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"I have palpitations, What Does my ECG Show"

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CASE REPORT

A 72-year-old female with past medical history of hypertension, hyperlipidemia, and asthma presents to the clinic with complaints of frequent brief episodes of palpitations. She reports that it feels like her heart is racing and beating very fast. She noticed this after using her albuterol inhaler multiple times in a short period of time. She denies any chest pain or chest pressure. No history of syncope. She is otherwise in her usual state of health. Vital signs are notable for heart rate of 59 beats per minute, blood pressure of 123/65. Cardiac physical exam is notable for regular rate and rhythm with no audible murmurs. Electrocardiogram is performed (*Figure 1*).

Questions:

1. Describe the ECG?
2. What pattern does this ECG demonstrate?
3. What is the next best step?

ELECTROCARDIOGRAM INTERPRETATION

Sinus bradycardia with heart rate of 59 beats per minute. Normal axis deviation. Short PR interval. There is a widened QRS complex with notable delta waves. Overall, ECG demonstrates sinus bradycardia with Wolff-Parkinson-White (WPW) pattern.

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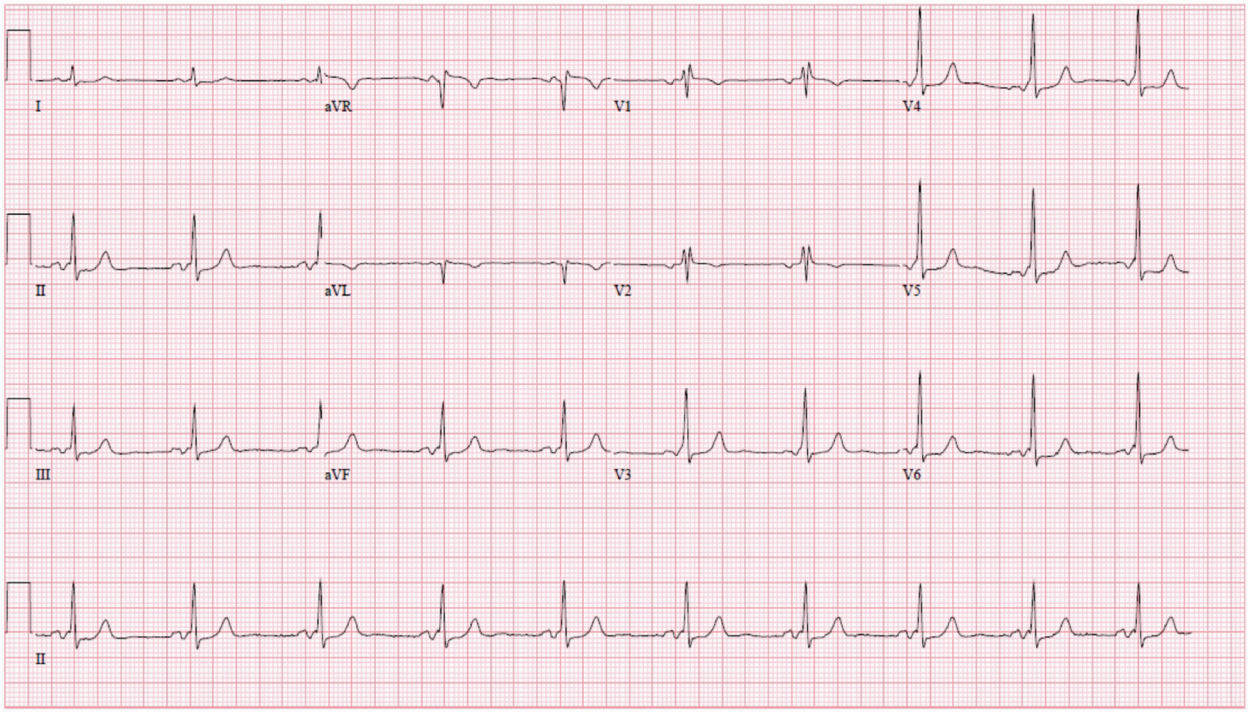
DISCUSSION

ECG findings of a short PR interval (circles) with evidence of delta waves (arrows) and wide QRS is characteristic for WPW pattern.[1] This ECG pattern indicates pre-excitation of the ventricle through a congenital accessory pathway that allows bypass of the conduction system and early depolarization of the ventricle as evident by delta waves. During sinus rhythm, the electrical impulse will travel through the AV node as well as through the accessory bypass tract and simultaneously depolarize the ventricle.[2] Due to the accessory pathway, there is possibility of a re-entrant circuit which can lead to a tachyarrhythmia. This reentrant circuit that involves the atria and ventricle is often abbreviated as AVRT. The term "WPW syndrome" is reserved for when a patient has ECG features consistent with WPW pattern, as described above, as well as documented episodes of tachyarrhythmias or clinical symptoms such as palpitations, pre-syncope, syncope, lightheadedness, or cardiac arrest. Syncope can occur if the tachyarrhythmia becomes too fast leading to poor ventricular filling and thus low cardiac output.

The general outpatient management for suspicion of WPW syndrome would be early outpatient cardiology consultation, transthoracic echocardiogram, exercise treadmill testing and obtaining a holter/event monitor. Primary care providers can streamline the process by ordering both the transthoracic echocardiogram and holter/event monitor while waiting for the cardiology outpatient evaluation. These tests assist the cardiologist to determine whether the patient has a high- risk or low- risk accessory pathway for sudden cardiac death.

