Effect of Gum Chewing on Gastric Luminal pH in Human

Dr. S. KAPICIOĞLU, Dr. Ş. TARLAN, Dr. N. KAYA, Dr. C. DEMİRCAN

Özet: *SAKIZ ÇİĞNEMENİN MİDE PH ÜZERİNE ETKİSİ.*

Sakız çiğneme tükrük bezinin sekresyonunu 4-10 misli artırmaktadır. Bu nedenle sakız çiğneme mide PH'sının regülasyonunda önemli bir rol oynayabilir. Bu çalışma sakız çiğneme ile mide PH'daki değişikliklerini incelemeyi amaçlamaktadır.

Çalışmaya katılan 20 gönüllüye intragastrik PH tespiti için PH metre elektrodu yutturuldu. Gönüllülerden kontrol grubu (n. 10) hiç bir şey çiğnemezken diğer 10 kişi saat 9 - 14 arası devamlı sakız çiğnediler ve her 15 dakikada bir PH ölçümleri yapıldı. Sakız çiğneme mide PH'sını önemli derecede artırdı. Sakız çiğneyenlerde kontrole göre saat 10'da %27, 11'de %27.8, 12'de %28.9, 13'de %41 ve 14'de %20.9 daha yüksek PH ölçüldü. Sakız çiğneyen grupta PH değerleri saat 10.30 da kontrol grubu değerlerine düştü, ancak tekrar yükseldi.

Bu sonuçlar sakız çiğnemenin mide PH'sını artırdığını ve asite bağlı mide özafagus hastalıklarının tedavisinde faydalı olabileceğini düşündürmektedir.

Anahtar kelimeler: Sakız çiğneme, mide pH

Summary: *Gum chewing is a major hysiological stimulus of salivary secretion. With stimulation, salivary output increases 4-10 times over resting flow rate. Maximal salivary flow rates is healthy subjects (200 ml/h) approche maximal gastric juice flow rates that occured during gum chewing. Therefore the gum chewing may play a role in regulating gastric pH in humans. The aim of this study to demonstrate the gastric acidification by measurement of gastric luminal pH values during with or withouth gum chewing.*
Gum chewing was significantly more effective in increasing gastric pH when gum chewing was started at 9.00 am until 2.00 oc'lock pm. As compared with placebo pH was increased by 27.0 % at 10 am, 27.8 % at 11 am, 28.9 % at 12 noon, 41.0% at 1.00 pm, 20.9% at 2.00 pm oc'lock.

The pH curves are almost similar with placebo and with gum chewing groups. The pH difference between two groups began after gum chewing. In chewing group pH decreased araund 11 oc'lock am, until placebo value, then increased to hight level again.

In conclusion the gum chewing stimulates salivary secretion and increased gastric luminal pH values. The results may be benefit to prevent gastroesophagial peptic disease.

Key words: Chewing gum, gastric pH, human.

Considerable evidence now demonstrates thatsalive and its components have multiple funcitons in the gastrointestinal (GI) tract. Saliva acids in bolus formation; it lubricates, protects and cleanses the pharyngeal and eosophageal1. Saliwary bicarbonate buffers eosophageal acid in common reflux2-6. Normal salivary flow decreases the duration of acid contact with eosophageal mucosa, an important factor in the development of gastroeosophageal reflux disease (GERD)2,4. If salivary flow is depressed or if the eosophagosalivary reflex is lost, a patient may

Table I: The mean of gastric luminal pH values for hours.

Time(hour)	pH values Mean ± SEM		Difference between two groups (%)
	Control group	Gum chewing	
9.00	1.88 ± 0.53	1.72 ± 0.56	-8.5
9.30	2.00 ± 0.66	2.55 ± 0.69	+21.6
10.00	2.17 ± 0.80	2.97 ± 0.88	+27.0
10.30	2.28 ± 0.60	2.42 ± 0.66	+5.8
11.00	2.42 ± 0.47	3.35 ± 0.81	+27.8
11.30	2.11 ± 0.40	3.03 ± 0.73	+30.4
12.00	1.82 ± 0.46	2.56 ± 0.61	+28.9
12.30	1.88 ± 0.53	2.28 ± 0.58	+17.5
13.00	1.84 ± 0.46	3.12 ± 0.79	+41.0
13.30	1.75 ± 0.40	3.00 ± 0.77	+42.0
14.00	2.08 ± 0.53	2.63 ± 0.72	+20.9

