

Dogma Disputed

ON THE ORIGIN OF DUODENAL-ULCER PAIN

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THE dogma that duodenal-ulcer disease cannot be diagnosed unless an ulcer crater is found is in every textbook of gastroenterology. Clinicians use radiology, or more frequently endoscopy, to make a diagnosis, and even after medical treatment or surgery the search continues for ulcer craters. The accepted end-point for both diagnosis and treatment, the appearance of the duodenal mucosa, is challenged. It is suggested that there is not just one syndrome but a whole spectrum of symptoms associated with a duodenal ulcer, arising from changes in the normal physiology of the duodenum, stomach, and oesophagus rather than the hole in the duodenum, and that most of the "duodenal-ulcer" pain arises from the lower oesophagus. It follows that the actual appearance of the duodenum is to a great extent irrelevant.

Emphasis should be on clinical history rather than on close examination of the barium meal or the endoscopic findings. It then becomes clear that there are two main types of duodenal-ulcer pain—one originating in the epigastrium just below the xiphisternum between the rib margins and the other arising elsewhere in the abdomen but usually in the right upper quadrant. Epigastric tenderness as a sign of duodenal ulceration is not located at the duodenal cap. Many patients present with bleeding from a huge duodenal ulcer but have no symptoms other than from blood loss. At laparotomy for duodenal ulceration some patients are closed up without a definitive procedure because the appearance of the duodenum is normal despite typical symptoms. Conversely scarred duodenal caps may be found in patients without any ulcer-like symptoms. These observations led to research on the epigastric pain of duodenal ulceration, because symptoms did not match the pathological appearances of a duodenal ulcer.

EXPERIMENTAL EVIDENCE

Epigastric-pain-reproduction Test

Bernstein and Baker instilled 0.1 normal hydrochloric acid into the oesophagus through a nasogastric tube and reproduced heartburn in patients with a hiatus hernia.¹ The epigastric-pain-reproduction test is a development of this but depends on a careful manometrically controlled perfusion within 5–10 cm of the gastro-oesophageal junction and a detailed analysis of symptoms. Epigastric pain can be reproduced in duodenal-ulcer patients with perfusion of 30 ml of 0.1 normal hydrochloric acid over a 4 min period.² If careful attention is paid to when the pain was last severe all patients with epigastric pain awoken by such pain at night for at least 4 weeks can be shown to have a positive pain-reproduction test with 100 ml or less acid, and this is abolished with a small amount of 2.74% sodium bicarbonate solution.³ All patients with other anatomical locations for their duodenal-ulcer pain have negative tests. The acid is known to act locally on the lower oesophagus because continuous simultaneous perfusion of the stomach with alkali

does not alter the pain-reproduction test, and consecutive instillation of 200 ml of acid into the stomach for 20 min does not reproduce epigastric pain.

Questionnaire Analysis of Symptoms

Between 1969 and 1973 a computerised database was established with information on the indigestion symptoms and lifestyle of patients with duodenal ulceration for a longitudinal study to monitor the effects of natural history or of surgery.^{4,5} Also symptoms have been analysed and correlated with barium meal findings and pentagastrin-acid-secretion tests.

Of 319 patients with duodenal ulceration, 174 had epigastric pain and 145 had non-epigastric pain—an 11/9 ratio. Symptoms of gastro-oesophageal reflux, acid regurgitation, and nocturnal waking were more frequent in the group with epigastric pain than in those with non-epigastric pain. The non-epigastric-pain group presented more frequently with haematemesis or melaena but overall had a similar number of previous bleeding episodes. They were more likely to have vomiting as a symptom.

There was no significant difference between the groups in age, height, social class, family history of duodenal ulcer, blood group, rhesus factor and saliva secretion status, length of attacks and remissions, previous perforations and bleeds, sugar consumption, and lifestyle (includes size of breakfast, lunch, and dinner, times of rising and going to bed, and alcohol and cigarette consumption). The two groups did not differ in radiological findings or results of pentagastrin tests when basal acidity, basal acid output, maximal acid output, and peak acid output were compared.

DUODENAL-ULCER PAIN ARISES FROM THE LOWER OESOPHAGUS

The suggestion that duodenal-ulcer pain arises from the lower oesophagus and not from the duodenal ulcer raises a number of points.

Some patients can have pain from the duodenum itself—usually from the right upper quadrant—and are less likely to have heartburn and gastro-oesophageal reflux symptoms. Duodenal ulcers cannot be characterised by a single pattern of symptoms. There is evidence that patients with non-epigastric pain have a strong competent gastro-oesophageal sphincter, which prevents reflux.⁶ Those with epigastric pain have a weaker sphincter, more reflux into the oesophagus, and are more likely to have heartburn. Thus, two groups of patients can be distinguished: (i) those with epigastric pain arising from the lower oesophagus and (ii) those with non-epigastric pain arising either from the ulcer or from other pathophysiological changes. In the first group the ulcer is present but does not give rise to symptoms, and in the second group the lower-oesophageal-pain mechanism is protected by a strong sphincter, which prevents gastro-oesophageal reflux. The existence of a third group—(iii) those who have an ulcer but no pain from the crater itself and also no pain from the lower oesophagus—is not generally recognised by doctors, and such patients are considered to be exceptions. However, it is logical when the concept that duodenal ulcers themselves need not necessarily cause pain is accepted. These are the patients who, without previous symptoms, suddenly present with bleeding, pyloric stenosis, or complications arising from treatment with cortisone or anti-rheumatic drugs.

Some patients may receive inappropriate treatment because they do not fit in with current diagnostic categories.

These patients have epigastric pain and heartburn (relieved by antacids), nocturnal waking, and attacks and remissions and are indistinguishable clinically from the usual duodenal-ulcer patient. After repeated investigation to exclude duodenal ulceration and hiatus hernia they are labelled X-ray negative and endoscopically negative, although clinically they appear to have typical duodenal ulcers. These patients represent the fourth group (iv), with pain arising from the lower oesophagus but a normal duodenal mucosa and no ulcer. Should they not be treated as though they did have an ulcer?

If the ulcer itself is not important, why is there such an obsessive search with endoscopy to find ulcers or minute erosions? There is little correlation between symptoms and microscopic changes in the oesophageal mucosa and between duodenal ulceration and the appearance of the mucosa of the stomach and duodenum.⁷ Endoscopists should be prepared to justify their search for craters and erosions and the expense of biopsies by correlating their findings with symptoms and assessing whether such findings alter decisions about treatment. They must also be challenged to reproduce pain from the duodenum or the ulcer. If the pain of duodenal

ulceration does not necessarily arise from the ulcer, why do endoscopists see it as the end-point for treatment with H₂ histamine blockers? I propose four possible end-points for medical treatment: a healed ulcer and no symptoms; a healed ulcer but symptoms present; an ulcer present and no symptoms; and an ulcer and symptoms present.

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