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Internal Medicine

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ABSTRACT & COMMENTARY

Is BMI Good Enough to Measure Visceral Adiposity?

By Seema Gupta, MD, MSPH

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

SYNOPSIS: A large international study found frequent discordance between body mass index (BMI) and waist circumference (WC), driven by the substantial variability in visceral adiposity. Within each BMI category, patients with the highest WC were more likely to have higher cardiometabolic risk.

SOURCE: Nazare JA, et al. Usefulness of measuring both body mass index and waist circumference for the estimation of visceral adiposity and related cardiometabolic risk profile (from the INSPIRE ME IAA Study). *Am J Cardiol* 2015;115:307-315.

Worldwide, the prevalence of obesity has reached epidemic proportions. This epidemic is not simply a consequence of poor diet or sedentary lifestyles, but is a complex, multifaceted state in which environmental, biological, and genetic factors all play essential roles. Increased body mass index (BMI) is associated with higher all-cause mortality compared with normal weight individuals, as well as mortality from cardiovascular disease. Abdominal adiposity is recognized to be associated with a number of conditions leading to increased cardiometabolic risk (CMR). Studies have demonstrated that measuring the indices of abdominal adiposity, such as waist-to-hip ratio (WHR) and waist circumference (WC), may be superior to BMI in detecting CMR in both sexes.[1] Measuring WC is a simple procedure requiring a tape measure. However, researchers do not completely

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agree and have questioned the clinical relevance of measuring WC instead of BMI to assess cardiovascular disease risk, since both BMI and WC have been found to be highly correlated and each can independently predict CMR.[2] When conducting assessments of central adiposity, directly assessing visceral adiposity tissue (VAT) by computed tomography allows more accurate evaluation since VAT is more metabolically active than other adipose tissue sites and appears to contribute to many metabolic abnormalities associated with weight gain.

In their study, Nazare et al evaluated the relevance of adding WC to BMI for the estimation of VAT and CMR. Between 2006 and 2008, 297 physicians recruited 4504 patients from 29 countries. Final analysis included data from 4109 patients. Researchers measured both BMI and WC and assessed VAT and liver fat by computed tomography.

The study found that although there was a strong correlation between WC and BMI, about 30% of participants displayed discordant values for WC and BMI quintiles ($r = 0.87$ and $r = 0.84$ for men and women, respectively, $P < 0.001$). VAT and WC showed considerable between-subject variability within each BMI category. Increasing gender-specific WC tertiles correlated with significantly higher VAT, liver fat, and a more adverse CMR profile within each BMI category.

Within each such BMI category, patients with the highest WC were more likely to have higher liver fat content, high-sensitivity C-reactive protein, and prevalence of type 2 diabetes mellitus, in addition to the classical risk factors. Authors contend that this finding in the study may provide further support for the hypothesis that the increased cardiovascular risk may be due, in part, to the excess VAT and liver fat, contributing to atherosclerosis, insulin resistance, and inflammation. They also hypothesize that based on findings, patients with high WC and low BMI may be more prone to VAT accumulation and CMR abnormalities, irrespective of ethnicity.

■ COMMENTARY

In recent years, there has been an increasing debate over which measure of overweight and obesity is best able to discriminate those individuals who are at increased CMR and visceral adiposity. While BMI is currently widely utilized as an index of general adiposity, measures of central adiposity, such as WC and WHR, may be superior in assessing these risks.[3] The current study conducted across several nations and ethnicities demonstrates that for any given BMI value, both men and women display substantial inter-individual variation in WC, revealing important differences in VAT and liver fat. This may explain the discordance between WC and BMI values observed.

While it has been debated whether WC should be measured in addition to or instead of BMI, the study clearly makes a case for measuring both since these two parameters seem to provide separate but important information.

This may be an example in which a strong correlation between BMI and WC at the population level may not necessarily imply the same at the individual patient level. The combined use of both measures in an office setting would allow for clinical stratification of patients based on their CMR, VAT, and liver fat content at a given BMI. This approach may be better able to provide an assessment of visceral adiposity and CMR of the individual patient. ■

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ABSTRACT & COMMENTARY

Effects of Coenzyme Q10 in Chronic Heart Failure Patients

By Harold L. Karpman, FACC, FACP

Clinical Professor of Medicine, UCLA School of Medicine

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

SYNOPSIS: Treatment with coenzyme Q10 in addition to standard therapy for patients with moderate to severe HF is safe, well tolerated, and associated with a reduction in symptoms and major adverse cardiovascular events.

