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Internal Medicine

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

Vol. 39, Issue 7, April 15, 2017
AHCMedia.com

ABSTRACT & COMMENTARY

Can Magnesium Cure Nocturnal Leg Cramps?

By Seema Gupta, MD, MSPH

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

SYNOPSIS: A small randomized, double-blind, placebo-controlled trial demonstrated that oral magnesium oxide was not superior to placebo for older adults suffering from nocturnal leg cramps.

SOURCE: Maor NR, Alperin M, Shturman E, et al. Effect of magnesium oxide supplementation on nocturnal leg cramps: A randomized clinical trial. *JAMA Intern Med*. Published online Feb. 20, 2017. doi:10.1001/jamainternmed.2016.9261. [Epub ahead of print].

Nocturnal leg cramps (NLC) are sudden, involuntary, painful, and palpable muscle contractions lasting seconds to minutes, occurring most often at night. They are common, occurring in nearly 50% of adults > 50 years of age.[1] There is no gender preference, but the prevalence and frequency increases with age. An exact understanding of the mechanism is lacking. Therefore, most cases of NLC occurring in adults seem to be idiopathic, but there can be potential predisposing factors. These include metabolic disorders such as hemodialysis and electrolyte imbalance; neurologic, endocrine, and vascular disorders; and prolonged sitting or inappropriate leg positioning. Certain medications, such as beta-agonists, beta-blockers with intrinsic sympathomimetic activity, potassium-sparing diuretics, angiotensin II receptor antagonists, benzodiazepines, oral contraceptives, and thiazide-like diuretics, also may be associated.

Quinine has been among the most studied pharmacotherapy for NLC and has been found to be low to moderately effective in reducing the frequency and intensity of NLC attacks.[2] However, the FDA has issued warnings about the use of quinine for NLC because of the associated risk of serious and life-threatening side effects, such as cardiac arrhythmias, thrombocytopenia, and severe hyper-

Financial Disclosure: *Internal Medicine Alert's* Physician Editor Stephen Brunton, MD, is a retained consultant for Abbott Diabetes, Actavis, AstraZeneca, Becton Dickinson, Boehringer Ingelheim, Cempra, Janssen, Lilly, Merck, Novo Nordisk, Sanofi, and Teva; he serves on the speakers bureau of AstraZeneca, Boehringer Ingelheim, Janssen, Lilly, Novo Nordisk, and Teva. Contributing Editor Louis Kuritzky, MD, is a retained consultant for and on the speakers bureau of, Allergan, Daiichi Sankyo, Lilly, and Lundbeck. Peer Reviewer Gerald Roberts, MD; Editor Jonathan Springston; and Executive Editor Leslie Coplin report no financial relationships relevant to this field of study.

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Internal Medicine Alert,
ISSN 0195-315X, is published twice a month by AHC Media, a Relias Learning company
111 Corning Road, Suite 250
Cary, NC 27518

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

POSTMASTER: Send address changes to *AHC Media, LLC, P.O. Box 74008694, Chicago, IL 60674-8694*

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sensitivity reactions.[3] On the other hand, magnesium is used commonly for NLC, and, other than diarrhea occurring with high doses, oral magnesium supplements generally are considered safe and relatively free of adverse effects. Although initial trials demonstrated the effectiveness of magnesium in treating NLC in pregnant women, subsequent studies have failed to demonstrate efficacy in older adults.[4] However, none of these studies have used oral magnesium oxide, and there is some evidence that, as opposed to magnesium citrate, magnesium oxide may be able to increase intracellular magnesium levels.

Maor et al hypothesized that the oral magnesium oxide supplementation may reduce the frequency and severity of NLC and subsequently improve quality of life and quality of sleep for older adults. The researchers conducted a randomized, double-blind, placebo-controlled clinical trial in northern Israel where 94 adults (39% male; mean age, 65 years) with four or more NLCs during the previous two weeks were randomized to once-nightly magnesium oxide (865 mg; equivalent to 520 mg of elemental magnesium) or placebo for four weeks. During the treatment period, both the magnesium and placebo groups experienced reductions in NLC episodes, from about eight weekly to five weekly. At four weeks, no significant differences were found between the two groups in severity or duration of NLCs or in quality of sleep or quality of life. No significant adverse effects were attributed to either magnesium or placebo. The authors concluded that oral magnesium oxide was not superior to placebo for older adults experiencing NLC.

