Alimentary tract

Heartburn – the acid test

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SUMMARY To determine whether symptoms of gastro-oesophageal reflux are related to the degree of oesophageal acid exposure, 190 patients (of 220 referred) with heartburn and acid regurgitation were compared with 50 normal subjects. A definite relationship between frequency of reflux symptoms and degree of oesophageal acid exposure was found both in patients with and without oesophagitis. We conclude that the frequency of gastro-oesophageal reflux symptoms is related to degree of oesophageal acid exposure.

Dyspepsia is a condition mainly characterised by the lack of a distinct definition.1 Symptoms vary from mild flatus to severe epigastric pain and may be caused by colonic dysmotility, gall stones, peptic ulcer, cancer and oesophagitis. Dyspepsia is a common complaint and often treated without further investigation. An attempt was recently made to analyse dyspeptic symptoms into more distinct subgroups in order to give better guidelines for treatment.1 One subgroup suggested was ‘gastro-oesophageal reflux-like dyspepsia’ mainly characterised by ‘substernal or epigastric discomfort and heartburn, a burning upper epigastric pain, regurgitation of acid and occasionally food’. If these symptoms are caused by acid exposure of the oesophageal mucosa, it would be logical to prescribe acid reducing antireflux therapy.

Oesophageal symptoms, however, may be caused not only by acid gastro-oesophageal reflux, but also by oesophageal dysmotility and diseases in adjacent thoracic and upper abdominal organs.3 The present study was performed to investigate whether patients with reflux-like dyspepsia actually do have increased acid exposure of the oesophagus.

Methods

STUDY POPULATION Two hundred and twenty consecutive patients with dyspeptic symptoms probably originating from the upper gastrointestinal tract were referred mainly by general practitioners for oesophageal investigation. For the purpose of this and other studies doctors in our region were encouraged to refer their patients to us. Thus, many patients with less severe disease are included who otherwise would not have come to an oesophageal laboratory. The patients underwent symptom evaluation, upper flexible endoscopy, oesophageal manometry and ambulatory 24 hour intraoesophageal pH monitoring. In 190 patients reflux like dyspepsia (heartburn and acid regurgitation) was present. Their median age was 49 years (range 17–75) and the majority were men (n=112). The remaining 30 patients suffered exclusively from epigastric distress, chest pain or asthma-like disease without concomitant heartburn or acid regurgitation. For comparison 50 endoscopically normal asymptomatic subjects underwent manometry and pH-monitoring. These were mainly recruited among the hospital staff. Their median age was 40 (range 30–77).

ENDOSCOPY Upper oesophagogastroduodenal endoscopy was performed as a standard procedure with a flexible endoscope under light topical anaesthesia of the pharynx. The presence or absence of macroscopic erosive oesophagitis was noted. Erosive oesophagitis was defined as grade I (Savary-Miller classification). None of the study subjects suffered from cancer or peptic gastroduodenal ulcers.

SYMPTOM EVALUATION Symptom evaluation was focused on the most typical
symptoms of reflux-like dyspepsia: heartburn and acid regurgitation. It was graded (1) if occasionally present, (2) if present one to three times daily, and (3) if more frequently or almost constantly present. The patients were interviewed before manometry and pH-monitoring by a nurse using a standard questionnaire.

**Oesophageal Manometry**

Using the stationary pull-through technique, position of the distal high pressure zone was determined using a water perfused catheter with distal side holes at 5 cm intervals. A hydrocapillary gas pressurised Arndorfer (R) pump was used, giving a flow of 0.5 ml per minute.

**Ambulatory 24 Hour Oesophageal pH Monitoring**

The Syntetics (R) ambulatory pH-monitoring system with antimony pH-electrode and solid state memory was used. After calibration at pH 7 and 1, the pH electrode was introduced nasally and placed with the tip 5 cm above the oral margin of the manometrically localised distal high pressure zone. The patients were instructed to carry on normally during the 24 hours. Only acid food and beverages were excluded. The monitoring was analysed on an ordinary personal computer using the Esophagram (R) (Gastrosoft) computer program. Gastro-oesophageal reflux was expressed as acid exposure time — that is, per cent time spent with a pH less than 4 during the total 24 hours as well as during eight three hour periods.