SOURCE: Mortensen SA, et al. The effect of coenzyme Q10 on morbidity and mortality in chronic heart failure. *J Amer Coll Card Heart Failure* 2014;2:641-649.

Many research groups have attempted to elucidate the pathways, deficiencies, and enhancement of the energy-generation centers of the myocardial cells, the mitochondria.[1] Coenzyme Q10 (CoQ10) influences mitochondrial function by contributing to oxidative phosphorylation and the generation of adenosine triphosphate, thereby augmenting energy production and enhancing efficient energy utilization.[2] Previously published meta-analyses of randomized, controlled trials using CoQ10 in patients with heart failure (HF) have mostly revealed that the drug had a positive effect on left ventricular ejection fraction (EF) whether or not improvement of the New York Heart Association (NYHA) functional class occurs.[3-5]

Mortensen and his associates[6] performed a prospective, randomized, double-blind, placebo-controlled, multicenter trial of CoQ10 as adjunctive treatment of chronic HF focusing on changes in symptoms, biomarker status, and long-term outcomes. Patients were enrolled in 17 European, Asian, and Australian centers from 2003 to 2010. The treatment group received CoQ10 in a dosage of 100 mg three times daily. The short-term (i.e., 16 weeks) aim of the study was a blinded evaluation of patient symptoms and functional status and the long-term (106 weeks) goal of the study was to test whether CoQ10 could reduce cardiovascular mortality and morbidity in HF patients as a composite endpoint. CoQ10-treated patients compared with the placebo group were found to have a significantly lower cardiovascular mortality, all-cause mortality, and a reduced incidence of hospital stays for HF. A significant improvement in NYHA class was also found to occur in the CoQ10-treated group.

■ COMMENTARY

Despite significant improvements in pharmacological HF therapy, Mortensen and colleagues have clearly demonstrated that adding 300 mg of CoQ10 daily significantly reduced cardiovascular death by 43% and all-cause mortality by 42%. Furthermore, CoQ10 supplementation improved symptoms according to positive changes in the NYHA functional classification after 2 years of therapy. The biological mechanisms behind the improvement of symptoms and survival in the HF patients who received CoQ10 may be multiple.[7-9] These include increased energy production in the failing heart, changes in oxidative phosphorylation, and improvement in endothelial function.[10] In addition, it has been demonstrated that CoQ10 may protect the myocardium against ischemia.[11] Many patients with HF are malnourished as a result of defects in substrate utilization and energy supply,[12] but whether a patient is malnourished or not, multiple metabolic dysfunctions may be present. It should be recognized that this trial of chronic HF recruited 420 patients over an 8-year period in nine countries in 17 centers; clearly, this trial had significant recruitment and performance issues, but the final results are clearly positive and should not be ignored. It is obvious that more research is required for further elucidation of the many molecular causes of heart failure that may be influenced in a positive way by CoQ10 or other drugs that become available in future years.

In summary, the results of the Mortensen study demonstrated that treatment with CoQ10 in addition to standard therapy in patients with

moderate or even severe HF is safe, well-tolerated, and appears to be associated with a reduction in symptoms, with improvement of the NYHA functional class after 2 years of therapy. ■

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ABSTRACT & COMMENTARY

Strengthen the Feet to Treat Plantar Fasciitis

By Joseph Scherger, MD

Vice President, Primary Care, Eisenhower Medical Center; Clinical Professor, Keck School of Medicine, University of Southern California, Los Angeles

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

SYNOPSIS: A strength training exercise improved self-reported 3-month outcomes in treating plantar fasciitis compared with inserts and stretching.

SOURCE: Rathleff MS, et al. High-load strength training improves outcome in patients with plantar fasciitis: A randomized controlled trial with 12-month follow-up. *Scand J Med Sci Sports* 2014;Aug 21 (Epub ahead of print).

