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Internal Medicine

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

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ABSTRACT & COMMENTARY

Is Provocative Cardiovascular Testing Indicated for Everyone with Chest Pain?

By Harold L. Karpman, MD, FACC, FACP

Clinical Professor of Medicine, UCLA School of Medicine

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

SYNOPSIS: In patients with acute chest pain evaluated in a chest pain evaluation center, the yield from routine use of provocative noninvasive testing in determining the presence of occlusive coronary artery disease was low.

SOURCE: Winchester DE, et al. Diagnostic yield of routine noninvasive cardiovascular testing in low-risk acute chest pain patients. *Am J Cardiol* 2015;116:204-207.

Cardiology guidelines have encouraged provocative cardiovascular testing even for low-risk patients with chest pain presenting to ED. However, the recent literature has suggested that indiscriminate testing of these patients resulted in a low yield of patients with hemodynamically significant coronary artery disease.[1] Winchester et al decided to determine the diagnostic yield of routine noninvasive provocative cardiovascular testing studies in an acute chest pain population preselected to be at low cardiovascular risk.[2] They established a chest pain evaluation center (CPEC) within the ED of the University of Florida. Patients were selected for observation within the CPEC if they had no known heart disease and no evidence of ischemia based on initial electrocardiogram and biomarker testing. The patient population consisted of 213 subjects who were predominantly women with a prevalence of diabetes (10.3%), hypertension (37.1%), hyperlipidemia (17.8%), and current tobacco use (23.5%). Exercise modalities included treadmill testing (49%), computed tomography coronary angiography (27%), and myocardial perfusion imaging (9%) in the 203 patients who were tested. Eleven participants had abnormal test results and only four were demonstrated to have obstructive coronary artery disease (CAD), based on invasive coronary angiography resulting in an overall diagnostic yield for obstructive CAD of only 2.5%.

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They concluded that in patients with acute chest pain evaluated in a CPEC, the yield of routine use of noninvasive testing for CAD was minimal and that the positive predictive value of an abnormal test was quite low.

■ COMMENTARY

The results of the study by Winchester et al correlated with the results of previously published studies that revealed the yield is quite low in low-risk patients with chest pain who undergo provocative testing.[1-4] In this study, the chest pain severity, duration, and character did not identify patients with positive noninvasive tests. However, it should be clearly recognized that the size of the study group was extremely small and almost certainly inadequate for definitive conclusions in this regard. Equally important, although the overall diagnostic yield for obstructive coronary artery disease in patients was only 2.5%, it would be a significant oversight to overlook significant obstructive CAD even in this small number of patients and despite the fact that the pretest likelihood of CAD was low. This is an important consideration, especially since the concern about doing provocative cardiac testing is only economic in nature and not related to the extraordinarily low volumes of complications associated with these provocative testing studies. Finally, the limitations of the Winchester study should be recognized in so much as the study population was extremely small, all came from a single center, and there may have been selection bias through triage in the CPEC.

In summary, the results of the Winchester study certainly do not overrule any clinician's decision in his care of chest pain patients, whether the patients are at high risk or low risk for CAD. In fact, even though only a small percentage of low-risk patients demonstrated significant obstructive CAD in this study, at this time and with the limited amount of published information available, it would appear to be prudent to perform provocative coronary artery noninvasive testing in most patients with significant chest pain while they are still in the hospital or soon after discharge, even if the hospital electrocardiograms and biochemical testing have been normal. However, the results reported in the Winchester paper would justify not performing noninvasive provocative CAD testing in extremely low-risk chest pain patients. Still, these patients should be carefully followed as outpatients and provocative testing should be performed if significant chest pain recurs. ■

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ABSTRACT & COMMENTARY

Inferior Vena Cava Filters and Recurrent Pulmonary Embolism

By Samuel Nadler, MD, PhD

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

SYNOPSIS: Placement of retrievable inferior vena cava filters in individuals with concurrent deep vein thrombosis does not reduce the risk of recurrent pulmonary embolism.

