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Internal Medicine

Evidence-based summaries of the latest research in internal medicine [ALERT]

Vol. 37, Issue 9, May 15, 2015
www.ahcmedia.com

ABSTRACT & COMMENTARY

Diet Sodas: Are They Really Diet?

By Martin S. Lipsky, MD

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Dr. Lipsky is a retained consultant for Health Solutions & Strategies.

SYNOPSIS: Increasing diet soda intake was associated with increasing abdominal obesity in an aging population.

SOURCE: Fowler PG, et al. Diet soda intake is associated with long-term increases in waist circumference in a bi-enthic cohort of older adults: The San Antonio Longitudinal Study of Aging. J Am Geriatr Soc 2015; DOI: 10.1111/jgs.13376.

Over the past several decades, concerns about increased sugar consumption has led some experts to recommend the use of non-nutritive sweeteners (NNSs).[1] However, while it may seem counterintuitive, the impact of NNSs and diet soda intake (DSI) on health outcomes appears to be negative.[2,3] To date, most studies exploring DSI focused primarily on middle-aged or younger adults rather than on older individuals. Since older adults are at high risk for cardiovascular disease, evaluating the impact of DSI on this group is important. To determine the relationship of DSI and health outcomes on older adults, Fowler and colleagues[3] studied a bi-ethnic cohort of older Mexican-American and European individuals as part of the San Antonio Longitudinal Study of Aging (SALSA). They assessed DSI and followed waist circumference (WC), an indirect measure of total and abdominal adiposity associated with greater cardiometabolic risk.[4]

SALSA gathered data on the health status of 749 Mexican-American and European-American elders, aged 65 and older, and tracked their health outcomes for more than 9 years. Interviewers recorded the number of sodas consumed and whether they were diet or regular at the beginning of the study and at each of three follow-up visits.

Financial Disclosure: Internal Medicine Alert's editor, Stephen Brunton, MD, is a retained consultant for Abbott, AstraZeneca, Boehringer Ingelheim, Janssen, Lilly, Meda Pharmaceuticals, Novartis, Novo Nordisk, Sanofi, and Teva; he serves on the speakers bureau of AstraZeneca, Boehringer Ingelheim, Janssen, Lilly, Novo Nordisk, and Teva. Peer reviewer Gerald Roberts, MD; executive editor Leslie Coplin; and managing editor Leslie Hamlin report no financial relationships relevant to this field of study.

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Internal Medicine Alert,
ISSN 0195-315X, is published monthly by
AHC Media, LLC
One Atlanta Plaza,
950 East Paces Ferry Road NE, Suite 2850
Atlanta, GA 30326.
www.ahcmedia.com

GST Registration Number: R128870672.
Periodicals Postage Paid at Atlanta, GA 30304 and at additional mailing offices.

POSTMASTER: Send address changes to
Internal Medicine Alert,
P.O. Box 550669,
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The investigators also measured WC, DSI, height, weight at onset and at each of the three intervals.

The study tracked 466 survivors for more than 9 years. After adjusting for initial WC, demographic characteristics, physical activity, diabetes mellitus, and smoking, the mean interval WC change of DSI users was 2.11 cm, almost triple the WC change 0.77 cm for those who did not consume diet sodas. In a subanalysis stratified for covariates, WC changes were consistently higher among DSI users.

The authors concluded that DSI was associated with escalating abdominal obesity and is a potential cardiometabolic risk factor in an aging population.

■ COMMENTARY

Personally, I like diets soda and used to recommend that individuals who drank soda to switch to diet soda to avoid the sugar and calories found in soft drinks. Intuitively it made sense that substituting 140 calories of sugar for a diet drink without calories would be beneficial. Unfortunately, this study adds to a growing literature associating the frequent use of DSI and NNSs with obesity and metabolic dysregulation. Therefore, for clinicians who advise older adults to drink diet soda as a strategy to help maintain their waistlines, these findings are sobering. Instead of helping, DSI may instead be a "belly" promotor. Among participants who reported occasional DSI — drinking less than one diet soda a day — waist circumference increased almost 2 inches, and among those who consumed diet sodas daily, their waist circumference increased more than 3 inches, or more than triple those with no DSI.[4]

The findings of an increase in waist size — the proverbial spare tire — correlates with a disproportionate increase in visceral fat, which is associated with a greater risk of metabolic disease, diabetes, heart attack, stroke, cancer, and mortality. A recent study[5] also linked drinking diet soda to an increased risk of heart disease in younger women.

