

Diet and Reflux

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Abstract: Despite lifestyle modifications are proposed as common therapy in gastroesophageal reflux disease, there is few evidence for an improvement of gastroesophageal reflux disease after dietary interventions. We conducted a systematic review of available studies evaluating the association of dietary factors with reflux symptoms evaluated in series and population, pH monitoring and esophageal manometry. Data are presented for fat, volume of meal, osmolality, thickening, carbohydrates, fiber, protein, coffee, chocolate, tea, mint, carbonated beverages, spicy food, and onions. Although, fat foods are considered as a putative deleterious factor, none of these factors is associated with an unequivocal protective or deleterious effect.

Key Words: GERD, diet, fat, pH-metry, manometry

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Speculation for a role of nutritional factors in gastroesophageal reflux disease (GERD) existed since many years.^{1,2} Despite lifestyle modifications are proposed as common therapy in this condition,³ there is few evidence for an improvement of GERD after dietary interventions. We conducted a systematic review of available studies evaluating the association of dietary factors with reflux symptoms evaluated in series and population, pH monitoring, and esophageal manometry.

FAT

Fatty foods are commonly reported to aggravate GER symptoms. Because the high-fat content of a meal was thought to be responsible for reflux, patients with reflux disease are advised to avoid high-fat meals. Nevertheless, studies to determine whether high-fat meals promote GER have produced conflicting conclusions and putative mechanisms remain discussed.⁴ A recent review of the literature shows that no controlled trial supports the effectiveness of such a recommendation.⁵

Few epidemiologic studies supporting the deleterious effect of fat are available. Fatty food was reported to be associated with heartburn in 2000 individuals with

GER.³ Greasy/rich foods was reported to be associated with reflux symptoms in a telephone survey including 1200 individuals aged 18 years or more with 37% of them reporting heartburn at least once every 4 to 6 months.⁶ Daily intakes of total fat, saturated fat, cholesterol, percentage of energy from dietary fat, and average fat servings were significantly higher in healthy employees at a Houston medical center with GERD symptoms than in those without. In addition, there was a dose-response relationship between GERD and saturated fat and cholesterol. The effect of dietary fat became nonsignificant when adjusted for body mass index (BMI). High-saturated fat, cholesterol, or fat servings were associated with GERD symptoms only in participants with a BMI > 25 kg/m².⁷

At the contrary, population-based, case-controlled studies from Sweden and China also failed to detect any association between fat and symptoms of reflux disease.^{8,9} Total calories from fat was not associated with reflux in a cross-sectional survey in 211 community subjects using validated questionnaires on reflux, energy expenditure (Harvard Alumni Activity Survey), and dietary intake (Harvard Food Frequency Questionnaire).¹⁰

These results are consistent with the First National Health and Nutrition Examination Survey (NHANES I) epidemiologic study of 12,349 persons who were followed for a median of 18.5 years. Although higher reflux disease hospitalization rates were associated with an increase BMI, this was not associated with a higher fat intake.¹¹ However, hospitalization could be an inadequate marker for reflux in a population survey and some putative confounding factors are not analyzed.

Individuals with GERD in these studies may have avoided ingesting certain foods associated with precipitating symptoms or may have increased their intake of other foods that ameliorate these symptoms. Neither the degree nor the pattern of dietary change is known. This potential bias is likely to have lowered the observed significant association between high-dietary fat intake and GERD as it is highly unlikely that high-fat foods were used to ameliorate GERD-related symptoms. Although an observed increase in any particular dietary item may reflect a higher overall caloric intake, effects for dietary fat were independent of the total caloric intake.

