Physicians' Views Differ on Diet's Role in GERD

BY JOHN R. BELL
Associate Editor

Patients with gastroesophageal reflux disease may ask about the effect of dietary acids or medications on their disease, and despite the availability of "low acid" versions of coffee and orange juice, experts have different opinions on the impact of lifestyle factors in GERD.

Dr. Roy Orlando believes that high-acid foods can contribute to existing problems, because his patients have consistently reported worsening of symptoms after consuming them. "There are many things that can be irritants, but it's not all the time, and it's not in every person," said Dr. Orlando, professor of medicine and section chief, gastroenterology and hepatology, at Tulane University Medical Center, New Orleans.

As far as causation, however, "coffee in and of itself is not likely to be the cause of a disease—the disease being reflux disease," he said. "But if you have, or you develop, reflux disease, coffee could then be an irritant. The same thing is true if you have an ulcer of your stomach, or you have an irritation to the lining of your stomach from something entirely independent from the coffee you drank."

Thus, even though coffee and other beverages are not proved to cause gastritis, heartburn, GERD, or ulcers, such foods "can certainly irritate the lining of the stomach" in people who already have one such condition, according to Dr. Orlando.

However, Dr. Donald O. Castell attributed less importance to dietary acids in these diseases. "There are many things that people eat or drink every day which have an acid pH level," noted Dr. Castell, professor of medicine and director of the esophageal disorders program at the Medical University of South Carolina, Charleston, in an interview. "The most acidic are carbonated beverages, followed by citrus products, fruits, wine, coffee, and tea. By and large, these do not have any real effect on GERD except they may cause a transient burning symptom in the patient with esophagitis."

He cautioned that pH testing can be influenced by these products. "During testing with a pH sensor, the ingestion of any of these may produce an artificial lowering of the pH that mimics reflux," Dr. Castell said.

Dr. Orlando emphasized that manufacturers of "stomach friendly" products seem to be confusing GERD with the symptoms of heartburn, along with stomach distress. This may help to sell coffee and orange juice, he said, but physicians should ensure patients understand the differences between these ailments.

No proof exists that coffee causes GERD, ulcers, or heartburn, but it certainly can irritate the stomachs of patients who already have those conditions.

"The first thing one needs to be alert to is the fact that when [patients] talk about 'stomach problems,'… [these] are not esophagus problems," nor vice versa, he said in an interview. "So one needs to be sure one is accurate when talking about stomach-friendly products. If indeed the concern of the person who was drinking a coffee was that the region just …below the breastbone hurt or was sore or vaguely uncomfortable, that wouldn't be reflux, and that isn't heartburn."

Dr. Stuart Spechler noted that there is a paucity of research about the effects of dietary acids on GI and esophageal problems. "The data on the effects of coffee on heartburn have been contradictory at best, and it just doesn't seem to be a major player," said Dr. Spechler, the Berta M. and Cecil O. Patterson chair in gastroenterology at the University of Texas Southwestern Medical Center, and chief of gastroenterology at the Veterans Affairs Medical Center, both in Dallas.

His advice to patients, like Dr. Orlando's, is to avoid foods that aggravate symptoms. "What we usually tell our patients is, if you identify foods that give you trouble, then by all means avoid those. But I don't specifically prohibit coffee or orange juice. And the idea that making orange juice a little bit less acidic in terms of heartburn—I find this a little difficult to believe."

To qualify for a diagnosis of heartburn, the patient must report substernal discomfort that migrates in a superior direction, Dr. Orlando noted. Stomach problems, such as gastritis or gastric ulcers, include "a whole host of additional signs and symptoms," he said.

Aspirin in particular is a common contributor to GERD, he said, given that so many people now take it daily to protect the heart. And because patients often experience irritation only after weeks or months, they mistakenly attribute their symptoms to a beverage or food, rather than the medication.

It would be rare to find a patient whose current GI complaint was caused by something he was drinking in the absence of medication or underlying disease, Dr. Orlando said, acknowledging that it could happen if a patient is experiencing unusual stress, such as a student studying for an important examination who drinks many cups of coffee in order to stay awake.

Dr. Spechler noted that the latest medical treatments "are so good at stopping stomach acid that, by and large, what we used to call lifestyle modifications have really assumed a very secondary or even tertiary role. … Most of the [old] prohibitions—the dietary restrictions and things like that—were based more on ideas than on proof." Thus for patients who have no worsening of symptoms after eating high-acid foods, "I'm very lenient on diet for patients with reflux disease."

He added that physicians at his institution also advise patients to avoid eating meals close to bedtime and to avoid lying down immediately after eating.