■ COMMENTARY

NLCs can cause substantial distress and sleep disruption for many Americans. In practice, physicians often have very little to offer in terms of either a clear explanation of the etiology or treatment options. Often, preventive recommendations include stretching exercises for the affected muscle group, avoiding dehydration, and addressing secondary predisposing factors. Minerals and vitamin supplementation, including vitamin B complex and iron supplementation in iron deficiency states, also have demonstrated some benefits. Similarly, other pharmaceutical options may include trials of diphenhydramine, calcium channel blockers, and gabapentin to avoid the routine use of quinine because of its safety risk profile. In the Maor et al study, oral magnesium oxide was not found to be superior to placebo in treatment of NLC. However, it is noteworthy that there was a decrease in the mean number of NLCs per week from approximately eight to five in both groups, suggesting a rather significant placebo effect, which also may explain the wide use of magnesium by both patients and physicians. Clinically, it is an interesting finding that we must keep in mind when deciding whether to recommend magnesium for NLC, even though it was not found to be superior to placebo. ■

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ABSTRACT & COMMENTARY

Pioglitazone Improves Fibrosis Scores in Nonalcoholic Steatohepatitis Patients With and Without Diabetes

By David Fiore, MD

Professor of Family Medicine, University of Nevada, Reno

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

SYNOPSIS: In a meta-analysis of eight studies (five pioglitazone, three rosiglitazone) involving 516 patients with biopsy-proven nonalcoholic steatohepatitis, pioglitazone was found to improve fibrosis between six and 24 months. The clinical significance and potential harms of this treatment remain to be determined.

SOURCE: Musso G, Cassader M, Paschetta E, Gambino R. Thiazolidinediones and advanced liver fibrosis in nonalcoholic steatohepatitis: A meta-analysis. *JAMA Intern Med*. Published online Feb. 27, 2017. doi: 10.1001/jamainternmed.2016.9607. [Epub ahead of print].

Nonalcoholic fatty liver disease (NAFLD) ranges from a benign incidental finding to nonalcoholic steatohepatitis (NASH) and eventually can lead to fibrosis and liver failure. Unfortunately, there is no established treatment for NASH, which is the second leading cause of liver transplantation.[1-3] Previous studies have demonstrated that early NASH (NASH Clinical Research Network Scale F0-F2) has minimal effect on morbidity and mortality, but advanced fibrosis (F3-4) predicts poor outcomes.[4,5] Reversal of fibrosis has been demonstrated with improvement of associated clinical conditions and weight loss, but there are no approved treatments directed at NASH specifically.[6] Based on studies in diabetic patients and a prior meta-analysis by one of the current authors, the current meta-analysis was designed to assess the effect of thiazolidinedione therapy in NASH with advanced fibrosis.[6]

Using an inclusive search strategy, the authors identified 12,553 studies, of which 216 unique publications met inclusion criteria. Of those, only 16 were randomized, controlled trials assessing thiazolidinedione treatment of NAFLD. Only eight included post-treatment histological assessment. Of those eight studies, five were on pioglitazone and three on rosiglitazone, and included 516 patients. The primary outcome was improvement in advanced fibrosis, defined as an improvement in the NASH Clinical Research Network Scale from F3-4 to F0-2. Secondary outcomes were at least a one-point improvement in fibrosis of any stage and NASH resolution. Adverse effects of thiazolidinedione therapy, including weight gain, lower limb edema, congestive heart failure, bone fractures, cancer, and anemia, also were examined. Studies were abstracted by two of the authors independently, and agreement was good to excellent, with K statistics of 0.88 for study selection and 0.92 for quality assessment. Six trials were believed to contain low risk of bias (the two trials with higher risk of bias both evaluated rosiglitazone). Statistical heterogeneity was low for all outcomes evaluated.

Pooled results of the randomized, controlled trials demonstrated that thiazolidinedione therapy was associated with improvement in advanced fibrosis, and the effect size was significant — both when looking at all patients with NASH and only patients with NASH and advanced fibrosis at baseline. Further, patients with and without diabetes had similar improvements, and the observed beneficial effects of thiazolidinedione were restricted to the effects of pioglitazone.