SOURCE: Mismetti P, et al. Effect of a retrievable inferior vena cava filter plus anticoagulation vs anticoagulation alone on risk of recurrent pulmonary embolism: A randomized clinical trial. *JAMA* 2015;313:1627-1635.

Inferior vena cava (IVC) filters have increasingly been used as add-on therapy in patients with pulmonary embolism (PE), particularly if there is an additional clot burden in the legs, leading to concerns that an additional embolism would be life-threatening. However, there are no data from randomized, controlled trials to support this intervention. The PREPIC2 study is a randomized, open-label, blinded endpoint trial that specifically addresses this question. From August 2006 to January 2013, 399 patients with acute symptomatic PE associated with persistent lower limb venous thrombosis who were at high risk for clinical decompensation were randomized to retrievable IVC filter placement for 3 months, plus systemic anticoagulation for 6 months vs anticoagulation alone. High risk was defined as having, in addition to PE, one of the following: active cancer, chronic cardiac or respiratory insufficiency, ischemic stroke within the last 6 months, deep venous thrombosis (DVT) within the iliocaval segment or bilateral DVTs, signs of right ventricular (RV) strain or myocardial injury, and age > 75 years. Exclusion criteria included: previous IVC filter placement, inability to place an IVC filter, full dose anticoagulation for > 72 hours before randomization, recent surgery, allergy to contrast media, creatinine > 2.04 mg/dL, pregnancy, life expectancy < 6 months, or contraindication to systemic anticoagulation. The primary outcome was fatal or symptomatic pulmonary embolism recurrence at 3 months. Secondary outcomes included a 6-month time point, rates of major bleeding or death from any cause, and filter complications such as infection, hematoma formation, malposition of the IVC filter, or penetration of the IVC.

At 3 and 6 months, there were no statistically significant differences in rates of recurrent fatal or symptomatic PE between the group that received both an IVC filter and anticoagulation vs anticoagulation alone (6% vs 3%, $P = 0.50$, and 7% vs 4%, $P = 0.54$, respectively). There were no differences between both groups at 3 months for secondary outcomes, such as recurrent DVT (0.5% vs 0.5%, $P > 0.99$), major bleeding (8% vs 10%, $P = 0.63$), and death (15% vs 12%, $P = 0.55$). Similarly, there were no observed differences in either primary or secondary outcomes at 6 months. The two groups were well-matched demographically and had similar rates of anticoagulation with vitamin K antagonists (83% vs 88.9%), INR (2.3 vs 2.3), duration of anticoagulation (median 182 days vs 181 days), and percentage of time spent with INR within the target range of 2-3 (58.3% vs 61.5%). Remarkably, there was a very high rate of filter retrieval at 3 months; of 193 filters inserted, 153 (79.3%) were removed. Of the 40 filters not retrieved, no attempt was made in 16 patients due to illness, filter thrombosis, patient refusal, or persistent indication for filter placement. Only 11 patients had failure of the filter to be retrieved, three due to adherence to the IVC wall and eight due to a tilted position of the filter.

■ COMMENTARY

Since their introduction, the rates of IVC filter placement for both DVT and PE have increased dramatically.[1] Since the introduction of removable IVC filters around 2001, rates of placement have further risen three-fold. Some of this increase is attributable to prophylactic placement for patients at high risk in whom anticoagulation is contraindicated, but in many instances, IVC filters are placed in individuals with PE who have persistent lower extremity DVTs when there is concern that additional embolism will lead to hemodynamic decompensation. The Mismetti et al study questioned the efficacy of this practice, and no benefit was observed with retrievable IVC filter placement.

Previous studies had demonstrated mixed results with non-retrievable IVC filters. Published in 1998, the first PREPIC study demonstrated a decreased rate of PE at 12 days after filter placement (1.1% vs 4.8%, $P = 0.03$) in patients with DVTs, but no difference at 2 years in symptomatic PE (3.4% vs 6.3%, $P = 0.16$). However, there was a significant increase in the rates of recurrent DVT (20.8% vs 11.6%, $P = 0.02$).[2] An 8-year follow-up study of this group demonstrated decreased rates of symptomatic PE (6.2% vs 15.1%, $P = 0.008$), but an increased rate of recurrent DVT (35.7% vs 27.5%, $P = 0.042$) without changes in mortality.[3] In that study, anticoagulation was mandated for 3 months only, but 61% of patients with IVC filters placed were anticoagulated at 8 years. There was concern the device was causing recurrent DVTs, and this prompted the notion that retrievable filters would prevent early PE recurrence but avoid long-term DVTs.