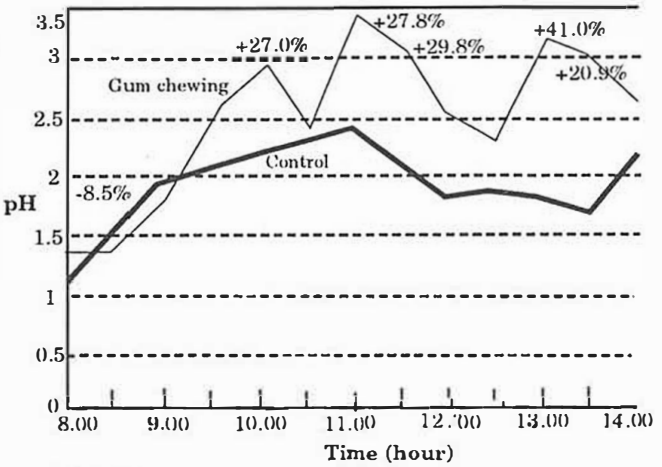

Figure 1

be predisposed to develop GERD2,7,8. Salivary epidermal growth factor (EGF) stimulates GI mucosal proliferation via a direct luminal effect in the eosophagus and stomach9-14. The salivary enzymes lingual lipase (LL) and salivary amylase initiate fat and starck digestion15-18. They are particularly significant in patients with pancreatic insufficiency such as neonates and patients with cystic fibrosis17,19.

Gum chewing is a major physiological stimulus of salivary secretion. Maximal salivary flow rates in healthy subjects (200 ml/h) approche maximal gastric juice flow rates that occured during gum chewing 20. The saliva play a minor role in regulating postprandial gastric acidity in humans, although saliva may play a more major role in regulating gastric acidity in ruminants 21. This does not exlude a role for human saliva in clearing much smaller amounts of gastric luminal pH value.

Although, there are many studies about the gum chewing, which is a potent stimulus of salivary secretion via the cholinergic parasympathetic nervous system 22-24 would also be potent stimulus of cholinergic vagal gastric secretion 5,25-28, there is little information on effect of regulation in gastric luminal pH in human 20.

nunaim of this study to demonstrate the gastric asidification by measurement of gastric luminal PH values during with or withouth gum chewing.

MATERIAL ve METHODS

Twenty healthy adult male volunteers participated in these experiments. Their age range were 20 ± 2 years. Subject had no history of smoking or of taking non-steroidal anti-inflammatory analgesics, or antisecretory use. They ingested no medication for at least 15 days prior to enrollment in the study. Before entrance in the study, each individual had a medical history taken and physical examination performed. All had normal biochemical and hematological values.

Intragastric acidity was monitored continuously using intragastric combined glass electrods which allow accurate and reproducible measurements 29.

The experiment started at 7 am and lasted at 2 pm. The volunteers were admitted to a special ward. They were fasted since midnight. The electrodes were passed transnasally at 7 am and positioned in the gastric corpus under fluoroscopic control. The typ of the glass electrode was situated 8-10 cm below the cardia. Measurement began at 9 am. All subjects were stayed in the bed during the study.

RESULTS

The mean of luminal pH values in shown in the figure and table 1. Gum chewing was significantly more effective in increasing gastric pH when gum chewing was started at 9 am until 2pm. As compared with placebo pH was increased by 27.0% at 10 h am 27.8% at 11 h am, 28.9% at 12 h am, 41.0% at 1 h pm 20.9% at 2 h pm. The pH curves as shown in the figure 1 are almost similar with placebo and with gum chewing groups.

The pH difference between two groups began after gum chewing, In chewing group pH decreased around 11 o'clock am, until placebo value, then increased to hight level again.

DISCUSSION

In this study mean gastric luminal pH increased 4-10 fold above basal pH in response to gum chewing for 4 hours. There are many studies about the gum chewing, which is a potent stimulus of salivary secretion via the cholinergic parasympathetic nervous system 22-24 would also be a potent stimulus of cholinergic vagal gastric secretion25-28.