As for adverse effects, thiazolidinedione therapy was associated with a mean 2.7% weight gain compared with controls and a higher odds ratio for lower limb edema (2.36) without any significant difference in agents, randomized, controlled trials, or trial duration. There was no increase in congestive heart failure, but this outcome was reported in only half the studies.

■ COMMENTARY

In addition to the exciting findings from this study, this meta-analysis has some strengths that often are missing from other meta-analyses. Primary among them are the high quality of the studies overall and the heterogeneity of the studies included. On the other hand, only 516 patients were included in the eight studies, and only 197 of those patients were on pioglitazone. Although it's impressive that the authors found a benefit from treatment with pioglitazone, the small numbers do not allow discussion of treatment harms (other than the documented weight gain) with any confidence. This is especially concerning given the FDA's recent warning that pioglitazone may be associated with bladder cancer, as well as concerns about congestive heart failure.[7,8] Although the finding that pioglitazone reduced fibrosis in patients with NASH is very exciting, it must be tempered with the realization that we

still do not know if it improves clinical outcomes such as ascites, encephalopathy, the need for liver transplant, or death rates. Even if pioglitazone can reduce liver-related complications of NASH, many of these patients have significant comorbidities that may affect their lives regardless of regression of liver fibrosis. Therefore, quality of life and overall mortality studies will be required to address this issue.

Our quandary is how to treat patients with NASH, given that progression is variable, there are no approved medical treatments, lifestyle changes are effective but difficult, and NASH with advanced fibrosis often is fatal. Clearly, the first step in all patients with NASH is aggressive lifestyle changes, with a focus on alcohol avoidance and weight loss.[9] After that, the question remains: Which patients are likely to benefit from drug treatment, specifically with pioglitazone, given the lack of data about patient-oriented outcomes, such as ascites, need for transplant, and death, and the lack of information about adverse effects? It seems that a reasonable approach is to start (or continue) pioglitazone in diabetic patients with NASH and advanced fibrosis while holding off on starting pioglitazone in those with less advanced NASH unless there is another indication. Patients with advanced fibrosis who are not diabetic present an especially difficult dilemma given the high probability of poor outcomes combined with the lack of convincing data about the benefits of drug treatment. In these patients, a careful discussion of the pros and cons of treatment with shared decision-making would be most appropriate.

Although this meta-analysis gives us hope, the small number of patients and lack of outcome data on adverse effects must give us pause. With any meta-analysis, it's always important to remember that "three second graders don't equal a sixth grader." That is, we need larger studies to confidently evaluate the benefits and (most importantly) the harms of new medical therapies. ■

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ABSTRACT & COMMENTARY

Polyneuropathy in the Metabolic Syndrome

By Michael Rubin, MD

Professor of Clinical Neurology, Weill Cornell Medical College

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

SYNOPSIS: The metabolic syndrome, independent of the diagnosis of diabetes, is associated with the development of polyneuropathy.

SOURCE: Hanewinckel R, Drenthen J, Ligthart S, et al. Metabolic syndrome is related to polyneuropathy and impaired peripheral nerve function: A prospective population-based cohort study. *J Neurol Neurosurg Psychiatry* 2016;87:1336-1342.

Updated by the International Diabetes Federation in 2006, criteria for diagnosing metabolic syndrome include central obesity based on waist circumference in addition to abnormalities in two of the following: triglycerides, (> 150 mg/dL), high-density lipoprotein cholesterol (HDL-C; < 40 mg/dL for men or < 50 mg/dL for women), blood pressure (systolic ≥ 130, diastolic ≥ 85), and fasting plasma glucose (≥ 100 mg/dL). Elevated fasting glucose and impaired glucose tolerance may increase the risk of polyneuropathy in prediabetes. What role, if any, apart from diabetes or prediabetes, does metabolic syndrome play as a risk factor for polyneuropathy?