The results of the current study seem to indicate that placement of a retrievable IVC filter does not improve outcomes in patients with PE at high risk for decompensation. It should be noted this study was powered with the assumption of an 8% incidence of mortality at 3 months, and the study demonstrated a far lower rate. Thus, it was underpowered for its primary outcome, and the study was terminated at an interim analysis due to futility. There may be subsets of patients in whom retrievable IVC filters may change mortality. However, this study specifically included those patients at highest risk for decompensation, including patients with residual proximal DVT (69%) and with RV strain (66%), and found no benefit. Even in patients who seem most vulnerable to additional embolism, retrievable IVC filter placement plus anticoagulation did not improve outcomes vs anticoagulation alone. ■

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ABSTRACT & COMMENTARY

Long-term Weight Loss Rivals Medications and Ablation for AF Rhythm Control

By Cara Pellegrini, MD

Assistant Professor of Medicine, UCSF; Cardiology Division, Electrophysiology Section, San Francisco VA Medical Center

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

SOURCE: Pathak RK, et al. Long-term effect of goal-directed weight management in an atrial fibrillation cohort: A long-term follow-up study (LEGACY). *J Am Coll Cardiol* 2015;65:2159-2169.

Atrial fibrillation (AF) — a condition affecting millions of people — currently has no cure. Symptoms are managed via medications, ablation, and, in select cases, with pacemaker implantation and ablation of the AV node. The rhythm control strategy often leads to disappointing results, with meta-analysis data suggesting only slightly better than half the patients undergoing AF ablation are "free of AF" at just over a year mean follow-up. Antiarrhythmic drug results are generally worse. Further, the risks of procedural complications, drug side effects, and toxicities must be recognized. Recently, there have been several publications from Dr. Sanders and colleagues in Australia examining the effects of weight loss and risk factor management (including blood pressure control, diabetic control, and treatment of sleep apnea) on symptomatic AF burden that have been promising, but follow-up has been short. Their latest effort looks at longer-term follow-up of weight loss and effects of weight fluctuation on AF rhythm control.

The LEGACY study reports on the four-year mean follow-up of 825 obese patients with AF who were offered weight management. Patients were grouped according to degree of weight loss success: group 1 (≥ 10%), group 2 (3-9%), group 3 (< 3%). AF outcomes were measured by 7-day ambulatory monitors. Symptoms were assessed with the well-validated AF severity scale; in addition to weight, blood pressure, metabolic, and inflammatory markers, echocardiographic parameters were followed. Weight loss ≥ 10% was associated with a six-fold greater probability of arrhythmia-free survival compared to the other two groups. At final follow-up, 45.5% of group 1 were free of AF without the aid of ablation or antiarrhythmic medications. Almost double that (86.2%) were AF free with the addition of ablation(s) ± antiarrhythmic drugs (only 10% of patients in group 1 were on an antiarrhythmic at follow-up). Weight loss was associated with a dose response benefit in metabolic and inflammatory markers, blood pressure control, and need for anti-hypertensive medications, and even echocardiographic parameters. For example, left atrial volume fell from 37.6 mL/m^2 to 30.9 mL/m^2 in group 1. Notably, weight fluctuation of > 5% partially offset the benefit of weight loss, with a two-fold increased risk of arrhythmia recurrence compared to those with < 2% weight fluctuation. The authors conclude long-term sustained weight loss was possible and was associated with a significant reduction in AF burden.

