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T89

LONGITUDINAL MEASUREMENTS OF TOOTH WEAR IN PATIENTS WITH GASTRO-OESOPHAGEAL REFLUX DISEASE (GORD).

DW Bartlett, *DF Evans, †L Blunt, BGN Smith. Dept of Conservative Dentistry, UMDS, Guy's Hospital, London, *GI Science Research Unit, St Bartholomew's and the Royal London Schools of Medicine, †Department of Manufacturing and Mechanical Engineering, University of Birmingham, UK.

GORD is known to cause palatal dental erosion in susceptible patients, particularly those experiencing regurgitation. The destruction of enamel and dentine caused by erosion from regurgitated gastric juice can be catastrophic leading in some cases to the complete loss of coronal enamel and dentine. It is important to identify the aetiology of the erosion and initiate preventive measures to prevent further tooth wear. Accurate measurement of erosion is important to establish the damage caused by regurgitation and therefore the effect of GORD on teeth. The aim of this study was to measure tooth wear in patients with palatal erosion and GORD and compare it to a group of controls with no symptoms or erosion.

Tooth wear was measured over a 6 month period in 23 patients with GORD and erosion and 12 controls. Metal disks 0.1 mm thick and 2 mm in diameter were cemented to the palatal surface of upper central incisors and clear of the opposing dentition. Impressions of the disk and surrounding tooth surface were taken and repeated after 6 months. Wear was measured with a contacting laser prolifometer using the metal disks as fixed reference points. Wear was recorded as a change in depth from the centre of the metal disk to a point on the surrounding tooth surface which was reproducible on both impressions. Reproducibility was within acceptable limits (r = 0.85). Wear was successfully measured in 13 erosion patients and 7 controls. The most common reason for failure was decementation of the metal disks over the 6 month assessment period. A significant increase in enamel loss was observed between the patients (median 33.5 µm, range 11.4-108.2) when compared to the controls (2.6 µm, range 0.5-15.8, [p = 0.004]).

In conclusion, palatal tooth wear was significantly increased in patients with GORD when compared to controls. We recommend that dentists should consider anti-reflux medication or a specialist referral in patients with worsening palatal tooth wear and symptoms of GORD.

T90

THREE TO FOUR YEAR PROSPECTIVE STUDY OF PROGNOSTIC INDICATORS IN GASTRO-OESOPHAGEAL REFLUX DISEASE

NI McDougall, BT Johnston, JSA Collins, RJ McFarland, AHG Love. Dept. of Medicine, Queen's University Belfast, Northern Ireland, UK.

Aims: Data on the long term natural history of gastro-oesophageal reflux disease (GORD) are sparse. This study was designed to determine the clinical outcome 3 to 4 years after initial diagnosis of GORD and to identify specific prognostic indicators of a poor outcome.

Method: 101 patients with GORD were followed up by postal questionnaire assessing symptomatology and therapeutic requirements, repeat endoscopy and repeat oesophageal pH monitoring (pH mon.) a minimum of 3 years after diagnosis.

Results: 77 (77%) patients responded (39 male, mean age 49yrs (24-79yrs), mean follow-up period 39 months (range 32-54 months)) of whom 28 had grade II-III oesophagitis at initial endoscopy, 17 had normal endoscopy but abnormal pH mon. (%time pH < 4 of >4.8%) and 32 had normal endoscopy and pH mon. but typical reflux symptoms. Forty-three patients had repeat endoscopy, 45 had repeat pH mon. and 25 refused repeat investigations. At follow-up, 32 (42%) patients were taking acid suppression therapy (acid supp.) and a further 25 (33%) had frequent heartburn. Based on repeat investigations, a further 15 patients started acid supp. giving a total of 47 (61%) patients who required acid supp. 3-4 years after diagnosis.

Using the chi-squared test or t-test for independent samples, the following factors were shown to predict an ongoing need for acid suppression therapy at 3-4 year follow-up: oesophagitis on initial endoscopy (p=0.009), abnormal pH mon. (p=0.0005), increased age (p<0.0005) and increased body mass index (BMI) (p=0.001). Presence of oesophagitis gave patients a relative odds ratio of being 4.3 times more likely to require acid supp. at follow-up. Gender, smoking status, alcohol intake and lower oesophageal sphincter pressure had no effect on outcome. Regression analysis confirmed that age (p=0.0007), BMI (p=0.04) and endoscopy result (p=0.04) all significantly affected outcome. pH mon. did not contribute to the regression analysis as an independent factor.

