lung neoplasm and mimicked local tumour recurrence.

Case report

A 64 year old white man presented with productive cough and fever. The chest radiograph showed right upper lobe collapse. Bronchoscopy revealed obstruction of the right upper lobe bronchus by a squamous cell carcinoma. The tumour was staged T₁N₀M₀ (stage I) and the patient underwent a right upper lobectomy with an uneventful postoperative course. Fourteen months later a routine follow up chest radiograph showed a small well defined round lesion, 15 mm in diameter, localised in the apical segment of the right lower lobe. Bronchoscopic examination showed patent bronchi but brush cytology from the right lower lobe yielded squamous carcinoma cells. This was considered to be a second primary cancer and staging procedures did not show spread. The patient underwent a second thoracotomy and segmental resection was performed. The edges of the lung were sutured with continuous 3/0 silk. The pathological specimen showed 0.2 cm of tumour free tissue around