Early studies attempted to find a rationale for reduced fat regimens by measuring postprandial esophageal pH after a fatty meal in normal subjects and patients with GERD. Upright but not recumbent acid exposure was increased during a 3-hour postprandial period in 10 healthy volunteers after a high-fat meal (40 g) when

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compared with a low-fat meal (11 g) with identical protein content, volumes, and calories.¹² In this study, no differences in esophageal reflux were found between low-fat and high-fat meals in 10 GER patients.¹² The median number of reflux episodes increased from 0 after a moderately high fat (32 g) meal given at 19:30 hours to 7 after a late-evening very high-fat (72 g) meal, including peppermint and chocolate in 8 healthy volunteers. Persistent nocturnal reflux was observed only after the high-fat meal. Between 23 hours and 7 hours, when subjects were supine, the median acid exposure time pH less than 4 increased from 0% after the standardized meal to 7.9% after the high-fat meal.¹³ However, these differences cannot be attributed solely to the effects of high fat, because the high fat meal also contained chocolate that was not present in the lower fat meal and was much more caloric. Furthermore, the authors studied reflux at a different time of day and during longer recumbency after the high-fat meal. More esophageal reflux was observed after a high-fat (70%) versus low-fat (16%) liquid meal during a 3-hour recumbent postprandial period in 20 asymptomatic healthy volunteers. This difference was not observed in the upright position. This observation was confounded by differences in caloric contents of the 2 meals and by failure to randomize the test sequences.¹⁴ Acid reflux episodes and duration of pH less than 4 were increased after a fat meal than after a standard meal in 8 patients with reflux.¹⁵

High-fat (52% or 44 g fat) meal did not evoke more reflux than a low-fat (24% or 20 g fat) isocaloric (3.18 MJ) meal when evaluated during 3 hours in either 13 healthy subjects and 14 patients with reflux disease in the recumbent or the sitting position.¹⁶ No difference was observed according to the time at pH less than 4 between an hamburger meal (34 g fat) and a sausage biscuit meal (60 g fat) in 12 patients with GERD.¹⁷ The frequency of reflux episodes and the fraction time pH less than 4 were not different in 12 healthy volunteers received an isocaloric isovolumetric solid-liquid meal with either a low (842 kcal, 10% fat, 14% proteins, 76% carbohydrates) or a high-fat content (50% fat, 18% proteins, 32% carbohydrates) in a randomized, double-blinded fashion.¹⁸ The mean percentage of time at pH less than 4 and the mean number of reflux episodes evaluated over a longer postprandial period (6 h) after a balanced 2.8 MJ (23% fat) meal were also similar to those after an equicaloric 2.8 MJ (58% fat) high-fat meal in 13 healthy volunteers.¹⁹ The failure to confirm fat induced reflux, the large standard errors in several of the studies, the poor designs in some and the inconsistent pattern of findings lead some authors to conclude that the few positive findings among these reports could be the result of type II errors.²⁰

By contrast with the popularly held notion that fat provokes reflux by reducing basal lower esophageal sphincter pressure (LESP), fat seems to provoke reflux by increasing the incidence of reflux during transient LES relaxations (TLESRs) at least in resting recumbent patients with reflux esophagitis.

The LES response to ingestion of fat and protein meals with equivalent caloric value was first evaluated in healthy individuals. Fatty meals decreased LESP significantly compared with protein meals.²¹ In another study, the amount of dietary fat necessary to reduce the LESP seems to be rather low, because whole milk (3.3% fat) but not nonfat milk was reported to reduce LESP in healthy volunteers.²² No significant difference in the frequency and duration of TLESRs was observed in 8 patients with GERD after 1 hour after fat meal when compared with standard meal but the frequency of TLESRs evaluated for 2 hours increased after the fat meal.¹⁵

LESP and esophageal pressures were recorded for 3 hours after isocaloric (3.18 MJ) high-fat (52% or 44 g fat) and low-fat (24% or 20 g fat) meals on 2 different occasions, some in the recumbent position and the others in the sitting position. The rate of TLESRs, their association with reflux and basal LESP were unaffected in either group regardless of body posture.¹⁶ The mean LESP, the frequency of TLESRs with or without GER were not different between isocaloric, isovolumetric solid-liquid meal with either a low-fat (842 kcal, 10% fat, 14% proteins, 76% carbohydrates) or a high-fat content (50% fat, 18% proteins, 32% carbohydrates) administered in 12 healthy volunteers in a randomized, double-blinded fashion.¹⁸