In patients who are incidentally noted to have "WPW pattern" on routine ECG, it is very important to take a detailed history to determine whether the patient is symptomatic (palpitations, lightheadedness, pre-syncope, syncope).[3] For symptomatic patients with a WPW pattern, exercise treadmill stress testing

Figure 1. 12-Lead electrocardiogram.



assists in risk stratification of high or low- risk accessory pathways. An abrupt loss of the delta wave during exertion identifies a low-risk pathway as with elevated heart rates the impulse travels preferentially down the native conduction pathway rather than the bypass tract.

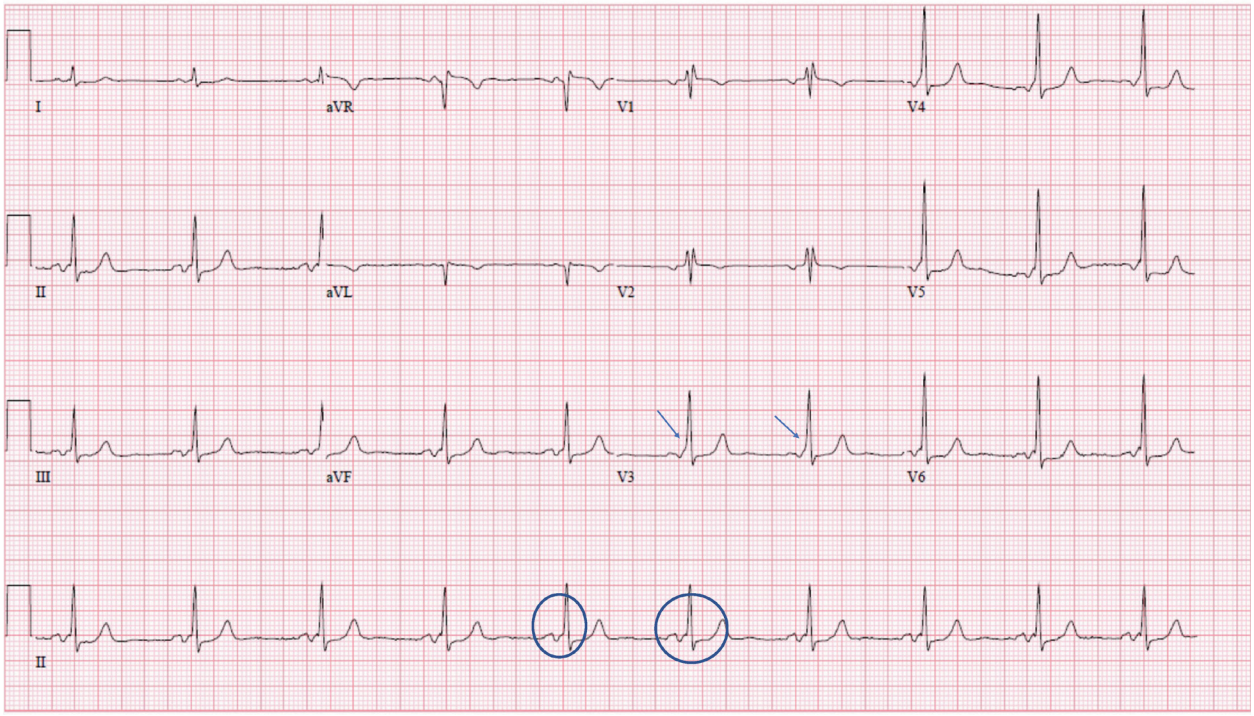
In general, AV nodal agents should be avoided in patients with pre-excitation and features of high-risk pathway or history of atrial fibrillation. AV nodal blocking agents can allow the electrical impulse to travel preferentially down the accessory bypass tract and could lead to ventricular arrhythmias. After testing, the cardiologist can determine the risk of initiating or continuing AV nodal blocking agents in low- risk patients. Electrophysiology studies can also help stratify the pathway and catheter ablation is class 1 indication for recurrent symptomatic WPW syndrome.[2]

One entity to be particularly vigilant for in the emergency setting would be pre-excitation (presence of delta waves) atrial fibrillation and rapid ventricular response. The treatment strategy in this case would be to use procainamide if hemodynamically stable or synchronized cardioversion if unstable. The use of any other medications, such as AV nodal blocking agents adenosine and even amiodarone, can potentially cause the bypass tract to conduct fibrillation rate in a 1:1 ration potentially leading to ventricular fibrillation.

CLINICAL COURSE

The patient was diagnosed with WPW syndrome given WPW pattern on ECG with symptoms of palpitations that was suspected to be due to paroxysmal tachyarrhythmia. Transthoracic echocardiogram demonstrated left ventricular function of 73% without significant valvular abnormality. On review of prior ECGs, there was abrupt loss of delta waves which suggests low risk pathway. Holter monitor did not demonstrate any supraventricular tachycardia. She was referred to electrophysiology with plans for monitoring symptoms and limiting albuterol use. If she continues to have recurrent symptoms, then she would be scheduled for catheter ablation.

Figure 2. 12-Lead electrocardiogram



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