Conclusions: 61% of GORD patients still require acid suppression therapy 3-4 years after initial diagnosis. Age, body mass index and the presence of oesophagitis at initial endoscopy all predict those who will require long term acid suppression therapy.

T91

PRIOR DIAGNOSIS OF BARRETT'S OESOPHAGUS IS RARE IN PATIENTS WITH OESOPHAGEAL ADENOCARCINOMA

C M Brown, R Jones, T Shirazi, B Codling, R M Valori. Gloucester Gastroenterology Group, Gloucestershire Royal Hospital, Great Western Road, Gloucester, GL1 3NN, England

Background: Endoscopic screening is usually offered to patients with Barrett's oesophagus to detect treatable oesophageal adenocarcinoma. This policy will only be effective if the group at risk is identified by endoscopy after consulting with dyspepsia.

Aims: To determine the frequency of prior consultation for dyspepsia, endoscopy and diagnosis of Barrett's oesophagus in a consecutive series of patients with oesophageal adenocarcinoma.

Methods: All new diagnoses of oesophageal adenocarcinoma from a well defined postcoded district (pop: 280,500) during a 5 year period (1990-4) were identified. Open access endoscopy has been available in this district for >17 years and the overall rate of endoscopy is 1% of the population per year. Details of previous consultation, investigation and diagnosis of Barrett's oesophagus were obtained from general practitioner and hospital records.

Results: Full details were available for 58 of 67 patients presenting with oesophageal adenocarcinoma. 30 (58%) of these patients had consulted their general practitioner at least once for dyspepsia, 11 (19%) had been endoscoped, 14 (24%) had had a barium meal and only 4 (7%) had a diagnosis of Barrett's oesophagus prior to the diagnosis of oesophageal adenocarcinoma.

Conclusions: In the presence of an established open-access endoscopy service a diagnosis of Barrett's oesophagus prior to diagnosis of oesophageal adenocarcinoma is rare. If current referral patterns for endoscopy remain unchanged 93% of patients destined to develop oesophageal adenocarcinoma will never present with a pre-cancerous lesion. If all patients consulting with dyspepsia are endoscoped <50% of prospective cancer patients will be available for screening. Other risk factors are required to select patients for endoscopic screening.

T92

BARRETT'S OESOPHAGUS, HIGH GRADE DYSPLASIA AND EARLY ADENOCARCINOMA. COMPARISON OF ENDOSCOPIC BIOPSY AND SURGICAL PATHOLOGY FINDINGS.

AJ Cameron, H.A. Carpenter. Mayo Clinic, Rochester, Minnesota 55905, USA.

Because of the increased cancer risk, we follow patients with Barrett's oesophagus with endoscopy every 1-2 years. Using standard Olympus GIF-100 instruments, 4-quadrant biopsies are taken every 2 cm and from any focal abnormality. Oesophageal resection is usually advised for invasive adenocarcinoma (AC) or high grade dysplasia (HGD). To determine the extent and distribution of early AC and HGD in Barrett's oesophagus, and the value of endoscopic biopsy, we examined 30 consecutive oesophageal resection specimens in detail. None of these patients had a visible tumor mass on endoscopy, but all had a biopsy interpreted as HGD or AC. Following surgery, the resected oesophagus was mapped in 2 by 1 cm blocks, and about 100 microscopic slides per case examined.

19 patients (14 male, mean age 60) had oesophagectomy for a preoperative diagnosis of HGD. Histological mapping showed submucosal AC in 2 (10.5%), 17 having HGD only. 12 patients (11 male, mean age 62) had a preoperative biopsy suggesting AC, without obvious cancer on endoscopy. One patient had node metastases, and resection was not done. Histological mapping in the other 11 confirmed AC in 5 cases (4 intramucosal, 1 submucosal); in 6, only HGD was found.

Endoscopic findings did not distinguish early AC from HGD:

Endoscopic appearance	Adenocarcinoma	High grade dysplasia
Flat Barrett's	1 (14%)	10(43%)
Nodular area	3 (43%)	2(9%)
Single nodule	1 (14%)	2(9%)
Ulcer	2 (28%)	8(35%)
Stricture	0	1(4%)

The mean length of Barrett's oesophagus was 7.4 cm, mean area 37.3 sq. cm. The mean surface area of 7 adenocarcinomas was 1.1 sq. cm. HGD was found in all 30 cases, occupying a mean area of 6.5 sq. cm. The HGD involved large areas of the Barrett's in 11 cases, scattered multiple foci in 11, and a single area in 8. In 15 cases the total HGD area was 1 sq. cm or less. The mean area of low grade dysplasia was 15.7 sq. cm.