Instillation of fat directly into the duodenum aggravates reflux in patients with reflux disease, by increasing the proportion of TLESRs accompanied by reflux. Indeed, more GER in 11 GERD patients (but not in 12 asymptomatic controls) in the right lateral decubitus position when the duodenum was perfused using a cross-over study design with 10% intralipid (vs. saline) at a duodenal load of 6 g/h of fat. This effect was due to an increase in the incidence of reflux during TLESRs (65% vs. 91%), the rate of transient relaxations remaining unchanged. Duodenal fat decreased basal LESP from 16.9 to 12.4 mm Hg in normal subjects but had no effect in patients with esophagitis 18.8 versus 18.2 mm Hg.²³ These findings of increased reflux in patients with reflux disease but not in normal subjects differ from the earlier results. This disparity might, in part, be the result of the different methods used. Reflux was measured for only 30 minutes during the duodenal stimulus. This may have been insufficient time to detect an effect on relatively infrequent events in the normal subjects. The fat load to the duodenum used virtually 100% triglyceride compared with no fat at all on the control day. This contrasts with 16% to 65% load used in other studies. Then, fat increased the likelihood of reflux occurring during TLESRs. The mechanisms underlying this effect are not clear. It is possible that fat induced relaxation of the proximal stomach leads to increased pooling of liquid in the proximal stomach and thereby a larger reservoir of refluxate.

Fat in the small intestinal lumen intensified the perception of heartburn. Indeed, under the uniform stimuli of controlled acidifications of the esophagus across the range of dose-responses, physiologic loads of

duodenal fat (8 g/h) shortened the latency to onset of typical heartburn and increased the intensity and severity of heartburn in 11 fasting patients who had GERD.²⁴ The load of duodenal fat was less than the maximal loads that normally enter the duodenum during the first hour of a high-fat meal and would be about equal to loads entering in the first hour after a meal that might contain 40 g of fat.

How fatty meals intensify heartburn in subjects who suffer from pyrosis is poorly understood. Fat in the intestinal lumen is increasingly recognized to alter sensations that originate from the gastrointestinal tract. Fat enhances the perception of fullness (satiety), a response that originates largely at the level of the gut wall. Duodenally perfused fat also altered the discomfort threshold during gastric distensions with a balloon and changed the quality of sensation. In addition to these effects on visceral sensation, ingestion of fat has been shown to decrease sensitivity to cutaneous pain but this last effect does not seem to depend on the stimulation of sensors in the gut wall.

VOLUME OF MEAL, CALORIC LOAD, AND OSMOLALITY

Few adequate studies investigating whether voluminous meals do indeed cause more reflux are available. Then, on the basis of the criteria of evidence-based medicine, it cannot be concluded that the patients should be told to avoid voluminous meals and to eat smaller portions more often.²⁵ However, as it is pathophysiologically convincing, it might be reasonable to recommend that large meals be avoided.

A relationship between the amount of postprandial GER and the volume of a liquid meal has been suggested. Differences in the meal volume (500-mL low-fat meal vs. 800-mL low-fat meal) influenced GER evaluated during the 3-hour postprandial period in 20 healthy asymptomatic subjects.¹⁴

Postprandial esophageal acid exposure was significantly higher in 30 children with GERD after a high-volume meal than after a standard meal.²⁶ In this study, a higher postfeeding rate of reflux episodes caused by TLESRs was detected after the high-volume meal. The degree of gastric distension after a meal with high volume significantly correlated with the rate of GER episodes caused by low/absent LESP both at the first and the second postfeeding hour.²⁶ A prolonged hypotonia of the LES could allow larger volumes of intragastric content to reflux into the esophagus than phasic relaxations of the LES: this might explain the higher postfeeding esophageal acid exposure in patients on high-volume and osmolality meal than in the other groups of patients.