Incorporated as part of the Rotterdam Study, a prospec-

tive, population-based cohort study, initiated in 1990 of all inhabitants ≥ 55 years of age living in the Ommoord district of Rotterdam, the Netherlands, and expanded in 2006 to include all persons ≥ 45 years of age, a polyneuropathy screen was implemented in June 2013, and the current study included patients enrolled up to October 2015. Among 1,544 persons screened for polyneuropathy, 234 were excluded for logistical reasons, with 1,256 of the remaining 1,310 lacking adequate information to be included in this analysis. Metabolic syndrome was diagnosed based on any three of the following five criteria: increased waist circumference (≥ 94 cm for males, ≥ 80 cm for females), elevated triglycerides, reduced HDL-C (< 39 mg/dL in males, < 50 mg/dL in females, or specific treatment for reduced HDL-C), elevated blood pressure, and elevated fasting glucose (≥ 216 mg/dL, or use of glucose-lowering medication). Neuropathy screening included a symptom questionnaire, neurological examination, and nerve conduction studies of the sural sensory nerves bilaterally and a unilateral peroneal motor nerve. Statistical analysis included logistic and linear regression analyses, adjusted for age, gender, and height, and splines regression for continuous glucose levels.

Among 1,256 subjects, 45.5% male (n = 571) and 54.5% female (n = 685), with mean age of 70 years, type 2 diabetes was present in 13.9%, impaired fasting glucose in 12.2%, and metabolic syndrome in 52.5% (n = 659). Definite polyneuropathy was present in 5.1% (n = 64); probable polyneuropathy in 7.3% (n = 92), diagnosed by the presence of two abnormal elements on nerve conduction studies; and possible polyneuropathy in 17.4% (n = 218), diagnosed by the presence of one abnormal or two slightly abnormal elements on nerve conduction studies. Regardless of gender, metabolic syndrome was associated with definite polyneuropathy (odds ratio, 1.92; 95% confidence interval, 1.09-3.38), particularly in those individuals who had increased waist circumference and elevated triglycerides, regardless of the presence of diabetes. Diabetes, but not impaired fasting glucose, also was strongly associated with polyneuropathy.

■ COMMENTARY

Polyneuropathy affects 2-7% of the population and is idiopathic in at least 30% of patients. Diabetes, both type 1 and 2, is the most common known cause, and although rigorous glucose control significantly reduces the incidence of neuropathy in type 1 diabetes, it does not have the same effect in type 2, implying that factors other than blood glucose are causative. Evidence now supports the notion that components of metabolic syndrome may be responsible, and efforts to address these may positively affect the incidence of neuropathy, not only in diabetes but in non-diabetic obese individuals as well. ■

PHARMACOLOGY UPDATE

Desmopressin Acetate Nasal Spray (Noctiva)

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

Dr. Elliott is Assistant Clinical Professor of Medicine, University of California, San Francisco. Dr. Chan is Associate Clinical Professor, School of Pharmacy, University of California, San Francisco

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

The FDA has approved the first treatment for nocturnal polyuria. Desmopressin is a 9-amino synthetic analog of the pituitary hormone, vasopressin. Other formulations are approved for diabetes insipidus, primary nocturnal enuresis, hemophilia A, and von Willebrand disease (type 1). It is marketed as Noctiva nasal spray.

INDICATION

Desmopressin nasal spray is indicated for the treatment of nocturia due to nocturnal polyuria in adults who awaken at least twice per night to void.[1]

DOSAGE

For patient < 65 years of age who are not at risk for hyponatremia, the dose is one spray (1.66 mcg) in either nostril nightly approximately 30 minutes before going to bed.[1] For those ≥ 65 years age or younger patients at risk for hyponatremia, the dose is 0.83 mcg nightly. Clinicians may increase the dose to 1.66 mcg after at least seven days if needed, provided serum sodium has remained normal. The nasal spray must be primed before initial use and if not used for more than three days. Desmopressin acetate is available as 1.66 mcg per spray and 0.83 mcg per spray.

POTENTIAL ADVANTAGES

Desmopressin acetate nasal spray is currently the only FDA-approved agent for this indication.