A key question is whether the association between diet soda and adverse health outcomes is actually caused by DSI. The authors note that they are not establishing causation but reporting an association. However, they also note that their results are consistent with other studies linking DSI to an increased risk of diabetes, heart attack, stroke, and other major medical problems. However, if DSI is causal, the reasons why remain unclear.

So what are some possible reasons?[6,7] One is that there is no causal relationship and DSI is a marker of those with weight issues who are already on an increased waist circumference trajectory. Another common speculation is that DSI may trigger cravings to overeat, or because consumers overestimate their calorie savings, believe they have earned the right to eat more. The actual biological effect of DSI is still being explored, and it may be that DSI slows metabolic rate. Of interest, a recent study in Nature[8] found that artificial sweeteners changed the colonies of gut bacteria in mice in ways that adversely affected their glucose tolerance, suggesting a direct biological effect.

So at this time, what should one do with older patients? Perhaps counseling about the risks of DSI and stressing other alternatives, such as plain water, as a prudent strategy. ■

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Effects of Lifestyle Modifications on the Coronary and Carotid Atherosclerotic Burden

By Harold L. Karpman, MD, FACC, FACP

Clinical Professor of Medicine, UCLA School of Medicine

Dr. Karpman reports no financial relationships relevant to this field of study.

SYNOPSIS: Intensive lifestyle modifications (i.e., diet and exercise) are associated with a decrease in both coronary and carotid atherosclerotic burdens.

Jhamnani S, et al. Meta-analysis of the effects of lifestyle modifications on coronary and carotid atherosclerotic burden. Amer J Cardiol 2015; 115:267–275.

The guidelines for secondary prevention of atherosclerotic disease, published by the American Heart Association/American College of Cardiology[1] and the European Society of Cardiology,[2] clearly indicate that lifestyle changes, including dietary modifications and regular exercise, are both important forms of therapy for atherosclerotic cardiovascular disease; however, to date, randomized, controlled trials designed to establish the beneficial effects of these measures on atherosclerotic coronary or carotid artery disease progression have not been convincing. Because of the shortcomings, Jhamnani and colleagues[3] decided to perform a meta-analysis of the literature to critically examine and quantify the effectiveness of intensive lifestyle modifications on atherosclerotic disease progression. They focused their literature analysis on coronary and carotid arteries because of the important prognostic value of these arterial systems with respect to cardiac and cerebral vascular events and overall morbidity and mortality.

The Jhamnani meta-analysis included only randomized, controlled trials evaluating the efficacy of lifestyle measures comparing diet and/or exercise intervention vs usual care.[3] They included only studies that were prospective and which clearly reported the subject selection process, interventions, and follow-up and also included baseline and follow-up data on quantitative coronary angiograms and/or measurements of carotid intimal-media thickness. Trials in which subjects received medication along with diet and/or exercise were not included. When available, clinical outcomes were reported to complement the review. The results suggested that intensive lifestyle modifications were associated with a significant decrease in both coronary and carotid atherosclerotic burdens.

■ COMMENTARY

Published studies have demonstrated that compliance with short-term medical therapy ranges between 70-80%, whereas long-term medical therapy compliance drops to 40-50% and, finally, with respect to lifestyle changes, short- and long-term compliance is very low, at 20-30%.[4] A strong correlation exists between the level of compliance and the beneficial changes that occur in angiographically documented lesion stenosis, in effect demonstrating that a modest lifestyle change can lead to a decrease in atherosclerotic progression.5 The beneficial effects of exercise and diet have been well documented to improve cardiovascular and all-cause mortality,[6] often even halting or reversing atherosclerotic disease. Known cardiovascular risk

markers, such as lipid profiles, inflammatory risk markers, and total body weight, also improved with diet and exercise. It is important to recognize that the coronary angiographic studies essentially included patients aged 32-75 years with coronary artery disease with a predominance of men. The carotid intima-medial thickness populations studied were very broad, with an almost equal proportion of men and women.

This meta-analysis was not without limitations, since many of the trials, especially the coronary angiographic studies, were in relatively old populations, and the number of patients in each study was relatively small since there were ethical issues in having control groups that did not receive statin therapy and/or other medications. It was difficult to control the character of the diet in each study and the character and amount of exercise that was performed by the treated and the control groups of subjects. Further, randomized, controlled trials examining the effects of intensive lifestyle measures on atherosclerotic progression in coronary and carotid arteries are needed using up-to-date techniques. For example, coronary CT angiography would obviously simplify the evaluation of the coronary arteries and, in addition, would allow careful evaluation of soft plaque in addition to the degree of stenosis of the coronary arteries. ■

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ABSTRACT & COMMENTARY

Steroids for Severe Community-Acquired Pneumonia: More Evidence or More Uncertainty?