Few studies evaluated the role of meal caloric load. The mean percentage of time at pH < 4 and the mean number of reflux episodes evaluated over a 6-hour postprandial period were higher after a balanced 2.8 MJ 23% fat meal than after a balanced 1.6 MJ 25% fat meal in 13 healthy volunteers.¹⁹ At the contrary, no difference was identified regarding the fraction of time for which pH

was less than 4 between high-calorie and low-calorie meal in 12 healthy volunteers who received 2 solid-liquid meals with either 842 kcal (solid 582 kcal, liquid 260 kcal) or 582 kcal (31% reduction) in a randomized order.²⁷ In this study, a similar decrease in LESP and no difference in the number of TLESRs were registered after intake of both types of meal.

Foods with high osmolality could be the cause of pain in the acid-sensitive esophagus. Six and 9 of 11 "acid-positive" subjects using a conventional Bernstein test also developed pain with hypertonic saline and sucrose (630 mOsm/kg water) but only 1 of 15 acid-negative subjects.²⁸ Osmolality of 38 beverages, determined *in vitro*, did not correlate with the amount of heartburn reported by questionnaire in 394 people with heartburn.²⁹

THICKENING OF MEAL

Thickening of formula feedings is part of the therapeutic approach for GER in infants. Thickening of formula feedings with carob bean gum has a significant effect on the reduction of regurgitation frequency and amount in otherwise healthy infants (42 d old). This effect is caused by a reduction in the number of nonacid (pH > 4) GER episodes, but also because of a decrease of mean reflux height reached in the esophagus. However, the occurrence of acid GER is not reduced.³⁰

CARBOHYDRATE AND DIETARY FIBER

Total carbohydrate intake was shown to be similar between patients with or without reflux symptoms.⁷ However, observations from some individuals suggest that carbohydrates may be a precipitating factor for GERD symptoms.³¹ Yancy et al³¹ reported a case series of 5 obese patients who experienced almost-complete resolution of their GERD symptoms within a few days of initiating a very low-carbohydrate diet. These patients noted return or worsening of their GERD-related symptoms when they returned to a normal level of carbohydrate consumption. However, this conclusion is confounded by concurrent reduction of both caffeine and acidic high-osmolal food intake.³¹ Eight obese individuals were enrolled in a very low-carbohydrate diet trial (less than 20 g/d of carbohydrates). Mean Johnson-DeMeester score, evaluated within 6 days, decreased significantly and percentage time with pH < 4 and mean symptom severity assessed using the GERD Symptom Assessment Scale-Distress Subscale score.³² In this short series, the demonstration of improved Johnson-DeMeester scores and decreased percentage total time with a pH < 4.0 lends support to a physiologic rather than a placebo effect for the improved symptoms.

High dietary fiber intake seems associated with a decrease risk of reflux. In a Norwegian population where bread is one of the dominating sources of carbohydrates, increasing dietary fiber content in the predominantly consumed bread decreased the risk of reflux.³³ People who predominantly ate bread with 7% dry weight of

dietary fibers or more, had an approximately halved risk of having reflux symptoms compared with those who ate white low-fiber content bread. Fiber intake was also inversely associated with reflux in a cross-sectional study in a sample of American volunteers. In this study, fiber intake remained inversely associated with the risk of GERD symptoms in adjusted models for BMI, energy, or demographic features.⁷ The German National Health Interview and Examination Survey also suggested that white bread could be associated with reflux symptoms despite the consumption of fruits seemed to have some protective effect.³⁴ A fiber daily intake > 17 g was also associated with a decrease risk of frequent reflux in 211 community subjects but this difference did not reach significance.¹⁰