Saliva is the watery fluid that normal bathes the oral cavity, pharynx and eosephagus with a variety of ions minerals and proteins.

The acinar cells of salivary glands transport water via solute-slovent compling with NaCI30. The ductal system resorbs electrolytes, secretes proteins and carries saliva to the oral cavity.

Many physical stimuli are capable of enhancing salivation. The smell and taste of food are the most familiar25-28. The acts of suckling and chewing are also effective stimuli. With stimulation, salivary output increases 4-10 times over resting flow rate20.

Up to 1.5 dl of saliva are produced and swallowed per day20,28. The secretion of saliva follows a circadian rhythm which correlates with feeding31. Salivary flow rises during the day to a mid-afternoon peak, then decreases to near zero-during sleep.

The buffering ability of saliva is supplied primarily by bicarbonate5 and secondarily by proteins and phosphates. The capacity for acid neutralization of saliva is directly related to its bicarbonate content5. Increased salivary flow results in increased bicarbonate concentration and therefore increased acid neutralization. The weak bases in normal saliva are sufficient to neutralize small volume of refluxed acid6.

There are at least three theoretical mechanisms by which saliva secreted during gum chewing increase gastric luminal pH. First, the volume of saliva produced during gum chewing could be suffuciently large to dilute hydrogen ions in gastric juice, reducing hydrogen ion concentration32. Second, saliva contains bicarbonate ions and proteins that could neutralize or buffer hydrogen ions5,33. Although basal saliva contains ~ 5 mmol bicarbonati/liter this increases with salivary stimulation to 30 mmol bicarbonate liter or more5,27. In vitro studies have shown that 1 ml of basal saliva can neutralize µ13 mol hydrogen ion (titrating 0.1 N HCI to pH 4.0) and that 1 ml of bethanechol-stimulated saliva can neutralize 22 mol hydrogen ion. Therefore, if a sufficient volume of alkaline saliva is swallowed, it is possible that a detectable decrease in intragastric acidity could be demonstrated in vivo.

A third mechanism by which saliva could reduce acidity may be an inhibitor of gastric acid secretion contained in saliva. Extracts of salivary gland and saliva reduce gastric acid secretion when injected parenterally into animals34,35. Recent studies have suggested that the inhibitor of acid secretion in human salivary glands and saliva is urogastrone, a peptide closely hemologous with or indentical to epidermal growth factor (EGF)36-38. Although parenteral administration of urogastrone reduces gastric acid secretion in human39,40. It was unclear if this peptide could inhibit acid secretion when swallowed as a normal sonstituent of saliva41,42.

Numerous studies in rats have demonstrated that EGF protects the gastrointestinal mucosa from chemical injury. Animals that had the salivary glands resected were susceptible to a vari-

ety of experimentally induced gastric ulcers than control9,43-48. Epidermal growth factor is also the active cytoprotective factor in saliva9 and that his protective or proliferative effect was not due to decreased gastric acid secretion9-11. The cytoprotective action of EGF may also be related to the regulation of the mucus coat thickness and composition. But human studies of the role of salivary EGF in gastric cytoprotection show little agrement49,50.

In this study gastric luminal pH were significantly different when saliva was expectrorated by gum chewing. Our results suggest that salive plays a major rol in regulating gastric acidity in humans, although saliva may play a role in regulating gastric acidity in ruminants21.

In conclusion, the gum chewing stimulates salivary secretion and increases gastric luminal pH values. These results may be benefit to prevent gastroeosophageal peptic disease.

