Tumour-forming gastritis

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EDITORIAL SYNOPSIS  A case is described in which, over a period of 16 years, gastritis became increasingly polypoid until carcinoma supervened. The possible relationship between tumour-forming and polypoid gastritis to gastric cancer is briefly discussed.

The word gastritis is used in different senses by clinicians, radiologists, and pathologists and continues to cause confusion. The clinician applies it to a series of acute conditions in which the evidence for inflammation of the gastric mucous membrane is strong and often well supported by other investigations; he may use it to a varying degree and with less justification in cases of chronic dyspepsia, where there is no radiological evidence of peptic ulcer or other lesion. The radiologist is apt to diagnose gastritis on the thickness, shape, and rigidity of folds of mucous membrane that may be due to many variables, one of which is inflammatory change. Of gastroscopy, it may be claimed that certain appearances are recognizable as being due to inflammation and that within the recommendations of Schindler (1937) valid inferences may be drawn. Schindler (1947) has also linked a number of these gastroscopic appearances with their underlying histological changes. The gastric biopsy technique of Wood and Taft (1958), though the specimens are taken blindly, has helped to link clinical, gastroscopic, and histological conceptions of gastritis into a composite whole.

In his book 'Gastritis', Schindler (1947) described cases of 'tumour-forming' gastritis. These presented with atypical appearances of tumour either radiologically, on gastroscopy, or at operation. He described the microscopy of the condition as showing either atrophic hyperplastic gastritis or hypertrophic gastritis, and the designation 'tumour-forming' indicates only 'that hyperplasia or proliferation of such a degree is present as to produce the picture of a tumour either at x-ray examination, at gastroscopy, or at surgical operation'. Schindler does not suggest that this type of gastritis can undergo tumorous, in the sense of carcinomatous, change, and this makes the name 'tumour-forming' gastritis somewhat anomalous because certain types of gastritis are regarded as more prone than usual to become carcinomatous and the condition of atrophy of the mucosa accompanying pernicious anaemia is known to be associated with an increased incidence of gastric carcinoma. That Schindler's tumour-forming gastritis can be followed by carcinoma of the stomach is illustrated by the following case.

CASE HISTORY

C.B. was seen in 1936, when 35 years old, complaining of dyspepsia. A barium meal radiograph showed no peptic ulceration though slight deformity of the duodenal cap, but there was evidence of gastritis in the radiological sense; gastroscopy showed chronic hypertrophic gastritis. In 1951 radiographs showed gross thickening of the mucosa, which was polypoid in the fundus. Gastroscopy was performed twice in this year and showed similar polypoid changes of the mucous membrane, the appearances resembling those of Schindler's tumour-forming gastritis. In 1952, gastroscopy showed an increase of these polypoid changes but still no evidence of ulceration; the condition indicated need for frequent and regular supervision but at this stage the patient stopped attending the out-patient clinic. In 1953 he was seen at the Middlesex Hospital where radiographs for the first time showed a large suspicious ulcer on the lesser curve of the fundus; a total gastrectomy was performed, the condition proving to be carcinomatous. Naked-eye examination of the stomach showed the mucosa in the neighbourhood of the growth to be thrown into polypoid folds; a section of the growth (Fig. 1) confirmed its being a carcinoma and of mucous membrane (Fig. 2) showed atrophic gastritis.

DISCUSSION

The diversity of change in gastric mucous membrane has led to many classifications, depending upon the methods of study; these are steadily falling into patterns based upon histological appearances which are themselves likely to be altered by the method of obtaining specimens. Post-mortem autolysis was a frequent source of error until Faber (1935) initiated
the fixing of gastric tissue immediately after death, and the handling and clamping of the stomach at operation led to histological alterations which impeded the study of gastritis till Schindler (1947) introduced his technique of quick gastric biopsy with scissors at laparotomy. Though the biopsy tube of Wood and Taft (1958) has the disadvantages of blindness and small size of the specimen obtained this is a method of considerable value, and is easy and safe to perform. The relationships of gastritis to carcinoma of the stomach are discussed in many publications, though with inconstant conclusions. It may appear to precede the formation of tumours, or it may seem to result from the tumour's presence as a zonal phenomenon and there is little or no evidence of gastritis in a number of cases. Gastric atrophy (as distinct from atrophic gastritis) accompanies pernicious anaemia and is reported to have about three times the normal likelihood of subsequent change into gastric cancer; amongst other types of gastritis that are associated with carcinoma is the rare diffuse giant hypertrophic gastritis (Wood and Taft 1958). 'It is certain that adenomatous polyps (of the stomach) may become carcinomatous', according to Stout (1953), and this statement is generally accepted; Stout (1953) also stated: 'It is possible that gastritis polyposa may be a precancerous lesion, and this must be considered in any discussion of its treatment'. The difficulty here is the definition of gastritis polyposa; Schindler's tumour-forming gastritis can undoubtedly be a polypoid condition and is histologically quite different from ordinary adenomatous polyposis of the stomach though the size and location of the masses is variable. Cases of this type are often so suggestive of cancer that laparotomy is undertaken and Schindler mentions five such, in none of which was carcinomatous change found, although in another case at operation and one at necropsy lymphomatous infiltration had occurred.

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