Studies had suggested the potential mechanisms for GERD symptoms with alteration of dietary fiber intake. In the acidic environment of the stomach, large amounts of nitric oxide (NO) are produced nonenzymatically from nitrites in the diet. NO has a potent relaxing effect on the LES, and is as such likely to promote reflux. Dietary fibers are well known to scavenge nitrites in the stomach, thereby decreasing the availability of the substrate for nonenzymatic NO synthesis. This might reduce NO concentration in the gastroesophageal junction, and thus prevent reflux. In healthy volunteers, ingestion of lactose results in an increased number of TLESRs, increased reflux episodes, higher esophageal acid exposures, and more severe GERD symptoms.³⁵

Oral administration of fructo-oligosaccharides reproduced manometric and pH probe findings similar to those seen after the administration of lactose.³⁶ Fructo-oligosaccharides are generally poorly absorbed in the small intestine but completely fermented in the colon and are frequently found in many processed food products. Therefore, it has been suggested that colonic fermentation of malabsorbed carbohydrates leads to the production of short-chain fatty acids (SCFAs) that might be partially responsible for the increased GERD seen in individuals ingesting these carbohydrates. This hypothesis is enhanced by the observation that intracolonic infusion of SCFAs produces similar physiologic changes with respect to GERD as those seen with oral administration of lactose and fructo-oligosaccharides. Indeed, postprandial fall in LESP was documented after colonic infusion of SCFAs and the number of TLESRs increased after both lactose and SCFA colonic infusions.³⁵ One study that used geographic surveys suggested that dietary deficiency of fiber increases the risk of hiatus hernia.

PROTEIN

Few studies evaluated the role of dietary protein in reflux disease. Some observations indicate that whey-derived milk formulas could be more beneficial than casein-derived formulas in infants with GERD, likely because of a more rapid gastric emptying.

Protein intake was showed to be similar between subjects with or without reflux symptoms in the 371

respondents of an American cross-sectional survey.⁷ Protein meals increased LESP when compared with fatty meals in healthy individuals.²¹ The gastric emptying times associated with 3 whey-based formulas were significantly shorter than that associated with a casein-based formula in 9 gastrostomy-fed patients with spastic quadriplegia. Patients fed whey-based formulas had also significantly fewer episodes of emesis.³⁷ A prospective study assessed both the changes in the percentage of time GER was detected by scintigraphy and the gastric emptying using 3 different formulas on consecutive days in 28 infants under 1 year of age diagnosed to have GER by pH monitoring (pH being less than 4.0 for greater than 5% of the duration of the test). The patients received the same volume per single feeding of an isocaloric casein-predominant, soy, or a whey-hydrolysate formula in a randomized order. GER percent estimated for 60 minutes after these feedings was not significantly different according to the formula despite a significant difference was observed on GE between casein-predominant and whey-hydrolysate feedings.³⁸

DIETARY NITRATE

High concentrations of NO, derived from dietary nitrite in an acid environment, have been demonstrated in the gastric fundus and in the esophagus. Luminal NO can influence esophageal smooth muscle performance, LES function or gastric and esophageal acid exposure. However, dietary nitrate did not significantly affect esophageal motor or LES function, gastroesophageal acid reflux, or reflux symptomatology either in healthy volunteers or in GERD patients.³⁹

SALT

The use of table salt estimated from the frequency of meals of salted fish or meat and from how often the person added extra salt to regular meals was collected prospectively in the HUNT 1 study population from Nord-Trøndelag in Norway. A moderate and dose-dependent association between increasing frequency of meals of salted fish or meat and reflux symptoms was observed. The risk of reflux among people who ate salted food 3 times per week or more was significantly increased by 50% compared with those who never ate salted food. Similarly, the increasing use of extra table salt on regular meals was associated with an increased risk of reflux in a dose-dependent manner. The risk of reflux was 70% increased among people who always added extra salt compared with those who never did so.³³ In an American survey, sodium consumption was higher in patients with GERD symptoms than in those without and in those with esophagitis than in those without.⁷