■ COMMENTARY

Although obesity has not been proven causative of AF, increasing BMI values have been associated with incrementally higher AF risk. Whether obesity (and weight loss) modulates AF risk directly or via its impact on other cardiovascular risk factors is also unclear. The results of this study and others make clear that weight loss does result in reversal of negative cardiac remodeling, metabolic derangements, and symptomatic AF progression. The magnitude of the impact of weight loss is striking. Although the true AF burden was likely underestimated in this study due to the intermittent nature of the monitoring performed (across all groups), this was not different than many past studies of ablation and antiarrhythmic drug effect. Given the long follow-up duration in this study, weight loss alone compares quite favorably with other strategies for AF rhythm control.

It is important to recognize that participation in

a weight management clinic greatly enhanced the likelihood of sustained weight loss in this study. Eighty-four percent of those in the ≥ 10% weight loss group chose to attend this clinic as opposed to 57% in the 3-9% weight loss group and 30% in the < 3% weight loss group. Similar trends were seen for weight fluctuation, with smaller numbers of patients who participated in the clinic showing > 5% weight fluctuation. Thus, the recommendation our patients hear from us should not only be an admonishment to lose weight, but a referral to a program to maximize the patient's success in this long-term endeavor.

Obviously, weight loss and risk factor modification generally at any point is beneficial. Previous results have shown that weight loss concurrent with AF ablation enhances the results of the ablation. The superiority of the ≥ 10% weight loss group in the "total AF freedom," which included effect of ablation (performed in similar numbers across groups), medications, and weight loss, echoed that. The tantalizing prospect is the idea that weight loss and general risk factor modification can be a practical long-term strategy alone. In our clinic, we are pushing patients to have their sleep apnea treated and blood pressure controlled prior to consideration of ablation. Perhaps we should be giving more attention to a well-resourced weight loss effort early on as well. ■

PHARMACOLOGY UPDATE

Cholic Acid Capsules (Cholbam®)

By William T. Elliott, MD, FACP, and James Chan, PharmD, PhD

Dr. Elliott is Chair, Formulary Committee, Northern California Kaiser Permanente; and Assistant Professor of Medicine, University of California, San Francisco. Dr. Chan is Pharmacy Quality and Outcomes Manager, Kaiser Permanente, Oakland, CA

Drs. Elliott and Chan report no financial relationships relevant to this field of study.

The FDA has approved cholic acid in oral capsule form to treat two rare bile acid synthesis disorders. Patients with these inborn errors of metabolism lack the enzymes to synthesize cholic acid. As a result, the buildup of toxic bile acid intermediaries leads to manifestations of liver disease (cholestasis, giant cell hepatitis, and cirrhosis).[1]

INDICATIONS

Cholic acid is indicated for the treatment of bile acid synthesis disorders due to single enzyme defects (SED), and as adjunctive treatment of peroxisomal disorder (PD), including Zellweger spectrum disorder in patients who exhibit manifestation of liver disease, steatorrhea, or complications from decreased fat-soluble vitamin absorption.[2]

DOSAGE

The recommended dose is 10 to 15 mg/kg once daily or in two split doses.[2] In patients with concomitant familial hypertriglyceridemia, the dose is 11 to 17 mg/kg once daily or in two split doses and is adjusted based on clinical response. Liver function (aspartate aminotransferase [AST], alanine aminotransferase [ALT], bilirubin, international normalized ratio, etc.) should be monitored monthly for the first 3 months and every 3 months for the next 9 months, every 6 months for the next 3 years, then annually thereafter. If liver function does not improve after 3 months of treatment or if there is evidence (laboratory or clinical) of worsening liver function, treatment should be discontinued. Cholic acid capsules are available as 50 mg and 250 mg capsules.