By Richard R. Watkins, MD, MS, FACP

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Dr. Watkins reports he's received research support from Forest Laboratories. This originally appeared in the April 2015 issue of Infectious Disease Alert.

SYNOPSIS: A multicenter, randomized, double-blind, placebo-controlled trial involving patients with severe community-acquired pneumonia and evidence of high inflammation found less treatment failure in those who received steroids. However, in-hospital mortality did not differ between the groups.

SOURCE: Torres A, et al. Effect of corticosteroids on treatment failure among hospitalized patients with severe community-acquired pneumonia and high inflammatory response. JAMA 2015;313:677-686.

Community-acquired pneumonia (CAP) is the leading cause of mortality from infection in industrialized countries, and treatment failure occurs in 10-20% of cases despite appropriate antibiotic therapy.

Thus, effective treatment strategies that reduce the burden of CAP would have a major impact on public health. Several previous studies that investigated the role of corticosteroids in CAP produced mixed results. Torres and colleagues hypothesized that corticosteroids modulate the immune response in severe CAP, thereby decreasing treatment failure.

The study was conduced at three teaching hospitals in Spain. Patients were prospectively enrolled between June 2004 and February 2012. The inclusion criteria included age > 18 years, symptoms of CAP, a new infiltrate on chest radiograph, met severe CAP criteria, and a C-reactive protein (CRP) > 150 mg/L on admission. Patients were excluded if they had previously been on corticosteroids, had HIV, had uncontrolled diabetes mellitus, had gastrointestinal bleeding in the preceding 3 months, or had influenza or a condition that required treatment with methylprednisolone. All enrollees were randomized to receive either intravenous methylprednisolone or placebo for 5 days started within 36 hours of hospital admission. The primary endpoint was the rate of treatment failure, which the authors divided into early (clinical deterioration within 72 hours of treatment) and late (defined as radiographic progression, persistence of respiratory failure including mechanical ventilation, development of shock, and death between 72 hours and 120 hours after treatment initiation). Secondary endpoints were time to clinical stability, length of stay, and in-hospital mortality. CRP levels were obtained on days 1, 3, and 7 of treatment.

A total of 120 patients were randomized, 61 to the methylprednisolone group and 59 to the placebo group. Ninety patients (75%) were admitted to the intensive care unit. The antibiotic regimens chosen, mostly ceftriaxone with levofloxacin or azithromycin, did not differ between the groups. There was significantly less treatment failure in the steroid group (8 patients [13%]) compared to the placebo group (18 patients [31%) (P = 0.02). There were no statistically significant differences in the secondary endpoints between the steroid and placebo groups. At hospital day 3, decreases in CRP levels were greater in the steroid group, while those patients with persistent high CRP at day 7 had a higher percentage of treatment failure and mortality.

■ COMMENTARY

This study showed that patients with severe CAP who received methylprednisolone had reduced inflammation and less treatment failure compared to those who received placebo. However, these results need to be interpreted with caution. First of all, baseline cortisol levels were not measured. It is possible that undiagnosed adrenal insufficiency may have led to more treatment failures, especially in critically ill patients admitted to the ICU. Second, the placebo group had a higher proportion of patients with septic shock and acute respiratory failure requiring mechanical ventilation. This could be interpreted that steroids were given to patients who were less ill and, therefore, expected to have better outcomes. Third, the main treatment difference between the two groups was mainly due to less radiologic progression 72 hours or more from time of randomization. As noted in an accompanying editorial, the two likely explanations for this phenomenon are worsening pneumonia and the development of acute respiratory distress syndrome (ARDS).[1] It seems illogical that steroids would help with the former condition but plausible they might modulate the latter. One theory is that ARDS is caused by cytokine release from a Jarisch-Herxheimer-like reaction in tissue with a high bacterial genomic load after the initiation of antibiotics. Thus, steroids may block this inflammatory reaction from occurring. But additional larger and more definitive studies are necessary to confirm that less radiologic progression leads to improved mortality.

In the current (albeit outdated) IDSA/ATS clinical practice guidelines for CAP,[2] CRP is not a recommended diagnostic test, although it is used in European countries.[3] Torres and colleagues used a CRP level of >= 150 mg/L as an inclusion criterion for quantifying inflammation, yet only 57% of eligible patients (162/284) had this level. Therefore, the patients in their study represented only a fraction of those with severe CAP. The possible benefits of steroids for patients with severe CAP and lower CRP levels remain to be elucidated. Because the immunosuppressive effects of steroids could theoretically worsen an already severe infection, it is important to recall the old adage "primum non nocere" (first, do no harm). The results of the study by Torres and colleagues need further confirmation before the widespread use of steroids in severe CAP can be endorsed. ■

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Clinical Briefs

By Louis Kuritzky, MD

Consequences of NSAID Use in Patients Receiving Post-MI Antithrombotic Prophylaxis

SOURCE: Olsen AMS, et al. JAMA 2015;313: 805-814.