COFFEE

Many patients with GERD report that coffee aggravates their symptoms and some doctors discourage its use in GERD, but there is insufficient evidence to support such recommendations. Coffee can be a direct

irritant to the esophagus. In 66 patients with pain of possible esophageal origin, intraesophageal infusions of coffee or HCl of varying concentrations were performed. Acid-sensitive patients were also sensitive to infusion of coffee adjusted or not adjusted to pH 7.⁴⁰ Coffee was associated with significant amounts of reported heartburn when compared with water in 2 series of 317 and 384 patients with heartburn, respectively.^{29,41} Reported aggravating factors of heartburn included coffee in a telephone survey including 1200 individuals aged 18 years or more.⁶

Consumption of caffeinated beverages was not reported to be associated with heartburn in a population-based telephone survey of 2000 individuals with heartburn.³ Epidemiologic studies from China, USA, Canada, Italy, Japan, The Netherlands, Switzerland, Denmark, Finland, Norway, Sweden, and Germany reported that there was no significant association of reflux symptoms with coffee consumption when was evaluated as yes or no, or as the number of cups drunk per day or as coffee at least weekly.^{8-10,34,42,43} Coffee consumption was also not associated with risk of reflux disease hospitalization in the NHANES I study.¹¹

The HUNT 2 study in Norway identified a moderately strong inverse relationship.³³ Indeed, the multivariate analysis of data on the use of coffee collected from the HUNT 2 survey showed was a negative association between coffee intake and reflux symptoms with an approximate 40% decrease in risk among people who drank more than seven cups of coffee per day, compared with those who drank less than 1 cup. This finding differed markedly from the univariate analysis of coffee exposure, which showed a slight increase in risk of reflux when comparing the same groups as above (odds ratio = 1.2). The analyses revealed that the increased risk of reflux identified in the univariate data could be entirely explained by confounding from tobacco smoking. Given that it is likely that coffee intake induces symptoms in some susceptible individuals, the observed risk reduction might be explained by reversed causality: if individuals with reflux symptoms abstain from drinking coffee. To minimize the influence of reversed causality, and more accurately evaluate the long-term effects of coffee drinking on the risk of reflux, an analysis of prospective exposure data would be necessary.

Different coffees induce variations in GER in coffee-sensitive individuals. A double-blind, crossover study in 20 volunteers with coffee-sensitivity showed that a coffee from the USA was associated with less symptoms than an "untreated" coffee from Europe.⁴⁴ Differences in the coffee bean roasting process did not result in marked differences in coffee-induced reflux symptoms in GERD patients.⁴⁵

Regular coffee (300 mL) induced increased GER when compared with tap water and water-containing caffeine in 8 healthy volunteers. Decaffeination of coffee significantly diminished GER.⁴⁶ Indeed, the fraction time esophageal pH less than 4 induced by the intake of regular coffee could be reduced by decaffeination as

showed in a randomized, double-blind study testing 300 mL of either regular or decaffeinated coffee together with a standardized breakfast in 17 GERD patients.⁴⁷ At the contrary, coffee as 280 mL of regular paper-filtered coffee had no effect on postprandial acid reflux time or number of reflux episodes, either in 7 GERD patients or in 8 healthy subjects when compared with 280 mL of warm water. Coffee increased the percentage acid reflux time only when ingested in the fasting period in the GERD patients but not in the healthy subjects.⁴⁸ Differences in origin and process of coffee could explain some of these discrepancies.⁴⁴

LESP showed minimal changes in response to caffeine, but was significantly increased by both regular and decaffeinated coffee in healthy volunteers.⁴⁹ At the contrary, a coffee inhibitory effect on LESF was reported⁵⁰ and caffeinated instant coffee (150 mL) at either pH 4.5 or 7.0 decreased LESF in normal volunteers and patients with reflux esophagitis.⁵¹ In more recent studies, coffee had no significant effect on LESF in neither 10 fasting healthy volunteer⁵² nor in 7 GERD patients or in 8 healthy subjects.⁴⁸