POTENTIAL ADVANTAGES

The cholic acid capsule is the first FDA-approved treatment for these rare bile acid synthesis disorders.[1]

POTENTIAL DISADVANTAGES

The safety and effectiveness of cholic acid on extrahepatic manifestations of bile acid synthesis have not been established.[2] Cholic acid may cause decreased liver function in those without baseline liver impairment or exacerbation of liver impairment in those with.[2]

COMMENTS

The principle of exogenous cholic acid administration for these metabolic disorders is to establish an adequate pool of bile acids to improve micellar solubilization of fats and fat-soluble vitamins, stimulation of bile flow, and inhibition of cholesterol 7alpha-hydroxylase, reducing the production of toxic metabolites from cholesterol.[3] The clinical evidence supporting FDA approval involved a non-randomized, single-arm, open-label trial of 50 subjects with SED and 29

Continued on page 119

Clinical Briefs

By Louis Kuritzky, MD

We're Going to Be Hearing a Whole Lot More About NAFLD

SOURCE: Rinella ME. Nonalcoholic fatty liver disease: A systematic review. *JAMA* 2015;313:2263-2273.

Epidemiologic insights about disease prevalence might sometimes be perceived as belaboring the obvious. After all, who doesn't know that obesity has become an epidemic, that diabetes prevalence continues to rise unabated, and that hepatitis C is currently the most common cause of end-stage liver disease. Nevertheless, new epidemiologic sirens may sometimes awaken our motivation to address what might otherwise remain silent health burdens, with nonalcoholic fatty liver disease (NAFLD) being an excellent case-in-point.

Even the moniker "NAFLD" presumes we might automatically consider alcohol to be the default cause of fatty liver disease. While that might have been the case decades ago, the dual burdens of obesity and diabetes — both of which are direct antecedents to NAFLD — have changed the map of fatty liver disease on a global basis.

NAFLD portends important downstream consequences. Up to 30% of people with NAFLD have steatohepatitis, among whom approximately 20% will ultimately progress to cirrhosis. Since as many as 75 million to 100 million U.S. adults have NAFLD, this presents an epidemiologically compelling burden. Lifestyle intervention, when it leads to weight loss, is successful in improving liver pathology. There is some suggestion that independent of weight loss, a Mediterranean diet may have particular advantage.

Although no medication has been FDA approved to treat nonalcoholic steatohepatitis, some clinical data are supportive of pioglitazone (30 mg/d) or vitamin E (800 IU/d). Clinicians should maintain vigilance as recommendations for identification and management of NAFLD evolve. ■

Dietary Fat Used to Be the 'Bad Guy'

SOURCE: Mozaffarian D, Ludwig DS. The 2015 U.S. Dietary Guidelines: Lifting the ban on total dietary fat. *JAMA* 2015;313:2421-2422.

In March 2015, the Dietary Guidelines Advisory Committee (DGAC) released its report for review by the secretaries of Agriculture and Health and Human Services. The 2015 Dietary Guidelines for Americans will be derived from the DGAC report, and some clinicians may be surprised at new directions suggested by the DGAC.

For instance, dietary cholesterol has been eliminated as a "nutrient of concern" based on recent data clarifying the lack of a relationship between dietary cholesterol and cardiovascular (CV) events. Similarly, previous guidance suggested an upper limit on total dietary fat consumption; in contrast, the current DGAC report neither restricts dietary fat nor lists fat as a "nutrient of concern," based on the observation that reducing total fat has not been shown to improve CV outcomes.

Earlier guidance, which suggested limiting fat in the diet, often resulted in substitutions with increased amounts of carbohydrates, resulting in dietary modifications that commonly contained highly processed carbohydrates (such as added sugar.)

The new report includes advice that Americans consume excessive amounts of refined grain produces, such as white bread chips, white rice, crackers, and bakery goods. The U.S. populace has had more than a decade to ingrain the concept that dietary fats are "the bad guy." It will likely take a substantial amount of additional effort to clarify that replacement of fats with refined carbohydrates is not a healthful tradeoff. ■

Risks of Digoxin Use in Atrial Fibrillation

SOURCE: Washam JB, et al. Digoxin use in patients with atrial fibrillation and adverse cardiovascular outcomes: A retrospective analysis of the Rivaroxaban Once Daily Oral Direct Factor Xa Inhibition Compared with Vitamin K Antagonism for Prevention of Stroke and Embolism Trial in Atrial Fibrillation (ROCKET AF). *Lancet* 2015;385:2363-2370.

