Clinical Briefs in Primary Care™

Evidence-based updates in primary care medicine

By Louis Kuritzky, MD

Supplement to *Clinical Cardiology Alert, Clinical Oncology Alert, Critical Care Alert, Hospital Medicine Alert, Infectious Disease Alert, Neurology Alert, OB/GYN Clinical Alert, Primary Care Reports.*

VOLUME 19, NUMBER 6 PAGES 11-12 JUNE 2014

Zinc for the Common Cold

Source: Das RR, Singh M. Oral zinc for the common cold. *JAMA* 2014;311:1440-1441.

THE MECHANISM BY WHICH ZINC *COULD* impact the common cold is fairly straightforward: rhinovirus (the cause of the common cold) uses the ICAM-1 nasal epithelial cell receptor to latch on, and zinc ions block this. Since our bodies already usually have plenty of zinc onboard, the question is whether *supplemental* zinc can have an impact. In a *JAMA* clinical evidence synopsis, Das and Singh review 14 clinical trials of zinc in adults (n = 1781) and three in children.

Overall, common cold duration in adults was 7.5 days on placebo and 6.75 days when zinc supplementation was started within 24-48 hours of symptom onset, so folks got better about 1 day sooner. Disappointingly, symptom severity during illness was not alleviated.

Although not studied in adults, use of zinc as common cold prophylaxis in children has shown more impressive efficacy. When zinc supplements were provided at the onset of cold season, there was a 38% reduction in symptomatic colds compared to placebo (61.8% vs 38.2%). Whether similar benefits could be observed in adults has not been demonstrated. Similarly, at-risk groups among whom URI is particularly toxic (asthma, COPD, cystic fibrosis) have not been studied.

Many persons find zinc lozenges unpalatable, so whether the 1-day reduction in symptoms is worth the effort may be a personal decision. Guidelines from the American Academy of Family Physicians support the use of zinc within 24 hours of cold onset. ■

A Potential Key to Which Smokers Will Develop COPD

Source: Petersen H, et al. Rapid lung function decline in smokers is a risk factor for COPD and is attenuated by angiotensin-converting enzyme inhibitor use. *Chest* 2014;145:695-703.

IN THE BEST OF ALL WORLDS, WE WOULD convince smokers that they need to quit and assist them in that process. Even though smoking is associated with diverse risk in multiple anatomic compartments, it is lung disease that the public most commonly associates with smoking. Yet, we have to acknowledge that only a minority (20-25%) of smokers incur problems like chronic obstructive pulmonary disease (COPD). Is there a way to identify which smokers are most likely destined to develop COPD, thereby garnering an additional motivational tool — especially since so many smokers see consequences as either "it won't happen to me" or "it's a long, long way off"?

Petersen et al studied ever smokers (n = 809) who were free of spirometric abnormalities at baseline with periodic spirometry (q 18 months) for a mean of 6 years. Concordant with current guidelines, the metric utilized to quantify lung function was post-bronchodilator FEV1. One premise of the investigation was that study subjects who exhibited the most rapid rate of decline in FEV1 would also be most likely to develop COPD. Study subjects were categorized by rate of decline as rapid decline (≥ 30 mL/year), normal (0-29.9 mL/year), and no decline (unchanged from baseline). At the conclusion of the mean follow-up of 6 years, about one-third of the subjects fell into each of these categories.

As hypothesized by the authors, subjects who experienced rapid decline were approximately twice as likely to develop COPD. Another interesting insight from this trial was an evaluation of the association of various medications with outcomes. Seven different medication classes were addressed that were being used by these patients: angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers, beta-blockers, CCBs, statins, insulin, and oral hypoglycemic agents. ACE inhibitors were associated with a 45% lesser incidence of rapid decline, but no other statistically significant associations with other medications were demonstrated. The authors posit that salutary effects of ACE inhibitors upon development of COPD are related to anti-inflammatory activity of this class.

These data suggest that we may be able to identify smokers destined to develop COPD by rate of decline in FEV1. ■

While You're Fixing OSA with CPAP, Are You Fixing BP Too?

Source: Fava C, et al. Effect of CPAP on blood pressure in patients with OSA/hypopnea. *Chest* 2014;145:762-771.

THE ASSOCIATION BETWEEN OBSTRUCTIVE sleep apnea (OSA) and hypertension is well established, with putative mecha-

nisms including hypoxia-induced sympathetic activation, as well as renin-angiotensin-aldosterone activation. One would hope, then, that if OSA is *causing* the elevation in blood pressure (BP) — rather than just being an innocent bystander, or for instance being associated through a common comorbidity such as obesity — treatment of OSA might reduce BP.