A group of Danish researchers studied 48 consecutive patients referred to physical therapy for plantar fasciitis pain of at least 3 months' duration. The diagnosis was confirmed by ultrasound thickening of the plantar fascia. The patients were randomized to two treatment groups. All were given heel cups for additional support.

The stretching group performed a conventional treatment consisting of crossing the affected heel over the opposite knee and pulling the toes back for 10 seconds. This was repeated in sets of 10 three times a day.

The strengthening group performed heel lifts from a raised platform, such as a step, with a rolled towel placed under the toes to cause maximum dorsiflexion. The heels were raised for more than 3 seconds, held for 2 seconds, and then lowered over 3 seconds. Three sets of 12 repetitions were performed every other day. After 2 weeks, the

weight was increased by wearing a backpack filled with books. The heels lifts were increased to 10 repetitions 4 times a day. The weight was increased again after two more weeks, and five sets were performed every other day.

Outcomes were measured between the two groups at 1, 3, 6, and 12 months. The primary outcome was at 3 months. The strengthening group had significantly more improvement at 3 months than the stretching group, with a functional foot index score that was 29 points lower ($P = 0.016$). The differences between the two groups declined at 6 and 12 months, with the scores of the stretching group no longer statistically different from the strengthening group.

■ COMMENTARY

Human feet have similar intrinsic muscles as the hand. Human feet are able to grab things and perform functions no longer used in a civilized society that wears shoes and uses feet mainly for walking. As pointed out by Christopher McDougall in his fascinating book, *Born to Run*,[1] our feet are splinted by supportive shoes and the muscles become weak. A lack of muscle strength in the feet may be a common cause of foot problems such as plantar fasciitis.

Conventional treatment of plantar fasciitis is based on rest, supports, and stretching. If these do not work, steroid injections and even surgery are done. The muscles of the feet have been largely ignored in treatment, and the feet are left in a weakened condition. Runners with plantar fasciitis either give up or keep their goals limited. Some runners now strengthen the feet by going barefoot or wearing shoes with minimal support.
This is a breakthrough study using foot strengthening as a primary treatment for plantar fasciitis. The subjects using foot strengthening recovered faster. It would be interesting to see if the strengthening group was able to perform more activities than the stretching group.

> [The strengthening group had significantly more improvement at 3 months than the stretching group ...]

In my clinical practice, I have developed an exercise I call the "foot grip." This is an isometric contraction of the intrinsic muscles as if to grab something with the bottom of the feet, similar to a hand grip. These can be performed hundreds of times a day, even in shoes, while sitting, standing, or lying down. No equipment is needed. At Eisenhower Medical Center, we are currently doing a pilot study of foot grips as a treatment for plantar fasciitis. My own experience has shown rapid improvement, and if the pilot study is successful, we will proceed with a controlled trial similar to the one reported here.

There is likely to be a paradigm shift toward foot strengthening as the new primary method of treating plantar fasciitis. ■

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Internal Medicine [ALERT]

Clinical Briefs

By Louis Kuritzky, MD

Ambulatory BP Monitoring

SOURCE: Turner JR, et al. *Am J Medicine* 2015; 128:14-20

The benefits of hypertension treatment (HTN), often cited as a 25% reduction in myocardial infarction, 40% reduction in stroke, and 50% reduction in heart failure, have generally been demonstrated in clinical trials based on an office blood pressure measurement. Since a substantial minority of patients enrolled in HTN trials — approximately one-third according to numerous estimates — ultimately turn out to have white coat HTN (wc-HTN), we may be underestimating the actual benefits of HTN treatment. Patients with wc-HTN do not suffer the same increased risk of cardiovascular events as HTN patients; hence, their inclusion in HTN trials "dilutes" treatment effects.

Since 2011, the United Kingdom regulatory agency NICE (National Institute for Health and Care Excellence) has asked that primary care clinicians obtain ambulatory BP monitoring (ABPM) on all patients suspected of HTN prior to initiation of treatment. Why? Because no treatment is indicated in the one-third of patients who typically turn out to have wc-HTN. United Kingdom calculations indicate that routine application of ABPM in primary care will save tens of millions of dollars.