A study tested the hypothesis that differences in the processing of raw coffee beans can account for some of the variability in effects of coffee drinking. Coffees were selected to represent several ways that green coffee beans are treated: instant and ground coffee processing, decaffeination method (ethyl acetate or methylene chloride extraction), instant coffee processing temperature, and steam treatment. Consumption of coffee was followed by a sustained decrease in LESF except for 3 of the 4 coffees treated with ethyl acetate regardless of whether or not they contained caffeine.⁵³

TEA

Tea was associated with significant amounts of reported heartburn when compared with water in 384 patients with heartburn.²⁹ Tea drinking was not associated with any effect on the risk of reflux symptoms, independent of the adjustment for potential confounding variables in China, USA, Norway, and Germany.^{9,11,33,34,54} Tea drinking had no significant effect on reflux evaluated by 3-hour ambulatory pH-metry in healthy volunteers.⁴⁶

CHOCOLATE AND SWEETS

Chocolate was reported to be associated with heartburn in population-based survey in 2000 individuals with heartburn³ but lack of association between reflux symptoms and chocolate was demonstrated in a Norway survey.⁸ No studies have addressed the effect of chocolate abstinence on reflux symptoms.

Ingestion of 120 mL of chocolate syrup has been shown to decrease mean basal LESF in 6 and 9 healthy volunteers, respectively, providing a rationale for the pathogenesis of chocolate-induced reflux symptoms.^{22,55} Compared with ingestion of a dextrose control solution of similar volume, osmolality, and calories, postprandial

ingestion of chocolate resulted in a significant increase in acid exposure evaluated by intraesophageal pH monitoring in the first postprandial hour in 7 patients with esophagitis.⁵⁶ The sample sizes of these few studies are too small for definite conclusions to be drawn.

Sweets such as candy bars have been regarded as causing reflux because of their high osmolality and high-fat content.²⁵ Indeed, consumption of sweets at least once a day was a risk factor for reflux symptoms in a German survey.³⁴

CITRUS FRUITS AND JUICES

Citrus fruits and juices can be a direct irritant to the esophagus. Increased heartburn was reported after ingestion of 17 orange and grapefruit juices in a questionnaire study of 394 patients.²⁹ The authors explained this finding by a significant correlation between the titratable acidity of the juices and the heartburn score. This effect was also reported in 76% with orange, grapefruit, and tomato juices in another series of patients with heartburn.⁴¹ Heartburn was also associated with citrus fruits and juices consumption in a population-based telephone survey in 2000 individuals with heartburn.³ No difference in citrus fruits servings per week was observed between asymptomatic patients and heartburn patients in a Swedish case-control study.⁸

Fifteen out of 16 Bernstein-positive patients with pain of possible esophageal origin, were sensitive to intraesophageal infusions of orange juice, and tomato drink adjusted or not to pH 7, suggesting that acidity is not the only factor involved in the juice effect.⁴⁰ LESP measured for 60 minutes remains was decreased in a first study in 6 healthy volunteers after 240 mL orange juice (pH 3.5) ingestion.²² LESP was unchanged after the ingestion of 250 mL of orange juice in 8 patients with orange juice-induced heartburn and increased in 9 healthy volunteers in another study.⁵⁷

CARBONATED BEVERAGES

Carbonated beverages were reported to be associated with reflux symptoms in 26% to 52% of patients with heartburn.^{3,29,41} Carbonated soft drink consumption was a predictor of heartburn (odds ratio = 1.3) during sleep in 3806 subjects who participated in a multicenter, longitudinal cohort study of the cardiovascular consequences of sleep-disordered breathing and reported having this symptom.⁵⁸ However, in this study, only few nutritional items were analyzed as putative confounder factors.