Beta-blockers, calcium channel blockers, and digoxin are among the commonly used choices for rate control in patients with atrial fibrillation (AF). The role of digoxin is based on limited data, most of which is not recent. Large clinical trials of novel anticoagulants for patients with AF have been completed within the last decade. Since a substantial minority of patients enrolled in AF anticoagulant trials were receiving digoxin as part of their therapeutic regimen, these data provide a window of observation about associations of digoxin with outcomes.

The Rivaroxaban Once Daily Factor Xa Inhibition Compared with Vitamin K Antagonism for Prevention of Stroke and Embolism Trial in Atrial Fibrillation (ROCKET-AF) enrolled more than 14,000 patients with AF to compare rivaroxaban with warfarin. More than 5000 AF patients were being treated with digoxin at baseline (37% of total ROCKET-AF participants).

At a median follow-up of approximately 2 years, digoxin treatment was associated with increased all-cause mortality (17% relative increase), vascular death (19% relative increase), and sudden death (36% relative increase).

Because these results have been obtained from a post-hoc analysis of a clinical trial, they cannot be regarded as definitive. Nonetheless, the results should prompt reconsideration of the various choices available for rate control in AF, and hopefully will stimulate performance of a randomized trial to provide more conclusive evidence. ■

Continued from page 117

with PD.[2] In addition, there was an extension trial of 12 new subjects (10 SED and 2 PD) and 31 rolled over from the first study, and lastly 18 patients from case reports or case series (15 SED and 3 PD). Due to the nature of the disease and the lack of a placebo group, a post-hoc method was used to assess efficacy relative to baseline.[3] These include 1) improvement in laboratory criteria (e.g., ALT, AST, total bilirubin, normalization of prothrombin time, no evidence of cholestasis) and 2) weight increased or stable at > 50th percentile and alive at the last follow-up. The average treatment duration was 310 weeks for SED and 254 weeks for PD. Sufficient data to evaluate treatment effect were available from 44 SED subjects and 24 PD subjects. Responders were defined as having at least two of the laboratory criteria and were alive at the last follow-up, or at least one laboratory criteria, as well as body weight and survive for at least 3 years on treatment. The rates of response were 64% for SED and 46% for PD. The majority of SED subjects (84%) had a deficiency of 3beta-hydroxysteroid dehydrogenase. The drug was well tolerated, with diarrhea being the most commonly reported adverse event (3%).

CLINICAL IMPLICATIONS

The cholic acid capsule is the first approved treatment of SED and PD. The FDA is recommending a prospective, long-term observational study in a routine clinical setting of patients aged 3 weeks or older with SED or PD who exhibit manifestation of liver disease, steatorrhea, fat soluble vitamin deficiency, or a neuropathic process related to vitamin deficiency.[3] At a body weight of 20-30 kg and a dose of 15 mg/kg/day, the 90-day wholesale cost is $74,700. ■

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CME QUESTIONS

1. **The yield of noninvasive cardiovascular testing in low-risk patients presenting with chest pain to the ED:**
   a. was moderately significant.
   b. was quite low.
   c. was quite low but was high enough to justify the cost of noninvasive cardiovascular testing if the patient has one or more cardiac risk factors.

2. **Unlike many previous studies of inferior vena cava filters for the prevention of pulmonary embolism, the PREPIC2 study:**
   a. included patients with contraindications to anticoagulation.
   b. showed about an 80% rate of filter retrieval.
   c. excluded patients with coexistent proximal deep venous thrombosis.
   d. excluded patients with right ventricular strain.
   e. excluded patients with systolic blood pressures < 100 mmHg.

3. **What percent weight loss is associated with a significant prolongation of time free from atrial fibrillation in AF patients?**
   a. < 3%
   b. 3-6%
   c. 6-10%
   d. > 10%

[IN FUTURE ISSUES]

Glycemic Control and CV Outcomes in Type 2 Diabetes

Can the Bacteria in Your Gut Explain Your Mood?

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.

What are the Clinical Implications of This Rhythm?

