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SALIVARY HUMAN EPIDERMAL GROWTH FACTOR (hEGF) IN NORMALS AND PATIENTS WITH PEPTIC ULCER DISEASE. *David M. Maccini, M.D., and Bruce C. Veit, Ph.D.*, William Beaumont Army Medical Center, El Paso, TX.

Epidermal growth factor inhibits gastric acid secretion and has a cytoprotective effect on the upper gastrointestinal tract. This study was undertaken to determine whether patients with endoscopically proven active peptic ulcer disease have a salivary deficiency of human epidermal growth factor (hEGF) when compared to patients with a normal esophagogastroduodenoscopy (EGD).

Saliva was collected from fasting subjects prior to EGD. The levels of EGF were measured by radioimmunoassay. Statistical evaluation was performed by analysis of variant followed by Student's t-test.

The concentrations of the peptide were lower in patients with active peptic ulcer disease (3.1 ± .54ng/ml, mean ± SE, n=25) compared with normal subjects (4.9 ± .56ng/ml, n=58, p<0.03). No significant differences in salivary hEGF were noted between patients with a normal EGD and patients with gastritis (3.85 ± .86ng/ml, n=13), esophagitis (4.5 ± 1.3ng/ml, n=7), or Barrett's esophagus (5.3 ± 1.5ng/ml, n=6). There were no differences in the salivary levels of hEGF between males and females, or between smokers and nonsmokers. There was no correlation of hEGF levels with age.

The pathophysiologic significance of this finding is uncertain. Lower salivary hEGF may reduce one of the defensive mechanisms responsible for protecting the gastroduodenal mucosa from injury by physicochemical agents, thus contributing to ulcer development.

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PREVENTION OF ENDOTOXIN-INDUCED GASTRIC INJURY BY MISOPROSTOL M. Mahatma, M.D., R. V. Yagci, M.D., J. Sitton, BS, M. Sbeiti, M.D., S. Hoda, M.D., S. Nelson, M.D., N. Agrawal, M.D., FACG. Tulane University School of Medicine, New Orleans, La. 70112.

Endotoxin associated tissue injury, including polymorphonuclear (PMN) cell infiltration, is mediated by tumor necrosis factor (TNF). We have previously demonstrated that the prostaglandin E_1 analog, misoprostol, is a potent inhibitor of endotoxin-induced TNF by systemic mononuclear phagocytes (Clinical research 38 (1) : 351, 1990). Since TNF is implicated as an important mediator of tissue injury in septic shock, we investigated the effect of misoprostol on endotoxin-induced gastric lesions.

Methods: Four groups of rats (n=6-8) were pretreated with intragastric (1) saline; (2) misoprostol 200 mcg/kg; (3) misoprostol 100 mcg/kg; (4) Maalox plus (2 ml) 15 minutes before intravenous endotoxin (E. coli lipopolysaccharide) 5 mg/kg. After 270 minutes the animals were sacrificed and stomachs were removed for inspection and histologic examination. PMN cells were counted in 5 high power fields (40X obj.) in the mid-body of stomach by a pathologist blinded to the study group. In a separate experiment serum TNF levels were measured at 90 and 270 minutes after IV endotoxin 5 mg/kg (n=7) to demonstrate rapid clearance of TNF from serum. TNF levels were measured by the L929 cytotoxicity assay and verified by neutralization of cell lytic activity by addition of rabbit antibody against murine recombinant TNF.

	PMN INFILTRATION (±SEM) / HPF (40X OBJ.) MUCOSA	SUBMUCOSA	N
Saline	88.5 ± 9.9	31.0 ± 6.0	6
Misoprostol 200 mcg/kg	19.5 ± 2.4**	7.5 ± 2.2*	7
Misoprostol 100 mcg/kg	45.1 ± 5.6*	4.3 ± 1.2*	6
Maalox Plus	79.5 ±11.1 NS	29.8 ± 8.5 NS	6

**P<0.001, *P<0.01 compared with saline.

Results: The serum TNF levels at 90 and 270 minutes were 32,432 ± 5,900 and 1,772 ± 751 u/ml, respectively, in the endotoxin treated group. TNF is not detected in serum of rats not challenged with endotoxin. Endotoxin induced diffuse mucosal edema, vascular congestion, mucosal and submucosal inflammation in all animals. No gross macroscopic lesions or hemmorhage was observed. Misoprostol in doses of 100 and 200 mcg/kg significantly attenuated the edema, vascular congestion and acute inflammation induced by endotoxin (P<0.01). These lesions were not prevented by antacid.