Conclusions: #1 The area of Barrett's oesophagus involved with HGD or early AC is often very small. #2. Multiple biopsies can fail to detect these lesions due to sampling error. #3 The distinction between HGD and AC is not always possible on biopsy.

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T93

OESOPHAGEAL CANCER IS AN UNCOMMON CAUSE OF DEATH IN PATIENTS WITH BARRETT'S OESOPHAGUS.

A. van der Burgh[1], W.C.J. Hop[2], J. Dees[1], M. van Blankenstein[1], Division of Gastroenterology[1], University Hospital Rotterdam and Institute of Epidemiology and Biostatistics[2], Erasmus University, Rotterdam, The Netherlands

The incidence and outcome of Barrett's Carcinoma (BC) in patients with Barrett's Oesophagus (BO) was ascertained in a cohort of 166 patients with BO >3cm and without carcinoma who had been identified between 1973 and 1986 at upper GI endoscopy. Their vital status or cause of death was ascertained in 1986 and 1994. In both studies 155 patients (93%) were traced. In 1986 4 cases of BC had developed, in 1994 another 4. The incidence of oesophageal cancer was 1:170 and 1:180 patient-years respectively, the final follow-up comprised 1440 patient-years (average 9,3 years).
In 7 out of 8 patients the tumour was symptomatic: 6 complained of dysphagia and 1 of recurrent reflux symptoms. One tumour was diagnosed at endoscopic follow-up.
Three patients had carcinoma *in situ*, five invasive cancer. Six patients underwent surgical resection. Three survived, one died from unrelated causes, one from postoperative complications and one from metastases. One refused treatment and another was considered unfit for surgery. Both the latter died from unrelated causes.
Of the total group 79 patients have died at a mean age of 75 years, but only two from BC (2,5%).

Conclusions:
1. The incidence of oesophageal cancer in patients with BO is one 1 in 180 patient-years.
2. Oesophageal cancer is an uncommon cause of death in patients with BO (2,5%).
3. The patients in this cohort would not have benifited from an endoscopic surveillance programme.

T94

BARRETT'S OESOPHAGUS : THE ROLE OF OESOPHAGEAL BILE REFLUX

CK MacKay*, MTP Caldwell, , RC Stuart*, TN Walsh, JN Baxter*, PJ Byrne, TPJ Hennessy
University Departments of Surgery, St, James Hospital, Dublin, and Glasgow Royal Infirmary*.

Oesophageal bile reflux has long been implicated in the pathogenesis of Barrett's Oesophagus. Measurement of bile reflux in a physiological millieu has, until recently, been difficult. Bilitec 2000 allows assessment of bile reflux in an ambulatory setting. Thirty-five patients with Barretts underwent simultaneous 24hr ambulatory pH and bile reflux monitoring. These were compared with 20 patients with uncomplicated gastro-oesophageal reflux disease (GORD), and 14 normal controls. All were allowed normal diet and the interdigestive and supine periods analysed in order to exclude food artefact.

Results:	**Barrett's** n= 35	**GORD** n=20	**Normals** n=14
Esophageal bile reflux (%time absorbance > 0.14) Median (range)]			
interdigestive	22(10-67)	4.2 (2.3-7.3) **	1.9(0.5-7.8) *
supine	17(8.8-61) **	2.0(0.2-7.2) **	0.5(0.1-4.4) *
DeMeester Acid Score Mean (SEM)	78.2(13.4) *	37.3(7.2)	6.8(1.3) **

**p<0.001 , *p<0.05, Wilcoxon rank sum test

Conclusion: Oesopahgeal bile reflux is significantly increased in patients with Barrett's oesophagus when compared with those with uncomplicated GORD and normal patients. This data supports the hypothesis that bile reflux may be involved in the pathogenesis of Barrett's oesophagus.

T95

Efficacy of the palliative treatment of cardia- and oesophagus-carcinoma with high-dose rate intraluminal radiotherapy; follow up of 89 patients.

P.J. Bus[1], J.H. Meerwaldt[2], N. Van Bentem[1], P. Noach[2], G.H. van Olffen[1], (introduced by M. van Blankenstein). Department of Gastroenterology[1] and Radiotherapy[2], Medisch Spectrum Twente, Enschede, the Netherlands.