Regarding the role of NSAIDs in heartburn or GERD, Dr. Spechler said that most studies have not shown NSAIDs to exacerbate these conditions but cautioned that that at least one has shown that these drugs may increase reflux. Thus "there are some concerns" about allowing patients with reflux to continue on NSAIDs.

"If the pills get stuck in the esophagus, they can be caustic; they can actually cause burns in the esophagus," he explained. Moreover, "there has been an association of strictures with use of NSAIDs. But in general, we don't see major problems anymore. We're seeing fewer strictures. The medical treatments are so good that restricting [NSAIDs] has become a very secondary feature of care for the patient with reflux disease."

He said that his institution will soon publish a study examining the effects of ibuprofen on reflux, in which the drug was associated with an increase in reflux among some patients.

"It did not show an enormous increase in acid reflux, but it did show a significant increase," he said. However, "we don't know if that's ibuprofen specific or if that's [caused by] all NSAIDs."

Dr. Orlando, however, indicated that he believes lifestyle modification is an integral part of long-term treatment. After putting the patient on an H_2 antagonist or a proton pump inhibitor for 4-8 weeks, the problem usually subsides.

However, "if the patient hasn't adjusted their lifestyle, then I'll guarantee that those symptoms will come back again. So, in my mind, the medications are a crutch or a handle to capture the disease more efficiently, to get whatever damage may have been done to the lining of the stomach and the lining of the esophagus under control, and to remove what may be provocative things within the lifestyle—including diet—that may have contributed to the symptoms that you can control." ■

Serum Markers May Help Diagnose Nonalcoholic Fatty Liver

BY HANNAH BROWN
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GLASGOW, SCOTLAND — Serum fibrosis markers—currently used as a research tool—have high sensitivity and specificity for diagnosing more severe forms of nonalcoholic fatty liver disease, according to a presentation at the Diabetes U.K. Annual Professional Conference.

Diagnosis of the most severe forms of nonalcoholic fatty liver disease (NAFLD), which include the onset of steatohepatitis and subsequent fibrosis and cirrhosis, requires measurement of the extent of inflammation and the presence of fibrosis. Currently, only liver biopsy can identify patients with these symptoms; such patients must be managed more aggressively than patients with less severe forms of the disease, particularly with respect to cardiovascular risk factors. However, biopsy is expensive and dangerous for the patient.

Dr. Christopher Byrne, head of the endocrinology and metabolism unit at the University of Southampton (England), said he believes "in the future, noninvasive serum markers might be better. Research is beginning to suggest that within NAFLD, a scoring system such as that using ELF [enhanced liver fibrosis assay, which looks at several serum biomarkers of fibrosis] might prove useful." When combined with age as a risk factor, the three markers assessed by the ELF blood test—hyaluronic acid, procollagen III amino terminal peptide (PIIINP), and tissue inhibitor of metalloproteinase 1 (TIMP-1)—have around 85% specificity and sensitivity for moderate to severe NAFLD, he noted.

Alanine aminotransferase (ALT) and GammaGT, plasma markers currently used to help guide diagnosis for NAFLD, are not very accurate, according to Dr. Byrne. "ALT is an extraordinarily poor proxy. Both GammaGT and ALT are in the normal range in patients who have quite extensive NAFLD when they get to biopsy," he explained.

NAFLD is one of the most common forms of chronic liver disease in developed countries, affecting 10%-24% of the general population, especially people with type 2 diabetes. Liver damage is caused by accumulation of lipids, oxidative stress, and inflammation from the release of proinflammatory cytokines. The associated marked insulin resistance in NAFLD has led some scientists to propose that it might be a malignant form of metabolic syndrome.

"Even adjusting for obesity, patients with NAFLD have marked increases in nonesterified fatty acid accumulation," said Dr. Byrne. "So release of these from adipocyte depots into circulation is abnormal in these patients. But we don't know why [it is] associated with marked insulin resistance."

He presented research showing that a group of 1,974 type 2 diabetes patients with NAFLD had a significantly higher prevalence of coronary, cerebral, and peripheral cardiovascular disease than a group of 418 type 2 diabetics without fatty livers. "NAFLD is associated with increased mortality, especially at the more severe end," said Dr. Byrne. "In these patients, even adjusting for all conventional cardiovascular risk factors and features of the metabolic syndrome, NAFLD is an independent cardiovascular risk factor. If you find NAFLD, think accelerated cardiovascular risk and treat aggressively."

Treatment recommendations include initial weight loss in patients that are obese; limited evidence suggests that pharmacologic therapy with glitazones also can be used to increase insulin sensitivity and decrease liver fat content. "Glitazones show promise," said Dr. Byrne. "A new indication for glitazone therapy may prove to be NAFLD." ■