Conclusion: This study suggests that misoprostol significantly attenuates (P<0.01) endotoxin-induced gastric injury by inhibiting TNF release from mononuclear phagocyte induced injury.

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DOES ERADICATION OF H.PYLORI HAVE A LONG-TERM BENEFIT IN PATIENTS WITH DYSPEPSIA AND GASTRITIS? Barry Marshall, MD, FACG, Susie Hoffman, RN, Cheryl Boyd, MS, Richard McCallum, MD, FACG. Department of Internal Medicine, University of Virginia (UVA), Charlottesville, VA 22908.

INTRODUCTION: Eradication of H.pylori (HP) may be achieved with bismuth subsalicylate and antibiotic combinations. **AIM**: To assess the long term clinical outcome of such therapy on patients with gastritis. **METHODS**: 244 patients attending the UVA Dyspepsia clinic since 1987 received a clinical questionnaire prior to upper GI endoscopy and biopsy for HP. Infected patients were offered therapy for HP and were followed up by ^{14}C-urea breath test and/or endoscopy 4 weeks post-therapy. Overall, eradication was achieved in 85% of treated patients. All patients were mailed a second questionnaire in 1990. **RESULTS**: 118 (48%) responded. 14 of these (all HP positive) had an actual peptic ulcer in addition to gastritis at baseline. Respondents included 31 who never had HP (HP0), 21 who still had HP (HP1) and 63 in whom HP had been eradicated (HP2). The mean follow-up time was 21 months from baseline (initial presentation), or in group HP2, 15 months after eradication was first documented. The symptom data are summarized in the table below:

	HP0	HP1	HP2*	
Total N	23	17	62	
Much better or cured %	22	18	52	
Slightly better %	48	29	35	
Unchanged %	26	35	11	
Slightly worse %	0	12	0	*P<0.006
Much worse %	4	6	2	missing = 13

CONCLUSIONS: In patients with dyspepsia and histologic HP positive gastritis, total symptom relief and/or improvement occurs in 87% following successful eradication of HP. Such treatment has no long term harmful effects. Patients failing HP eradication therapy do not obtain symptomatic relief from standard therapeutic measures, whereas HP negative patients with dyspepsia seem to benefit. A long-term double-blind study is required to confirm these findings.

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PEG REMOVAL AND REPLACEMENT: TO SCOPE OR NOT TO SCOPE. J.C.Meeroff, M.D., F.A.C.G. and M.Raybeck, M.D., Fort Lauderdale, Florida.

Between 5/89 and 4/90 we have removed under direct endoscopic observation 22 original PEG devices (18 Sacks-Vine and 4 Ponsky-Gauderer original devices). In 15 cases PEG replacements were placed. In 5 cases endoscopy demonstrated stoma ulcerations which preclude replacement at the time and in 1 case PEG was no longer necessary. Removal of original PEG devices was successful in all 22 cases. In 3 cases(14 %) minor complications occurred (dislodgement of the T-bar or mucosal entrapment of the mushroom head). Replacement was always attempted with dome-type devices and completed in 7 cases with dome-type devices ("button") and in 9 cases with balloon type devices. Dome type devices were not always successfully introduced because fistulous tract were too long not allowing mushroom heads to enter the stomach. Either longer dome devices or balloon type devices were used in those cases. None of the problems encountered during removal/replacement of PEG devices could have been solved without endoscopic visualization.

Conclusion: PEG devices can not be removed and/or replaced blindly. Endoscopy allows direct observation of the events occurring in the stomach during the procedure leading to safe and correct completion of the procedure. Cost efficiency means not only gross dollars saved initially but most importantly maintaining adequate quality of care and avoidance of unnecessary risks and future complications.