Fava et al performed a systematic review and meta-analysis of randomized, controlled trials that reported data on BP in patients treated by continuous positive airway pressure (CPAP) for OSA (n = 2566). Overall, when compared to no treatment, OSA treatment was associated with a statistically significant reduction in BP, which was particularly prominent during the night (3.8/1.8 mmHg reduction). Although at first blush this degree of BP lowering might not seem that important, CPAP treatment trials are characterized by three limitations: 1) they are short term — most are 3 months or less, 2) the fact that patients entered a CPAP trial does not guarantee full compliance with the CPAP, and 3) less than half of the patients had hypertension, so we would anticipate a very small change in BP in a non-hypertensive population. Indeed, in subgroup analysis it is demonstrated that trials with longer duration and among patients with better compliance, BP reductions were greater.

Recognition that OSA is associated with resistant hypertension, coupled with the knowledge that CPAP treatment of OSA improves BP, bolsters both the therapeutic rationale for CPAP and inclusion of OSA screening as a component of evaluation for patients with resistant hypertension. ■

Clinical Briefs in Primary Care™ is published monthly by AHC Media, LLC.
Executive Editor: Leslie Coplin.
Editor: Stephen Brunton, MD.
Managing Editor: Neill L. Kimball.

Subscriber Information
Customer Service: 1-800-688-2421
E-Mail Address: neill.kimball@ahcmedia.com
World Wide Web: www.ahcmedia.com
Address Correspondence to: AHC Media, One Atlanta Plaza, 950 East Paces Ferry Road NE, Suite 2850, Atlanta, GA 30326.

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Colorectal Cancer Screening Through Stool DNA

Source: Imperiale TF, et al. Multitarget stool DNA testing for colorectal-cancer screening. *N Engl J Med* 2014;370:1287-1297.

In their most recent guidance on colon cancer screening (CCS), the American Cancer Society indicated that although a diversity of testing methods are available, since numerous patients are declining to be screened, "the best screening test is the one you can get done." This posture is an effort to reduce the number of unscreened persons, since when discovered early, most colon cancer is curable.

Stool DNA testing is not new. However, head-to-head clinical trials in the past decade have indicated that when compared to colonoscopy, sensitivity for detection of colon cancers and adenomas by older methods of stool DNA testing is lower. Stool DNA CCS is predicated on the fact that mutated and cancerous colonic epithelial cells are consistently excreted daily in the stool, even more commonly than blood is found. DNA panels for CCS, in theory, should be comparable to invasive screening methods, since abnormal DNA should be readily identifiable, and confirmatory colonoscopy and resection should follow. The tepid reception provided to CCS by the public is understandable: Many are put off by the preparation, expense, and inconvenience of colonoscopy. Additionally, in recently published clinical trials of persons undergoing screening colonoscopy, only a small percent actually harbor a cancer or advanced neoplasia (approximately 100 out of 3000 screenees), so it is easy to see why most folks will be correct when they think "it's probably not me."

Over the last decade, screening panels for stool DNA have been improved. Imperiale et al compared screening by fecal immunochemical testing (FIT) with stool DNA testing, based on a single stool sample for each, followed by colonoscopy in all patients, regardless of screening results.

Sensitivity for colon cancer was superior by DNA stool testing (92.3% vs 73.8%); similarly, sensitivity for high-grade dysplasia favored stool DNA testing (69.2% vs 46.2% sensitivity).

A positive FIT should lead to diagnostic colonoscopy; incorporation of stool DNA testing, when positive, might rightfully provide even further motivation. ■

Weight-Loss Surgery: Matching Expectations with Realities

Source: Li Z, Heber D. Managing weight loss expectations: The challenge and the opportunity. *JAMA* 2014;311:1348-1349.

The degree of success of weight-loss surgery (WLS) for improvement in metabolic derangements such as type 2 diabetes is impressive. Bariatric surgery has even been associated with improved mortality in morbidly obese individuals. Not everyone, however, enjoys the same degree of weight loss, and even though the endpoint of WLS in the minds of clinicians may be improvements in metabolic status and cardiovascular risk, in the minds of patients, the primary endpoint may be more cosmetically oriented. That is, just how much weight am I going to lose?

To better understand the expectations of WLS patients, Li and Heber interviewed patients seeking WLS and asked them (preoperatively) questions to better understand how much weight they expected to lose, the minimum weight loss with which they would not be disappointed, and how much risk they were willing to bear as part of WLS.

On average, patients expected to lose 38% of their weight; the Swedish Obesity Study found that 75% of gastric banding patients lost < 20% of their weight. Even though almost all (84.8%) of the WLS patients understood and accepted that there was a risk of dying from surgery, about one-third of these would no longer be willing to shoulder that risk if the weight loss was only 20%.

WLS has confirmed sustained metabolic and even mortality benefits. However, clinicians must confirm that patients concretely understand these parameters before embarking on such procedures, since some patients may no longer be willing to undertake risk if personally disappointing weight loss results were to occur. ■