Most patients receive antiplatelet treatment after an acute coronary syndrome. Combinations of antiplatelet agents (e.g., ASA, clopidogrel) reduce risk of recurrent MI — particularly stent thrombosis — but do have a modest increase in bleeding risk. Well, what about our post-MI patients who are taking appropriately prescribed antiplatelet agents who also require treatment with NSAIDs for disorders like osteoarthritis, migraine, etc? How does such multidrug co-administration affect risks?

Olsen et al recently published results from data collected through Danish nationwide administrative registries that assessed bleeding events and cardiovascular events based on prescription data from 62,971 adults. The cohort evaluated included adults over age 30 (mean age = 68 years) who were admitted for a first MI in the 2002-2011 interval.

Rates of bleeding were doubled when NSAID users (that is, NSAIDS + antithrombotic treatment) were compared with non-users (that is, antithrombotic treatment alone). Equally concerning, the hazard ratio for cardiovascular (CV) events was 40% greater in NSAID users than non-users, irrespective of particular type of NSAID prescribed or duration of use.

This report should prompt clinician vigilance in post-MI patients taking antithrombotic therapy to limit the use of NSAIDs to the minimum necessary. ■

Psoriasis Is Associated with Insulin Resistance

SOURCE: Gyldenlove M, et al. J Am Acad Dermatol 2015;72:599-605.

The pathophysiology of psoriasis has much in common with rheumatoid arthritis (RA). Recently, pharmacotherapies that had been primarily used in RA have enjoyed successful application in psoriasis, leading to marked improvements or even remission. Of concern, both RA and psoriasis have been recognized as risk factors for cardiovascular disease. Psoriasis is also a risk factor for diabetes, but the mechanism remains ill-defined.

A consistent finding in patients with type 2 diabetes is the presence of insulin resistance, usually associated with obesity. Might psoriasis also be associated with insulin resistance, independent of obesity? To answer this question, Gyldenlove et al compared psoriatic patients with controls of similar age (mean = 44 years) and BMI (mean = 26, [overweight]), all of whom had undergone laboratory screening (fasting glucose and A1c) to exclude undiagnosed diabetes.

Insulin sensitivity was substantially reduced in patients with moderate-to-severe psoriasis compared to controls, even though there was no manifest glucose intolerance. Hence, psoriasis might be considered a pre-diabetic state. ■

Difficult Questions About Testosterone and Mortality

SOURCE: Eisenberg ML, et al. Int J Impot Res 2014;27:46-48.

The number of men receiving treatment for hypogonadism has increased dramatically over the past decade. At the same time, some clinical trials have suggested that there are safety issues with testosterone. For instance, one trial of frail seniors was stopped early because of an increased mortality signal. Other clinical trials have not demonstrated similar risk. Ultimately, the only way the question about testosterone safety can be convincingly answered is by performance of a large randomized, prospective trial, similar to the Women's Health Initiative (WHI).

In the meantime, other trial data may inform clinician choices about testosterone replacement. For instance, Eisenberg et al published their results of a retrospective analysis of the andrology database at Baylor College of Medicine (Houston, Texas), which included 509 hypogonadal men, approximately half of whom were treated with testosterone replacement (injectable or transdermal).

In a 10-year follow-up interval, the mortality rate trended lower in men receiving testosterone replacement than controls, but the difference was not statistically significant. Although such results cannot provide a definitive answer to the question of the relationship between testosterone replacement and mortality, they are reassuring for hypogonadal men who are enjoying symptomatic improvement through testosterone replacement. ■

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CME QUESTIONS

1. Diet sodas are associated with all of the following except:

   a. Colon cancer

   b. Type 2 diabetes

   c. Weight gain

   d. Increased waist circumference

2. The effects of intensive lifestyle (i.e., diet and exercise) modifications consisting of diet and exercise activities on coronary and carotid atherosclerotic burdens:

   a. are hard to accurately measure.

   b. are of minimal value in most patients.

   c. have been demonstrated to be associated with a decrease in coronary and carotid atherosclerotic burdens.