Interpret the 3-lead rhythm strip shown in the figure. This tracing was obtained from a 69-year-old woman with a long-term history of palpitations. Her symptoms had been increasing over recent weeks, in association with "chest tightness" and dizziness. Her prior medical history was benign, and she was hemodynamically stable at the time this tracing was recorded. How would you interpret this rhythm strip? What are the clinical implications of this rhythm?

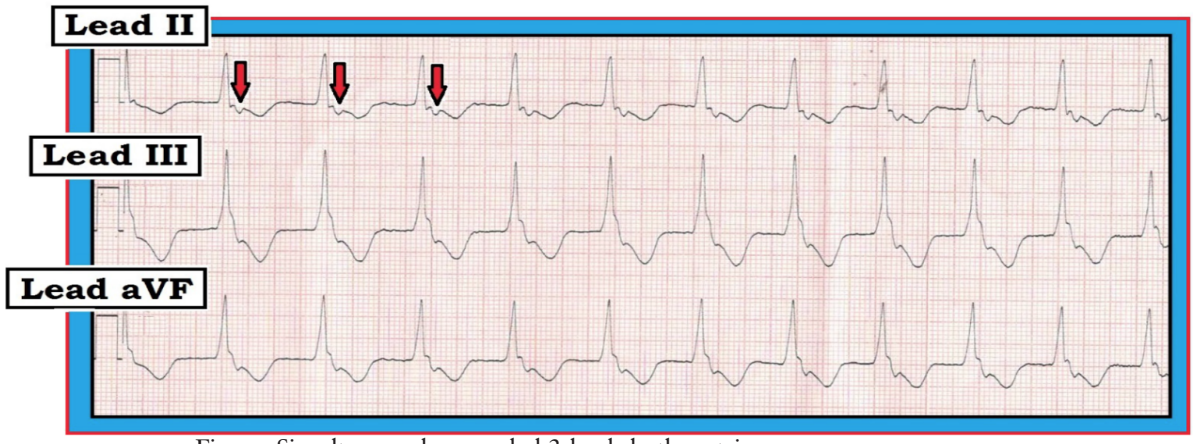

Figure: Simultaneously recorded 3-lead rhythm strip.

Interpretation: The rhythm is fairly regular, albeit with slight variation in rate.
• The QRS complex is wide (at least 0.12 second in duration).
• Normal sinus P waves are missing in lead II. Instead, there are retrograde (negative) P waves that are clearly seen occurring after the QRS in lead II (red arrows).
• The rhythm is accelerated idioventricular rhythm (AIVR), which is a slower form of ventricular tachycardia (VT).

AIVR: AIVR is an "enhanced" ventricular ectopic rhythm that occurs at a faster rate than the usual 20-to-40/minute intrinsic ventricular escape pacemaker. The range of AIVR is typically between 60-to-110/minute, which is slower than "fast" VT that usually does not cause hemodynamic disturbance below rates of 130/minute. This leaves an "overlap range" for AIVR vs fast VT when the ventricular rate is between 110-to-130/minute.

• AIVR generally occurs in one of the following clinical settings: 1) as a rhythm during cardiac arrest, 2) in the monitoring phase of acute myocardial infarction, or 3) as a reperfusion arrhythmia (following medical thrombolysis, acute angioplasty, or spontaneous reperfusion). It may also occur in patients with underlying coronary disease, cardiomyopathy and with digoxin toxicity, and rarely in otherwise healthy subjects without underlying heart disease.
• AIVR is often an "escape rhythm" in that it arises because both the sinoatrial (SA) and atrioventricular nodes are not functioning. If treatment is needed (because loss of the atrial kick results in hypotension), atropine is the drug of choice (in hope of speeding up the SA node to resume its pacemaking function). AIVR should not be shocked nor treated with antiarrhythmic medication, since doing so might result in asystole.

We do not know why the patient in this case presented with long periods of AIVR. Recent or remote ischemia/infarction and cardiomyopathy should be ruled out. The possibility of sick sinus syndrome with emergence of AIVR as an escape rhythm also should be considered as part of the workup.

NOTE: Please see ***http://tinyurl.com/KG-Blog-107*** for additional explanation of this case. The tracing discussed here is Figure 3 in the blog.