POTENTIAL DISADVANTAGES

Desmopressin can cause hyponatremia, which, in severe cases, may be life-threatening.[1] It is contraindicated in patients at risk for severe hyponatremia (e.g., history of hyponatremia, primary nocturnal enuresis, use of loop diuretics, use of corticosteroid, renal impairment, known or suspected syndrome of inappropriate antidiuretic hormone secretion).[1] The FDA states that it is not approved for all causes of night-time urination, and clinicians

should conduct an appropriate workup before considering administration.[2]

COMMENTS

The efficacy and safety of desmopressin were evaluated in two 12-week randomized, double-blind, placebo-controlled trials in adults (at 50 years of age) with nocturia due to nocturnal polyuria.[1,3] This is defined as having a six-month history of at least an average of two nocturic episodes per night. In addition, they had at least 13 documented nocturia episodes over six nights during screening. In both studies nocturnal polyuria was defined as a night-time urine production exceeding one-third of the 24-hour production. In the first trial, subjects were randomized to desmopressin 1.66 mg (n = 199), 0.88 mcg (n = 209), or placebo (n = 145). In the second trial, subject distributions were 143, 145, and 145, respectively. Studies had a two-week screening period, a two-week lead-in period, and a 12-week treatment period. The two co-primary efficacy endpoints were change in the mean number of nocturic episodes per night from baseline during the 12-week period and the percentage of subjects who achieved at least a 50% reduction from baseline. In the first trial, the 0.88 mcg dose did not achieve statistical significance with both co-primary endpoints. In the second trial, the lower dose was better than placebo in terms of mean reduction of episodes, but it did not achieve statistical significance with at least 50% reduction from baseline.[3] From a baseline of a mean of 3.2-3.4 episodes per night, desmopressin 1.66 mg showed a reduction of 0.3-0.4 compared to placebo (9-12% reduction). For the co-primary endpoint, 47% achieved a 50% reduction, compared to 27% in the first trial, and 49% and 29%, respectively, for the second trial (absolute difference of 21% and 20%). In the first trial, the data suggest that some subjects responded to the lower dose similarly to those treated with the higher dose.[2] This may be related to large inter-individual variation in systemic exposure to desmopressin. Because of this variation and greater risk of hyponatremia with the higher dose, both doses were approved.

CLINICAL IMPLICATIONS

Desmopressin is the first drug approved for nocturia due to nocturnal polyuria, defined as overproduction of urine at night. The cause may be idiopathic or due to other conditions such as edema-associated states.[2] Desmopressin acetate appears to provide clinically meaningful but modest benefit.[2] The price was not available at the time of this review. ■

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CME QUESTIONS

1. **Based on the study by Maor et al, which of the following is true about use of magnesium in treatment of nocturnal leg cramps.**
   a. Oral magnesium oxide is as effective as placebo.
   b. Oral magnesium oxide is superior to placebo.
   c. Oral magnesium citrate is as effective as placebo.
   d. IV magnesium citrate is superior to placebo.

2. **Which statement is true regarding fibrosis in patients with nonalcoholic steatohepatitis (NASH)?**
   a. Both pioglitazone and rosiglitazone were demonstrated to reduce fibrosis in patients with NASH.
   b. Pioglitazone, but not rosiglitazone, was demonstrated to reduce fibrosis in patients with NASH.
   c. Rosiglitazone, but not pioglitazone, was demonstrated to reduce fibrosis in patients with NASH.
   d. Neither pioglitazone nor rosiglitazone were demonstrated to reduce fibrosis in patients with NASH.

3. **Which of the following is true regarding metabolic syndrome?**
   a. It is associated with definite polyneuropathy regardless of the presence of diabetes.
   b. It is associated with definite polyneuropathy only in the presence of diabetes.
   c. It is associated with definite polyneuropathy only in the presence of diabetes in obese individuals.
   d. It is associated with definite polyneuropathy only in diabetic individuals with elevated triglycerides.

Clinical Briefs

By Louis Kuritzky, MD

Antisocial Behavioral Syndromes in the United States

SOURCE: Goldstein RB, Chou SP, Saha TD, et al. The epidemiology of antisocial behavioral syndromes in adulthood: Results from the National Epidemiologic Survey on Alcohol and Related Conditions-III. *J Clin Psychiatry* 2017;78:90-98.