ABPM is the most accurate tool for identifying wc-HTN. Additionally, it can help ascertain whether symptoms such as dizziness are potentially related to hypotensive episodes. It can also demonstrate whether treatment is truly providing 24-hour control of BP, which is usually not discernible in typical office practice where patients are evaluated during daytime hours.

ABPM is a much better predictor of cardiovascular risk than office blood pressure readings. At the current time in the United States Medicare only pays for ABPM when the diagnosis of wc-HTN is utilized. Private insurance coverage for ABPM varies. More routine inclusion of ABPM would likely help to clarify important HTN-related issues. ■

A New Oral Treatment for Hyperkalemia: Patiromer

SOURCE: Weir MR, et al. *N Engl J Med* 2015; 372:211-221.

Patiromer (PAT) is an oral non-absorbable polymer that works by binding potassium (K+) in exchange for calcium in the distal colon. Currently available oral treatments for hyperkalemia are burdened by GI adverse effects as well as limited efficacy. Hyperkalemia is particularly problematic in chronic kidney disease (CKD), which may be compounded by the need to administer ACE inhibitors or angiotensin II receptor blockers (ARB).

Weir et al performed a clinical trial of PAT in hyperkalemic patients with CKD stage 3 or 4 (eGFR = 15-59) who had been on a stable dose of ACE inhibitors or ARB for at least 4 weeks. Mild hyperkalemia (K+ = 5.1-5.4 mmol/L) was treated with PAT 4.2 g BID, and moderate-severe hyperkalemia (K+ = 5.5-6.4 mmol/L) with PAT 8.4 g BID.

At the end of 4 weeks, 76% of hyperkalemic patients treated with PAT had reached their target K+ of 3.8-5.0 mmol/L. Re-randomization to placebo or PAT for an additional 8 weeks showed that 85% of PAT-treated patients remained normokalemic, whereas 60% of placebo recipients drifted back into hyperkalemia. PAT was well tolerated: 11% of PAT patients experienced mild-moderate constipation.

PAT shows great promise as a new treatment for hyperkalemia. ■

Obesity Leads to Overdiagnosis of Airflow Obstruction

SOURCE: Collins BF, et al. *Chest* 2014;146: 1513-1520

Some commonplace disorders can readily misdirect clinicians about the presence of other important diagnoses. For instance, in patients with chronic obstructive pulmonary disease (COPD), deterioration of cardiac function, leading to congestive, can easily be misinterpreted as worsening COPD since fatigue, exercise, intolerance, and dyspnea are common to both. Could obesity misdirect clinicians in their diagnostic process for COPD? This report from the Veterans Administration system suggests that it can.

Collins et al reviewed data of obese veterans diagnosed with COPD who had undergone spirometry. Approximately half of COPD patients did not demonstrate airflow obstruction (necessary for the diagnosis of COPD) upon spirometry. After spirometry was performed, obese persons were less likely than normal weight individuals to have inhaler medications decreased or discontinued. The data found that as the degree of obesity increased in these COPD patients, the likelihood that airflow obstruction would be found on spirometry decreased.

Although clinicians may be tempted to diagnose COPD based simply on symptoms alone, these data indicate that obese patients are particularly likely to be misdiagnosed with COPD, incurring potentially inappropriate medications and distracting clinicians from attaining a correct diagnosis to explain patients' symptoms. Clinicians would be wise to follow clinical guidelines that indicate spirometry as the gold standard for COPD diagnosis. ■

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CME QUESTIONS

1. **Based on the study by Nazare et al, which of the following is the best measure to assess the cardiometabolic risk in office setting?**
   a. Body mass index only
   b. Waist circumference only
   c. Body mass index and waist circumference
   d. Waist-to-height ratio

2. **Coenzyme Q10 therapy of patients with chronic heart failure:**
   a. is hazardous over the long-term.
   b. is safe, improves symptoms, and reduces major adverse cardiovascular events.
   c. over the long-term does not improve the NYHA classification significantly.
   d. should not be given to patients who are receiving statin therapy.