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Upon completion of this educational activity, participants should be able to:
- describe new findings in the differential diagnosis and treatment of various diseases;
- describe the advantages, disadvantages and controversies surrounding the latest advances in the diagnosis and treatment of disease;
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[IN FUTURE ISSUES]

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Internal Medicine [ALERT]

ECG Review

By Ken Grauer, MD

Professor Emeritus in Family Medicine, College of Medicine, University of Florida

Dr. Grauer is the sole proprietor of KG-EKG Press, and publisher of an ECG pocket brain book.

Wide Tachycardia in a Patient with Chest Pain

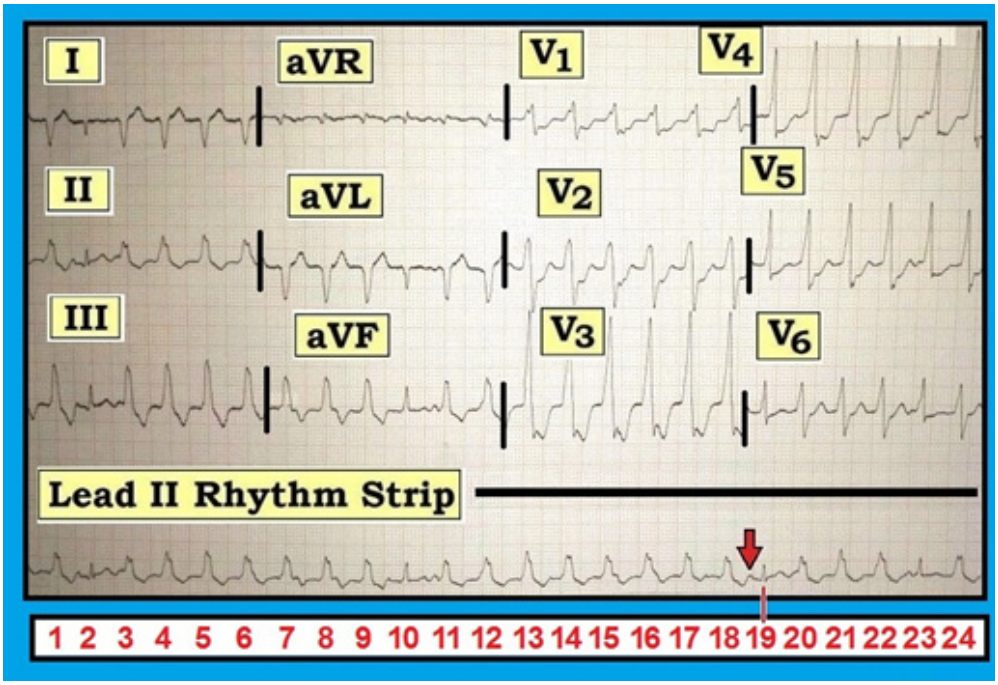

Figure: ECG from a 70-year-old woman with chest pain. What is the rhythm?

The 12-lead ECG and accompanying long lead II rhythm strip in the Figure was obtained from a 70-year-old woman with chest pain. She was hemodynamically stable at the time this ECG was recorded. What is the rhythm? How certain are you of your rhythm diagnosis? Is there any clue to the etiology of her chest pain?

Interpretation: This is a challenging tracing. Fortunately, the patient was hemodynamically stable, which provided a moment of time for analysis.

• The predominant rhythm is a regular WCT (wide complex tachycardia) without normal P waves. Statistically, the odds that a regular WCT rhythm without sinus P waves in an older adult with chest pain will turn out to be VT (ventricular tachycardia) approach 90%, even before one looks at the ECG.

• Assessment of Axis and QRS morphology during the tachycardia often provide additional clues that the rhythm is VT rather than supraventricular tachycardia with either aberrant conduction or pre-existing bundle branch block. For example, the extreme axis deviation seen here (i.e., all negative QRS complex in lead I) virtually proves this is VT. Support for this conclusion is enhanced by the fact that this all negative QRS waveform in lead I does not resemble any type of conduction defect.

• Definitive proof that the rhythm is VT is forthcoming from the long lead II rhythm strip. Note intermittent occurrence of narrow QRS complexes (i.e., beats #2,10,19,23) that interrupt the underlying WCT rhythm. These narrow (seemingly normal) conducted complexes are "capture" beats. They are each preceded by a sinus P wave (red arrow preceding beat #19 showing one such P wave). In addition, there is at least one "fusion" beat, as the QRS complex of beat #11 is intermediate in size and shape compared to the beat that precedes it (#10) and the beat that follows (#12). The presence of fusion and capture beats provides definitive proof that the rhythm is VT. ■

Note: Further discussion of this tracing is available on an ECG video and blog post found at this site: http://tinyurl.com/KG-Blog-102.