Carbonated beverages are associated with an increase in TLESRs or a reduction in the LESP. In a first study, no difference in this reduction was found between carbonated water, caffeine-free Pepsi, or regular Pepsi.^{59,60} The authors concluded that the changes in the LES are due to the gas rather than to other properties such as the caffeine level or pH. Resting pressure, overall length, and abdominal length of the LES were further measured by the same group using a slow motorized pull

through technique after ingestion of tap water and carbonated beverages in 9 asymptomatic healthy volunteers. All carbonated beverages but not tap water produced sustained (20 min) reduction of 30% to 50% in all 3 parameters of the LES.⁶¹ These results indicated that carbonation with distension of the fundus could be responsible for reduction of the resting LES status to a level normally regarded as defective.

MINT AND CARMINATIVES

Peppermint was reported to be associated with reflux symptoms in 8% of a population with heartburn.³ A population-based, case-controlled study from Sweden failed to detect any association between mint and symptoms of reflux disease.⁸ The effects of high spearmint dose (500 mg) and flavoring spearmint dose (0.5 mg) on esophageal symptoms were assessed in healthy volunteers. A significant increase in reported symptoms followed high-dose spearmint in 6 out of 20 subjects. One subject complained of chest pain, 3 complained of heartburn, 1 of regurgitation, and 1 had both heartburn and regurgitation. Of these, 6 subjects who reported symptoms with high-dose mint, only 1 reported symptoms with low-dose mint and he also had symptoms during placebo administration.

There is no significant effect either of high-dose or low-dose spearmint on the number of reflux episodes seen within 30 minutes of ingestion.⁶² Peppermint oil relaxes gastrointestinal smooth muscle by reducing calcium influx.⁶³ Cardamon-induced belching and intraesophageal pressure changes consistent with those seen in symptomatic refluxers.⁶⁴ In healthy volunteers, intragastric administration of a dose equivalent to 180 mg of peppermint oil, reduced LESP within 1 to 7 minutes of infusion.⁶⁵ Spearmint has no effect on LESP either at high-dose (500 mg) or low-dose (0.5 mg).⁶²

SPICY FOOD AND ONIONS

Spicy food induced heartburn was reported in 88% of patients with GERD symptoms.⁴¹ Reported aggravating factors included spicy foods in a telephone survey including 1200 individuals.⁶ Ten percent of heartburn patients questioned avoided eating chilli because it precipitated their abdominal pain.⁶⁶ However, pepper food consumption was not associated with reflux symptoms in a Chinese population.⁹

A study compared heartburn severity, number of episodes, and changes in esophageal pH induced by 3 meals. McDonald's Quarter Pounder, French fries, and chocolate shake; McDonald's Sausage Biscuit with Egg, cheese, raw onion, and chocolate milk; and Wendy's Chilli and red wine. Despite lower-fat content, chilli and red wine promoted more heartburn pain and reflux than the other meals but the specific role of chilli cannot be assessed from these data.¹⁷

Induced-induced upper gastrointestinal symptoms were not accompanied by the changes in esophageal motility. Indeed, no changes in esophageal manometric

parameters after the ingestion of a chilli suspension (5 g of chilli powder in 100 mL of water with a capsaicin content of 478 ppm) were observed in 16 healthy volunteers although half of them reported one or more reflux symptoms. Direct instillation of the same chilli suspension into the lower esophagus produced typical symptoms in all 15 volunteers tested. The esophageal mucosa itself seems sensitive to chilli, suggesting that at least some of the upper gastrointestinal symptoms produced by chilli are mediated by direct stimulation of sensitive-sensitive afferent nerve endings in the esophageal mucosa.⁶⁷

Spicy meals and chilli also frequently contains cooked onions, which have been reported to precipitate heartburn and decrease LES.⁶⁸ Raw onions increased heartburn episodes, belches, and number of reflux episodes.⁶⁹ In this study, ingestion of onions did not increase any of the reflux variables, including percentage of the time pH was less than 4 measured in 16 normal subjects.

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