case report

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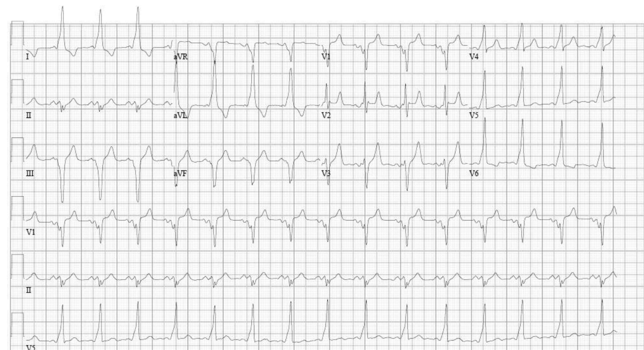
The Case of a Tell Tale Heart

Case Description

A 24-year-old Caucasian male presented late at night to the Emergency Department, complaining of "feeling sick after eating fast food" earlier in the evening. He described multiple complaints including general myalgias, productive cough without hemoptysis and nausea associated with two episodes of nonbloody emesis; most recently, he had diarrhea starting the day of presentation. He included a two-day history of left-sided pleuritic, nonradiating mild chest pain to his constellation of symptoms. He also had generalized multisystem complaints, and an almost completely positive review of systems. Interestingly enough, teased from the review of systems, the patient did report episodes of a "racing heart" during the past few days. Each of these episodes occurred at rest while he was lying in bed and involved subjective presyncope and self-resolved. He denied dyspnea and stated he was physically fit and able to tolerate physical exertion without difficulty. His review of systems was otherwise noncontributory outside of the wide range of presenting symptoms. He had no recent foreign travel or sick contacts and was still able to work as a general laborer in the construction industry. He had an unremarkable medical/surgical history. His family history was negative for major diseases, and there were no cases of sudden death or heart disease. He did not take any regular medications, except for some over-the-counter cold and cough remedies. He lived alone at home, had a girlfriend (who was healthy) and occasionally smoked and used caffeine (not noted in excess).

He was afebrile, all vital signs were within normal limits and he had an appropriate body mass index. Except for mild palpable chest wall tenderness his physical examination was normal: lungs clear to auscultation, no murmurs rubs or gallops, and no peripheral edema. His chest x-ray (both PA and lateral) was unremarkable, without cardiomegaly, effusions, infiltrates or pneumothorax. His EKG is shown below. (Figure 1) An echocardiogram was performed after electrophysiology cardiology consultation to rule out Ebstein's abnormality. The 2D-echocardiogram was normal. The patient underwent an electrophysiology cardiac study and planned ablation. He was found to have an atrioventricular (AV) node re-entry tachycardia using a posteroseptal pathway located in the CS and was ablated using a stinger G curve.

Figure 1. EKG demonstrating normal sinus rhythm at a rate of 91 beats per minute with Wolff-Parkinson-White PR interval 112 msec, posterior septal pathway.



Discussion

This is an example of a nonspecific emergency department presentation of a young, otherwise healthy person seemingly with a non-acute case of gastroenteritis, likely to self-resolve. Such a presentation could have easily been dismissed; however, because of careful examination and the interpretation of a simple, inexpensive test a potentially deadly condition was recognized and referred to the appropriate subspecialty for treatment.

Wolff-Parkinson-White syndrome (WPW) is a ventricular pre-excitation syndrome long known to medicine. In the asymptomatic population it is known

as the WPW pattern on EKG, but in the symptomatic it is know as WPW syndrome. It has been thought to occur in at least 3% of the general population. Normally the AV-node is the only pathway for electrical impulses to travel from the atria to the ventricles, but in individuals with pre-excitation syndrome there is an accessory extra-nodal atrial fiber accessory pathway or pathways.[1-4]

Very few WPW syndrome patients have a documented arrhythmia and even fewer present with sudden cardiac death. This extra circuit is known as the bundle of Kent (or an AV bypass tract), which directly transmits impulses and does not have the rate limiting effects of the normal AV-node, important as a regulator of the automaticity of the heart. Approximately 50% of people with the WPW pattern are asymptomatic and they are twice as likely to be males.[3-7]

Normal AV-node function can be monitored on an EKG by examining the P-R interval. These abnormal tracts are actually aberrant atrial tissue that can project inferiorly anywhere in the AV rings into the ventricular muscle (and are capable of carrying electrical impules). Normally a P-R interval on an EKG is 120 msec to 200 msec and starts with a normal P wave (atrial depolarization) leading to the QRS complex (ventricular depolarization). In pre-excitation syndrome, the P-R interval is affected by antegrade directional conduction. The P-R interval in WPW will be shortened (<120 msec) and will have a slurred upstroke known as the delta wave, which is perhaps the most recognizable feature of pre-excitation syndrome. (Figure 1) During the initial evaluation of WPW, the most important tasks are to:

1. Recognize the condition
2. Check the echocardiogram
3. Refer to cardiology/electrophysiology to evaluate conduction possibilities that may lead to lethal dysrhythmias

Patients are usually identified as having pre-excitation syndrome on a routine EKG done for other reasons and, most commonly, the presenting complaint does not stem from WPW itself. Most patients with the WPW pattern on EKG will present either in the first year of life or in their early twenties, a typical bimodal presentation. Most patients with WPW will never become symptomatic. Symptoms in WPW stem from the development of arrhythmias affecting cardiac output. The most common arrhythmias include the tachyarrhythmias and atrial dysregulation, such as flutter or fibrillation.[1,3,7-10]

When treating pre-excitation syndromes, it is important to rule out other associated structural abnormalities of the heart such as congenital hypertrophy, valvular disease or other known electrical conduction abnormalities. The most commonly associated aberrant pathway in myocardial conduction is Ebstein's abnormality. Up to 20% of patients' pre-excitation syndromes, such as WPW, have Ebstein's abnormality which is defined as bypass tracts found in the right free wall and posteroseptal region.[6,9]

Conclusion

For the primary care physician, WPW syndrome is most often found on a routine EKG done for other reasons. It is important to remember that it does represent an inherent abnormality in the electrical conduction system that requires further investigation. Prior to referral to a cardiologist an echocardiogram is indicated. Of course in symptomatic WPW, an urgent referral is indicated, preferably to a center with cardiac electrophysiology (EP) investigation capabilities.[9,11]

A question arises: Should we be screening for WPW in the clinic? The answer is both yes and no. There have been familial cases of WPW reported (thought to be autosomal dominant), but the genetics are still being investigated with molecular genetics research and combined EP research. Primary care should be screening in the sense that all physicians should consider WPW pattern on any interpreted EKG, in just the same way rate and rhythm is considered. As to when we should specifically investigate WPW, that comes down to clinical judgment. An EKG is a simple inexpensive test that is sensitive for a large variety of potentially life-threatening conditions. At best, an EKG can diagnose with almost certainty a potentially lethal and treatable condition such as WPW and at worst, when completely negative, can serve as a baseline for future EKGs your patient will undoubtedly require, thanks to the prevalence and incidence of coronary artery disease.[11]

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