REFERENCES

1. Hughes CV, Baum BJ, Fox PC, et al Oral-pharyngeal dysphagia: A common sequelae of salivary gland dysfunction. Dysphagia 1987; 173-177.
2. Helm JF. Role of saliva in esophageal function and disease. Dysphagia 1989; 4: 76-84.
3. Helm JF, Dodds WJ, Pelc LR, et al. Effect of esophageal emptying and saliva on clearance of acid from the esophagus. N Engl J Med 1984; 310:284-288.
4. Dodds WJ, Hogan WJ, Helm JF, et al. Pathogenesis reflux esophagitis. Gastroenterology 1981;81:376-394.
5. Helm JF, Dodds WJ, Hogan WJ, et al. Acid-neutralizing capacity of human saliva. Gastroenterology 1982; 83: 69-74.
6. Helm JF, Dodds WJ, Riedel DR, et al. Determinants of esophageal acid clearance in normal subjects. Gastroenterology 1983; 85: 607-612.
7. Best CH, Taylor NB. The physiological basis of medical practice. Baltimore, Williams & Wilkins, 1961, pp 585–598.
8. Helm JF, Dodds WJ, Hogan WJ. Salivary response to esophageal acid in normal subjects and patients with reflux esophagitis. Gastroenterology 1987; 93: 1393-1397.
9. Olsen PS, Poulsen SS, Therkelsen K, et al. Effect of sialoadenectomy and synthetic human urogastrone on healing of chronic gastric ulcers in rats. Gut 1986; 27: 1443-1449.
10. Olsen PS. Role of epidermal growth factor in gastroduodenal mucosal protection. J Clin Gastroenterol 1988; 10(suppl): S146-S151.
11. Konturek SJ. Role of epidermal growth factor ingastroprotection and ulcer healing. Scand J Gastroenterol 1988; 23: 129-133.
12. Sarosiek J, Bilski J, Murty VL, et al. Role of salivary epidermal growth factor in the maintenance of physicochemical characteristics of oral and gastric mucosal mucus coat. Biochem Biophys Res Commun 1988; 152: 1421-1427.
13. Stosheck CM, King LE Jr. Role of epidermal growth factor in carcinogenesis. Cancer Res 1986; 46: 1030-1037.
14. Dutta SD, Matossian H, Hamburger A, et al.
15. Hamosh M, Burns WA. Lipolytic activity of human lingual glands (Ebner). Lab Invest 1977; 37:603.
16. Liao TH, Hamosh P, Hamosh M. Fat digestion by lingual lipase: Mechanism of lipolysis in the stomach and upper small intestine. Pediatr Res 1984; 18:402-409.
17. Ahrams CK, Hamosh M, Hubbard VS, et al. Lingual lipase in cystic fibrosis. Quantitation of enzyme activity in the upper small intestine of patients with exocrine insufficiency. J Clin Invest 1984; 73: 374-382.
18. Fink CS, Hamosh P, Hamosh M. Fat digestion in the stomach: Stability of lingual lipase in the gastric environment. Pediatr Res 1984; 18: 248-254.
19. Dutta SK, Hamosh M, Abrams CK, et al. Quantitative estimation of lingual lipase activity in the upper small intestine in adult patients with pancreatic insufficiency. Gastroenterology 1982; 82: 1047.
20. Richardson CT, Feldman M. Salivary response to food in human and its effect on gastric acid secretion. Am. J. Physiol 1986; 250: 685-691.
21. Kay RNB. The rate of flow and composition of various salivary secretions in sheep and calves. J Physiol Lond 150:515-537, 1960.
22. Feldman H, Richardson CT. Role of thought, sight, smell and taste of food in the cephalic phase of gastric acid secretion in humans. Gastroenterology 1986; 90:428-433.
23. Schneyer LH, Emmelin N. Salivary secretion. In: Jacobsen ED, Shanbour LL, eds. Gastrointestinal physiology, MTP International Review of Science, Physiology secretion, Baltimore, MD: University Park Press, 1974.
24. Makhloug GM, Blum A. Salivary expressions of taste response to chemical stimulation. Scand J Gastroenterol 1971; 6: 523-526.
25. Blum AL. Salivary secretion in duodenal ulcer disease. Gut 1972; 13: 713-717.
26. Nagwani PL, Naik SR, Sachdev S, Srivastava PN, and Chuttani HK. Correlation of salivary and gastric acid secretions in duodenal ulcer patients in tropics. Gut 1979; 20: 585-589.

27. Schneyer LH, Schneyer CA. Inorganic composition of saliva. In: Handbook of Physiology. Alimentary Canal II, edited by Code CF. Washington, Dc: Am Physiol Soc 1967; chapt 33, p 497-530.

28. Thaysen JH, Thorn NA, Schwartz IL. Excretion of sodium, potassium, chloride and carbon dioxide in human parotid saliva. Am J Physiol 1954; 178: 155-159.

