LETTERS TO
THE EDITOR

Distribution of Campylobacter pylori in the upper and lower gastrointestinal tract: a microbiological and histological study

The presence of Campylobacter pylori in the stomach and in gastric metaplasia in the duodenum and its association with gastroduodenal disease is well established. The mode of spread and the form of the bacteria in transmission, however, is unknown. To ascertain whether C pylori could be detected by routine microbiological and histological methods we decided to map retrieval sites in the gastrointestinal tract from the mouth to the rectum.

Twenty two consecutive, unselected patients (13 men, nine women, median age 50 years) undergoing endoscopy for upper gastrointestinal tract symptoms were studied. At endoscopy (Olympus Q1F-KXQ or Q101 instrument, disinfected between use), macroscopic appearances were noted and any abnormality biopsied. Two adjacent biopsy specimens were also taken from the oesophagus at 35 cm, gastric body, antrum, first and second parts of duodenal mucosa. At sigmoidoscopy two biopsy specimens of rectal mucosa were taken 10 cm from the anus. A buccal scrape was obtained using a tongue depressor. From each site or scrape one biopsy specimen was cultured by highly selective microbiological methods for the presence of C pylori, and one biopsy specimen examined by routine histological methods for identification of Campylobacter-like organisms (CLO). C pylori or CLO were not identified in specimens from buccal scrapes, squamous type oesophageal mucosa, second part of duodenum or rectal mucosa by either microbiological or histological methods. In four patients C pylori was not identified at any site. Gastric and duodenal results in 18 patients positive for C pylori are shown in the table.

The aim of this study was simply to establish if C pylori could be recovered in a recognisable form from sites not previously described. This was not achieved and thus raises the question of how C pylori reaches the stomach and is established in numbers sufficient to be identified by biopsy and culture?

Three possibilities are considered. First, for infection to be established only a small loading dose might be required, and this is so transient that it could be impossible to recover sufficient organisms to identify by recognised techniques. To test this theory a reliable biological model must be developed. Human volunteer studies would be more appropriate if a completely effective treatment was guaranteed. Marshall, in attempting to fulfill Koch’s postulates, ingested 10 colony forming units in peptone water, but other studies repeating this experiment led to chronic atrophic gastritis in one volunteer.

Secondly, transmission could occur in a form not identifiable by present methods. Dimorphism of C pylori is described in the coccid and spiral/vibrio-like forms, which can be separated by means of a sucrose concentration gradient. The coccid forms prevail in old cultures; regrowth could be achieved but the infectivity and transmissibility have not been confirmed.

Thirdly, colonisation of the stomach could occur at a very early age and the organism might remain in the stomach in either dimorphic or very small numbers, with recrudescence when conditions are favourable. Gastritis in children is described in association with C pylori. Studies on eradication and subsequent relapse of C pylori investigated by restriction endonuclease DNA analysis showed that relapse was attributable to recrudescence rather than reinfection by a different strain, which would support the theory of transmission for life.

In conclusion, our inability to show recognisable morphological forms of C pylori at any site other than gastric type mucosa has stimulated us to explore further avenues of transmission, and microbiological and epidemiological studies are in progress.

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Primary malignant melanoma of the oesophagus

Malignant melanoma of the oesophagus is an extremely rare condition with only 110 cases reported in the world by 1985. Indeed, melanoma of the oesophagus was always regarded as metastatic at this site until the demonstration of melanoblasts in oesophageal squamous epithelium by de la Pava. We report a case of a malignant melanoma clearly arising from atypical melanoblasts.

A 61 year old woman had a six month history of regurgitation exacerbated by swallowing, and weight loss of one stone; she had no dysphagia.

Endoscopy showed a pale, polyoid tumour in the mid oesophagus, 25 cm from the incisor teeth. Several biopsy specimens were submitted for histological examination and showed a small cell anaplastic tumour with no evidence of squamous or glandular differentiation. Mucin stains were negative and an immunoperoxidase stain for leucocyte common antigen was negative. The appearances were interpreted as representing primary or secondary anaplastic carcinoma as no mucosal origin was demonstrable.

Barium, ultrasound, and computed tomography studies indicated no evidence of metastases and therefore an Ivor-Lewis oesophagectomy was performed. Initial post-operative course was satisfactory with no leak.

Pathology

The resected oesophageal tumour measured 20 cm after formalin fixation and contained a 3 × 2 cm smooth polyoid tumour projecting into the lumen 5 cm from the limit of resection. Immediately below and adjacent to the main tumour mass were two discrete satellite tumour polyps, 1 cm in diameter. On sectioning, the tumour had a soft pale consistency with a focal area of haemorrhage but no brown pigmentation.

The histological picture was dominated by interweaving fascicles of plump spindle cells with vesicular nuclei and frequent mitotic figures. Other areas, however, had epithelioid characteristics with the cells tending to cluster in nests. Only a few cells in the deeper parts of the tumour displayed faint melanin stippling of the cytoplasm. The squamous epithelium was immediately adjacent to the tumour masses contained numerous junctional nests of atypical melanocytes with pleomorphic nuclei, establishing the primary origin of the melanoma from the oesophageal epithelium. The tumour cells had spread peripherally beneath the intact squamous epithelium, distending submucosal lymphatic channels. All the tumour cells showed strong positivity with immunoperoxidase S-100. Electron microscopy revealed that the transformation showed numerous premelanosomes together with numerous vesicles and vacuoles. Junctional attachment complexes between adjacent tumour cells were a common feature.

Most cases of primary oesophageal melanoma are found to be polypoidal intraluminal masses with contrast radiology at endoscopy, and arise in the distal oesophagus. Problems can be encountered in establishing a pathological diagnosis, particularly on small endoscopy specimens. The melanoma cells may contain little or no melanin pigment. The differential diagnosis includes other spindle cell variants of squamous carcinoma, sarcomas, small cell carcinoma, carcinosarcoma metastatic melanoma to the oesophagus. In this case a diagnosis of primary malignant melanoma became established with the demonstration of atypical junctional activity affecting the oesophageal squamous epithelium at the margin of the neoplastic mass. Further confirmation came from the S-100 protein positivity and the demonstration of premelanosomes on electron microscopic examination.

The tumours are highly malignant. At presentation, 30–40% of cases have metastases. Chemotherapy is ineffective, although the use of radiotherapy alone, or as an adjunct