The National Epidemiologic Survey on Alcohol and Related Conditions-III (NESARC-III) has provided data about the prevalence of antisocial behavioral syndromes in the United States. The societal burden incurred from persons with antisocial personality disorder is substantial, since the disorder is associated with impulsive aggressive behaviors for which the sufferer lacks remorse. Consequences include acts of violence as well as unstable personal, marital, and societal relationships.

From a large diverse cross-sectional population sample (n = 36,309), interviewers used a National Institute on Alcohol Abuse and Alcoholism tool known as Alcohol Use Disorder and Associated Disabilities Interview Schedule-5. This tool helps researchers identify not only alcohol misuse, but also drug use disorders, mood disorders, anxiety, and personality disorders.

This survey found that 4.3% of adults fulfilled criteria for antisocial personality disorder. Rates were highest in younger adult white and Native American males.

Treatments for antisocial personality disorder are labor intensive. Antisocial personality disorder tends to remit over time.

Whether treatment for the numerous other comorbidities, such as substance abuse associated with antisocial personality disorder, will hasten time to remission is an area of needed research. ■

Recognizing Binge Eating Disorder

SOURCE: Kornstein SG. Epidemiology and recognition of binge-eating disorder in psychiatry and primary care. *J Clin Psychiatry* 2017;78(Suppl 1):3-8.

The diagnostic criteria for binge eating disorder includes at least weekly episodes of binge eating for at least three months.

Binge eating is described as ingesting, in a circumscribed period, a significantly larger amount of food than is typical; characteristically, sufferers feel a lack of control over how much they eat during the episode, and are not driven by sustained hunger.

In contradistinction from anorexia nervosa or bulimia nervosa, post-meal purging (or other compensatory measures such as hyper exercising or excessive laxative use) is not part of the diagnosis.

Binge eating disorder is more common than anorexia and bulimia, the two most well-known eating disorders. Part of the lack of recognition of binge eating disorder stems from its relatively recent inclusion as a specific eating disorder.

There are screening tools available for binge eating disorder appropriate for use in primary care settings, and Kornstein suggested inclusion of at least one screening question in routine care about eating habits such as "Do you ever feel a loss of control over how much you eat?"

Psychological as well as pharmacologic therapies (primarily antidepressants) have been demonstrated to produce some benefit for binge eating disorder. Because binge eating disorder is more common in persons with obesity, screening potentially is more useful in this population. ■

Preventing Sickle Cell Disease Pain Crises

SOURCE: Ataga KI, Kutlar A, Kanter J, et al. Crizanlizumab for the prevention of pain crises in sickle cell disease. *N Engl J Med* 2017;376:429-439.

P-selectin is a protein within endothelial cells, megakaryocytes, and platelets that functions during inflammation to enhance adhesion of leukocytes and other cells to the site of inflammation. In sickle cell disease, platelets, erythrocytes, monocytes, and neutrophils can aggregate to compound circulatory flow problems, which lead to painful vaso-occlusive crises symptoms. In animal models of sickle cell anemia, deficiency in P-selectin (and E-selectin) is protective from vaso-occlusive episodes.

Crizanlizumab is a monoclonal antibody that blocks interaction between P-selectin and its receptor. In a double-blind, randomized, placebo-controlled trial of crizanlizumab, subjects (n = 198) who had experienced at least two sickle-cell pain crises within the prior 12 months were in enrolled and followed for one year.

Patients on crizanlizumab enjoyed a 45% reduction in painful crises compared to placebo. Adverse events potentially related to treatment included arthralgia, diarrhea, pruritus, vomiting, and chest pain. Crizanlizumab has been demonstrated to be highly effective in reducing painful crises related to sickle cell disease. ■

Internal Medicine [ALERT]