**Statistical Analysis**

Results from pH monitoring in patients are severely skewed, thus only non-parametric statistical methods were used. Median and interquartile ranges described the results and the Mann-Whitney U-test tested the difference between two non-paired groups.

**Results**

Erosive oesophagitis was found in 33% of the 190 patients with reflux dyspepsia. The frequency of heartburn and acid regurgitation reported by the patients corresponded well to the median oesophageal acid exposure time as measured by 24 hour pH monitoring in patients with and without erosive oesophagitis. Patients with a normal oesophagus had a uniformly lower median acid exposure time. Patients with frequent symptoms exhibited significantly higher acid exposure of the oesophagus than those with less frequent symptoms, though even the latter suffered from significantly higher median acid exposure than asymptomatic subjects (Table).

All study groups showed the typical distribution of acid reflux during the 24 hours of pH monitoring but on clearly different levels. While little reflux was detected during the ‘sleep hours’ from midnight to six am, the acid exposure increased during the day with a peak in late afternoon and evening. Patients with almost continuous symptoms already had a high acid exposure in the morning (Figure).

**Discussion**

This study suggests that if a patient presents with reflux-like dyspepsia, there is a considerable chance that the acid exposure of the mucosa in the distal oesophagus is increased in comparison with asymptomatic subjects. The more frequent the symptoms, the greater the acid exposure. This is true for patients with and without macroscopic signs of erosive oesophagitis.

Increased acid exposure of the distal oesophageal mucosa may lead to symptoms of reflux such as heartburn and acid regurgitation. Depending on reflux intensity, clearance capacity, mucosal resistance and composition of the refluxed material erosive changes gradually may develop. This process is
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known as gastro-oesophageal reflux (GOR) which may be present both with and without macroscopic oesophagitis.

When a patient with GOR seeks medical advice, he has usually tried proprietary antacid or alginic acid compounds and is not satisfied with their effect. The increased oesophageal acid exposure in these patients indicates that efficient acid reducing anti-reflux therapy should be instituted in adjunct to general antireflux advice. The logic of this attitude was confirmed in a study of cimetidine for treatment of non-ulcer dyspepsia. Patients responding best were those with reflux symptoms.

The question remaining is one of dosing regime. Acid exposure after midnight is low, indicating against bed time medication. In patients with less severe symptoms, it seems logical to concentrate the acid reduction to afternoon and evening – for example, prescribing medication at lunch and evening meals. In patients with almost constant heartburn the whole day should be covered, starting with medication at breakfast. In a few patients with night symptoms, bed time medication may be considered.

In most patients with reflux-like dyspepsia, treatment must be instituted without further investigation. Resources are usually not sufficient to obtain upper endoscopy and pH monitoring in all patients who present with reflux-like symptoms. Should, however, symptoms persist or be insufficiently con-
trolled by medication it is advisable to send the patient for an upper endoscopy. Many patients, however, with increased acid exposure do not show signs of macroscopic oesophagitis, even if symptoms are severe. In these patients an intraoesophageal pH monitoring is of value. During this investigation the acid reflux profile may be determined as well as the temporal relationship between symptoms and recorded reflux episodes. In a few patients it may even be advisable to repeat the pH monitoring if normal results are obtained during the first investigation, as reflux may vary considerably from day to day.

Treatment for GOR traditionally has been focused on healing of macroscopic oesophagitis. In our opinion symptoms are equally or, at least to the patient, more important to treat. The goal is rapid relief of symptoms; restitution of the oesophageal mucosa is a secondary benefit that in many patients will be achieved only after longterm treatment. It is important to note that disappearance of macroscopic oesophagitis in the patient with an impaired anti-reflux mechanism does not indicate that the patient is cured. The reflux will continue and supportive treatment will be needed for years, if not for life.

In conclusion, our study shows that reflux-like dyspepsia is accompanied by increased acid exposure of the distal oesophageal mucosa. This finding supports the view that identification of these symptoms within the heterogeneous group of dyspeptic patients serves a practical purpose with regard to their treatment. Institution of effective acid secretory reducing medication seems to be a logical approach in patients with dyspepsia of the reflux type.

References