Background: High-dose rate intraluminal radiation (HDRIR) with or without external radiotherapy (ERT) in oesophageal cancer has been applied for tumour reduction and relief of dysphagia. The advantage of this method is maximum local radiation without overexposure of neighbouring organs.

Patients and methods: During a period of $3^3/_4$ year (jan 1992 untill nov 1995) 89 patients (male 66, female 23) were evaluated with respect to the survival, complications and need for dilatation before and after radiotherapy. According to the extent of the disease three different protocols were used:
1. Only locally advanced disease (tumor < 5 cm) without metastasis.
   Treatment: 2 x 7,5 Gy HDRIR and 50 Gy ERT.
   17 patients (14 male, 3 female), mean age 64 yrs.
2. Tumor ≥ 5 cm, N_{0-1}, M_0.
   Treatment: 10 Gy HDRIR and 10 x 3 Gy or 20 x 2 Gy ERT.
   35 patients (26 male, 9 female), mean age 69 yrs.
3. Patients with limited life expectancy (< 3 month) based on staging and Karnofsky-score.
   Treatment: 15 Gy HDRIR only.
   37 patients (26 male, 11 female), mean age 68 yrs.

Results: In group 1, 2 and 3 the mean duration of survival was 241, 197 and 175 days respectively, with a one year survival of 30, 17 and 8%.
Prior to the HDRIR 50 of the total group of patients (56%) required endoscopic dilatation. After radiotherapy dilatation was still needed in 30% of the patients, in most cases just once (76%). Only seven patients needed an endoprothesis (8%) and three patients (3%) a percutaneous endoscopic gastrostomy (PEG). There was no significant difference between the three groups in the need for dilatation, endoprothesis and PEG.
Patients with adenocarcinoma had a longer survival (211 days) than those with squamous carcinoma (152 days, p=0.039), especially in group 2.
Complications (stricture 30%, mild ulcerations 20%, fistula 0%) were independent of the dose of radiotherapy.

Conclusion: HDRIR gives good palliative results by restoring food passage. Repeated dilatations can often be avoided. It is a safe and well tolerated procedure.

T96

EARLY EXPERIENCE WITH BOTULINUM TOXIN IN THE TREATMENT OF ACHALASIA.

RRSH Greaves, HE Mulcahy, SE Patchett, PD Fairclough, EM Alstead, MJG Farthing. Digestive Diseases Research Centre, St.Bartholomew's and the Royal London School of Medicine and Dentistry, London.

The ideal treatment of achalasia should be effective, safe and long-lasting. Recent reports have suggested that intra-sphincteric injection of botulinum toxin fulfills at least some of these criteria. Its efficacy is reported to be about 70% compared to placebo, no causes of oesophageal perforation have been reported, and benefits are maintained for up to one year. We report our experience of botulinum toxin injection in a prospective series of unselected patients with achalasia.

Methods: Eleven consecutive patients with achalasia (8 male, mean age 55, range 20-87) were treated with 60 units of Botulinum toxin (Dysport®Porton Products Ltd.) into each of 4 quadrants at the lower oesophageal sphincter using a 5mm sclerotherapy needle under direct vision. Diagnosis was based on clinical, radiological and manometric criteria. Patients were assessed pre-treatment and 1 month after treatment using a symptom score (dysphagia, regurgitation and chest pain, each scored on a 0-3 scale with 3=every meal, 2=daily, 1=occasional) and oesophageal manometry. Median follow-up was 12 months (range 6-28).

Results: The injection procedure was simple to perform and free of adverse effects. Although treatment had a beneficial effect on dysphagia (median pre-treatment score 3 [inter-quartile range 3-3]; post-treatment score 2 [0-3]: p=0.03) one month following therapy, there was no significant improvement in chest pain or regurgitation scores. Similarly, no significant reduction in median lower oesophageal sphincter pressure was observed (29.5mmHg [21-42] pre-treatment, 28.5 [17.5-55.5] post treatment p=0.67). Furthermore 4 patients (36%) required further therapy within 3 months and the overall relapse rate was 64% (7/11) after 2 years. 1 patient refused further therapy despite apparent lack of improvement.

Conclusion: Although botulinum toxin injection has potential advantages over established treatment modalities in terms of safety and tolerance, this study questions its efficacy as a promising treatment for achalasia.