3. **The Danish study on the treatment of plantar fasciitis showed which treatment method resulting in more rapid recovery?**
   a. Stretching
   b. Orthotics
   c. Foot muscle strengthening
   d. Steroid injection

CME OBJECTIVES

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;
- identify cost-effective treatment regimens;
- explain the advantages and disadvantages of new disease screening procedures.

[IN FUTURE ISSUES]

The Accelerating Cost of Generic Drugs

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.

Is there AV Block?

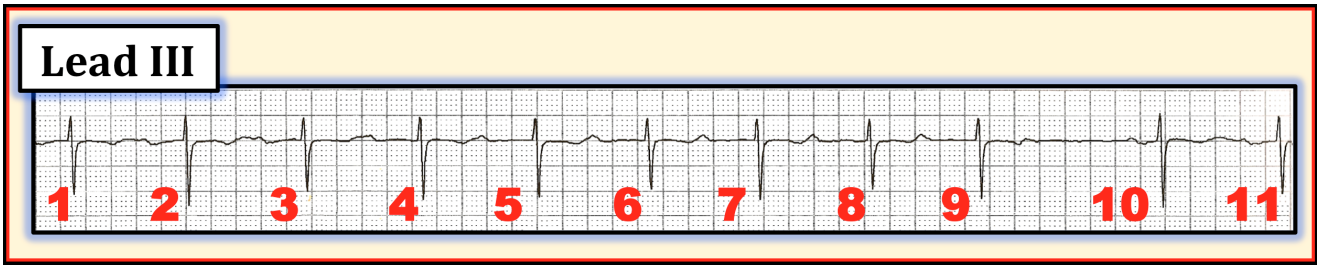

Figure: Long lead III rhythm strip. Is there AV block?

Does the long lead III rhythm strip shown below manifest a form of AV block? If so, what type of AV block? How does one distinguish AV block from other causes of relative pauses, such as PACs (premature atrial contractions) that are either blocked or conducted?

Interpretation: This is not an easy rhythm strip to interpret — primarily because P wave amplitude is small, and atrial activity is easily confused with the terminal portion of the ST-T wave in the first 9 beats of this tracing. In addition, there is some baseline artifact that adds to the difficulty of *P* wave recognition. The key lies with recognition that the small amplitude biphasic deflection preceding beat #10 is definitely a *P* wave, albeit one with a slightly prolonged PR interval (of 0.22 second). With this as a starting point, we can recognize that there is also a small-amplitude P wave deflection preceding beat #11. Note that the PR interval preceding beat #11 has increased slightly compared to what it was before beat #10. Use of calipers is essential for interpreting the rest of this tracing. We need to determine if atrial activity is present elsewhere — and if so, whether atrial activity is regular.

• Set your calipers to the P-P interval suggested by the distance between the P wave before beat #10 and the *P* wave before beat #11.

• At this approximate caliper setting, you should be able to "walk out" regularly occurring *P* waves for the entire tracing. Thus, the slightly changing shape toward the end of the ST-T wave for the first 8 beats is the result of superposition of a P wave with prolonged PR interval at slightly varying position during the cardiac cycle.

• Note the notch in the ST-T wave after beat #9. This represents a regularly occurring P wave that is nonconducted.

• Although difficult to appreciate an increase in PR interval from one beat-to-the-next for the first 8 beats in this tracing, it should be obvious that the PR interval preceding beat #9 is longer than the PR interval at the beginning of the tracing (before beats #1 and #2).

Impression: We have described the findings of a regular (or at least almost regular) atrial rhythm with progressively increasing PR interval until finally a P wave is nonconducted (after beat #9). The cycle then begins again with shortening of the PR interval before beat #10. This is 2nd degree AV block, Mobitz Type I (AV Wenckebach).

• Occasionally, Wenckebach cycles may be long and subtle as seen here. Use of calipers is invaluable for confirming regularity of the atrial response (which rules out PACs as the etiology). Comparing the PR interval at the beginning of a long Wenckebach cycle with the PR interval at the end of the cycle just before the beat is dropped helps to appreciate subtle but progressive PR prolongation. ■

Note: Further discussion of this tracing is available on an ECG video found at this site:
http://tinyurl.com/KG-Video-1