ECG Review

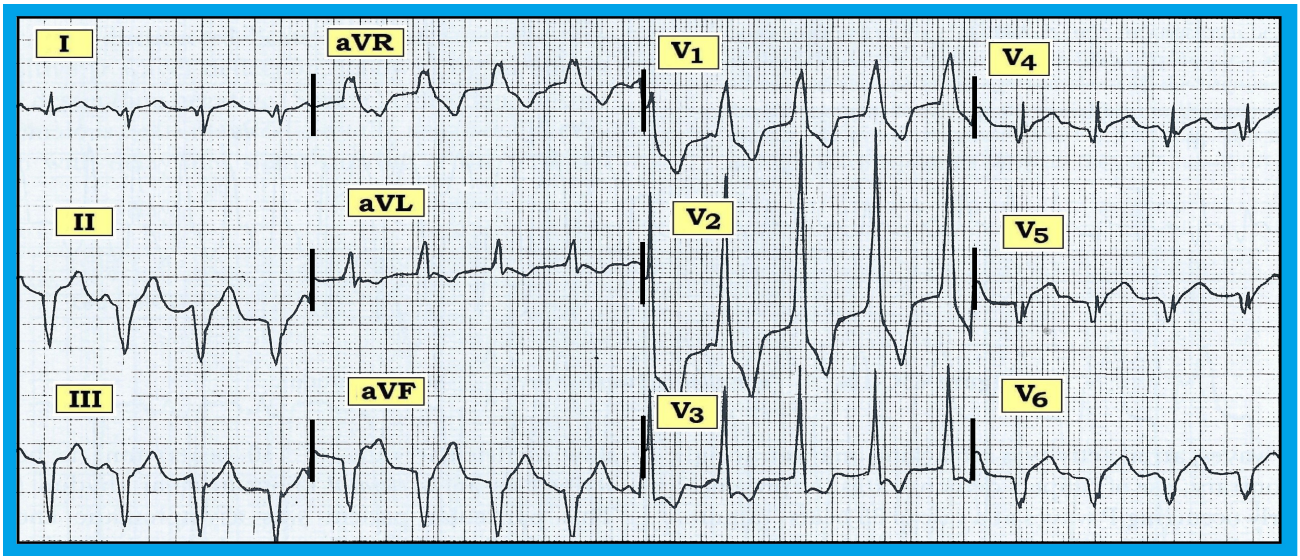
By Ken Grauer, MD

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.

Ventricular Tachycardia, Supraventricular Tachycardia with Right Bundle Branch Block, or Something Else?

The 12-lead ECG in the figure below was obtained from a hemodynamically stable older adult with new-onset palpitations. Is this ventricular tachycardia (VT)? How certain are you?



Although we were told this patient was hemodynamically stable at the time this ECG was recorded, knowing this does not help determine the etiology of the arrhythmia. Why? Some patients presenting with VT may remain alert and hemodynamically stable for a surprisingly long time (hours or even days). However, knowing this patient is stable provides an extra moment to contemplate your differential diagnosis.

Unfortunately, there is no simultaneously obtained long lead rhythm strip. Nevertheless, it should be clear that the rhythm is a regular wide complex tachycardia (WCT) at a rate of ~110/minute without clear sign of sinus-conducting P waves (i.e., there is no consistent upright P wave with constant PR interval in lead II). Thus, a ventricular etiology should be assumed until proven otherwise. Statistically, more than 80-90% of all regular WCT rhythms that lack sinus P waves will turn out to be VT.

Features that further increase the likelihood of a ventricular etiology for this tracing include: extreme axis deviation (i.e., all negative QRS in each of the inferior leads); marked QRS widening (to at least 0.14 seconds); all positive QRS in lead aVR; all negative QRS in lead V6; and QRS morphology not resembling any known form of bundle branch block or hemiblock. Based on these ECG features, the predicted likelihood of a ventricular etiology increases to more than 95%.

There is one additional clue that allows 100% certainty of a ventricular etiology in this case. Although subtle, note the presence of unmistakable P waves periodically punctuating the baseline. These are best seen in lead III (before the second QRS complex in this lead, and then notching the end of the QRS of the third beat) and in lead aVF (notching the ST segment of the first beat in aVF, as well as appearing before and after the last beat in this lead's recording). These P waves are regular and unrelated to neighboring QRS complexes, which defines this as AV dissociation.

The rate of the ventricular rhythm in the figure above is relatively slow for VT (i.e., well under 130/minute). Therefore, this rhythm is best classified as accelerated idioventricular rhythm, which is seen most commonly as an escape rhythm that occurs in association with acute or recent infarction — or following reperfusion of a major coronary artery. The need for an immediate cardioversion is rare, and the rhythm often resolves spontaneously. Clinical correlation is essential.

For further information and discussion about this case, please visit: http://bit.ly/2n3WFRI.