29. Etienne A, Fimmel CJ, Bron BA, Loizeau E, Blum AL. Evaluation of pirenzepin on gastric acidity in healthy volunteers using ambulatory 24 h intragastric pH monitoring. Gut 1985;26: 241-245.

30. Izutsu TK. Salivary electrolytes and fluid production in health and disease; in Sreebny LM (ed): The Salivary System. Boca Raton, CRC Press 1987; pp 95-122.

31. Dawes C. Circadian rhythms in human salivary flow rate and composition. J Physiol 1972; 220: 529-545.

32. Malhotra SL. Protective action of saliva in peptic ulceration. Studies on the effect of saliva on gastric secreti on with dye-dilution technique. Scand J Gastroenterol 1967; 2: 95-104.

33. Okosdinossian LT, El Munshid HA. Composition of the alkaline component of human gastric juice: effect of swallowed saliva and duodenogastric reflux. Scand J Gastroenterol 1977; 12: 945-950.

34. Menguy R, Berlinski M. Source of sialogastrone, a gastric inhibitory substance in human saliva. Am J Dig Dis 1967; 12: 1-6.

35. Menguy R, Masters YF, Gryboski W. Content of gastric inhibitory substance in saliva of patients with various gastric disorders. Gastroenteroloy 1964; 46: 32-35.

36. Elder JB, Williams G, Lacey E, Gregory H. Cellular localization of human urogastrone/epidermal growth factor. Nature Lond 1978; 271: 466-467.

37. Heitz PU, Kasper M, Van Noorden S, Polak JM, Gregory H, Pearse AGE. Immunohistochemical localization of urogastrone to human duodenal and submandibular glands. Gut 1978; 19: 408-413.

38. Hirata Y, Orth DN. Epidermal growth factor (urogastrone) in human tissues. J Clin Endocrinol Metab 1979; 48: 667-672.

39. Feldman M. Inhibition of gastric acid secretion by selective and nonselective anticholinergics. Gastroenterology 1984; 86: 361-366.

40. Feldman EJ, Aures D, Grossman MI. Epidermal growth factor stimulates ornithine decarboxylase activity in the digestive tract of mouse. Proc Soc Exp Biol Med 1978; 159: 400-402.

41. Gregory H, Walsh S, Hopkins CR. The identification of urogastrone in serum, saliva, and gastric juice. Gastroenterology 1979; 77: 313-318.

42. Hirata Y, Orth DN. Epidermal growth factor (urogastrone) in human fluids: size heterogeneity. J Clin Endocrinol metab 1979; 48: 673-679.

43. Konturek SJ, Brzozowski T, Piastucki I, et al. Role of mucosal prostaglandins and DNA synthesis in gastric cytoprotection by luminal epidermal growth factor. Gut 1981; 22: 927-932.

44. Majundar APN, Arlow FI. Aging: Altered responsiveness of gastric mucosa to epidermal growth factor Gastrointest Liver Physiol 1989; 20: G-554-G560.

45. Poulsen SS, Olsen PS, Kirkegaard P. Healing of cysteamine-induced duodenal ulcers in the rat. Dig Dis Sci 1985; 30-161-167.

46. Itoh M, John T, Imai S, et al. Experimental and clinical studies on epidermal growth factor for gastric mucosal protection and healing of gastric ulcers. J Clin Gastroenterol 1988; 10 (suppl 1): S7-S12.

47. Sakamoto T, Swierczek JS, Ogden WD, el al. Cytoprotective effect of pentagastrin and epidermal growth factor on stress ulcer formation. Ann Surg 1985; 201-290-295.

48. Olsen PS, Poulsen SS, Kirkegaard P, et al. Role of submandibular saliva and epidermal growth factor in gastric cyt protection. Gastroenterology 1984; 87: 103-108.

49. Ohmura E, Emoto N, Tsushima T, et al. Salivary immunoreactive human epidermal growth factor (IR-hEGF) in patients with peptic ulcer disease. Hepatogastroenterology 1987; 34: 160-163.

50. Richardson CT, Feldman M. Basal and shamfeeding-stimulated salivary flow in duodenal ulcer patients and healthy subjects. Scand J Gastroenterol 1988; 23: 765-768.