CLINICAL IMAGE

Palpitations in a Young, Healthy Female

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A 26-year-old African American female presented to her family medicine office with a recent pre-syncopal event that occurred early that day. Symptoms included intermittent dizziness, lightheadedness, and palpitations of a two-hour duration. She otherwise had a negative review of systems.

During the previous two months, the patient had experienced similar but less severe episodes of dizziness, lightheadedness and palpitations, which self-resolved within thirty minutes. The patient denied having an increased caffeine or other stimulant intake, and denied any alleviating factors.

An office electrocardiogram *(Figure 1)* was performed. Based upon the patient's symptoms of near syncope in conjunction with an abnormal ECG demonstrating a shortened PR interval, less than 0.12 seconds, and an associated slurred upstroke of the QRS complex, known as a delta wave, the patient was sent for an immediate assessment in the emergency department.

Upon presentation to the emergency department, the patient was found to be afebrile with a blood pressure of 112/72, heart rate regular at 102 beats per minute, and a respiratory rate of 22 breaths per minute with a pulse oximetry on room air at 98%. Physical examination revealed a patient in mild distress, but otherwise awake, alert, oriented, well developed, and well nourished. She was normocephalic and atraumatic with moist mucous membranes and normal tympanic membranes bilaterally. Pupils were equal, round and reactive, extraocular eye movements intact, no nystagmus, and no proptosis. No palpable neck masses were noted. Heart rate was regular and no murmurs, rubs, or gallops were appreciated. Lungs were clear to auscultation without wheezing, rales, or rhonchi. Abdomen was soft, non-tender, and without distention. Distal pulses were strong and symmetric bilaterally without peripheral edema. No exanthems, petechiae, or ecchymosis was noted.

In addition to the office ECG that accompanied the patient upon presentation to the emergency department, two department ECG's were performed *(Figures 2 and 3)*. Significant department diagnostic results included a negative urine pregnancy test, and unremarkable complete blood count, complete metabolic panel and thyroid stimulating hormone, and troponins. Her chest radiograph was unremarkable.

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FIGURE 1:

Office electrocardiogram

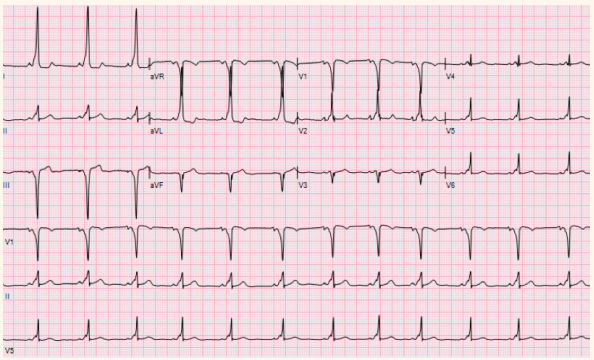


FIGURE 2:

ER Department ECG 1

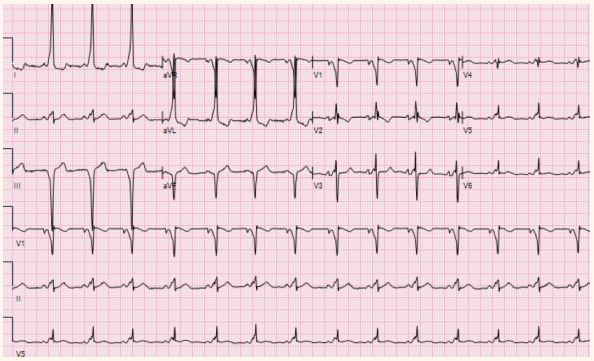
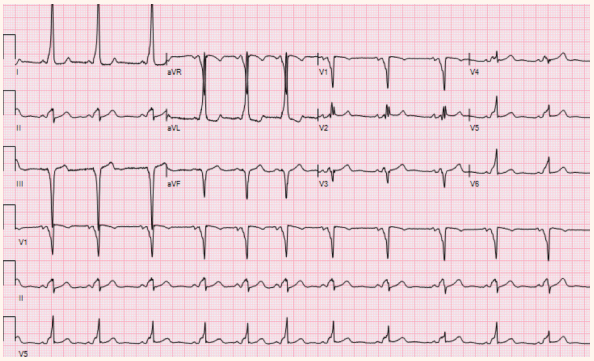


FIGURE 2:

ER Department ECG 2



QUESTIONS:

1. **What is the diagnosis based upon the electrocardiogram (ECG) findings?**

   A. Brugada syndrome
   B. Left bundle branch block
   C. Wolff-Parkinson-White pattern
   D. Myocardial infarction
   E. First degree atrioventricular block

2. **What is the most serious complication of this condition?**

   A. Sudden cardiac death
   B. Heart failure
   C. Syncope
   D. Cardiomyopathy
   E. Palpitations

3. **What is the preferred long-term management of this condition to reduce frequency and intensity of symptoms and decrease mortality?**

   A. Oral doses of verapamil
   B. Oral doses of amiodarone
   C. Mitral valve replacement
   D. Catheter ablation
   E. Digoxin

ANSWERS:

1. **What is the diagnosis based on ECG findings?**

   Correct Answer:
   C. Wolff-Parkinson-White Pattern

Wolff-Parkinson White (WPW) pattern is ventricular pre-excitation with characteristic ECG findings including a short PR interval, less than 0.12 seconds, and slurring of the QRS upstroke, known as a delta wave, which results in a widened QRS complex, lasting greater than 0.10 seconds.[1,2,3]

Incorrect answers:

Brugada syndrome has a characteristic ECG finding of coved ST elevation in leads V1-V3 followed by a negative T wave and is caused by sodium channelopathy.[4]

Left bundle branch block has a characteristic ECG with widened QRS, greater than 120ms, tall broad or notched "M-shaped" R waves in lateral leads (I, V5-6) and deep S waves in right precordial leads (V1-3).[5]

First degree AV block is defined as prolonged PR interval on ECG greater than 200ms.[6]

2. **What is the most serious complication of this condition?**

   Correct Answer:
   A. Sudden cardiac death (SCD)

For an individual with WPW syndrome, there is an estimated annual 0.25% per year risk of SCD, or about a 4% lifetime risk of SCD.[7] SCD in WPW syndrome occurs in most cases because of the rapid ventricular response due to conduction from the AP during atrial fibrillation that deteriorates into ventricular fibrillation.[2,3]

Incorrect answers:

Palpitations and syncope are symptoms associated with WPW, not complications. Cardiomyopathy is not a complication of WPW syndrome. Heart failure is an infrequent complication of WPW syndrome that is not as serious as sudden cardiac death.[2,3]

3. **What is the preferred long-term management of this condition to reduce frequency and intensity of symptoms and decrease mortality?**

   Correct Answer:
   D. Catheter ablation

In patients with WPW syndrome, non-pharmacologic therapy, namely catheter ablation of the accessory conduction pathway, is the current first-line therapy to decrease frequency and severity of symptoms and prevent SCD.[1,2,3,8]

Incorrect answers:

Oral verapamil, amiodarone, and digoxin are contraindicated in patients who have atrial fibrillation (AF) as these AV nodal blocking agents could enhance conduction over the AP and cause increased ventricular contraction, thus increasing the risk to the patient for developing ventricular fibrillation or SCD.[2]

HOSPITAL COURSE OF PATIENT

The 26-year-old female was admitted to the hospital for her symptoms and concerning changes on ECG. The patient was evaluated by cardiology and had successful treatment of her Wolff-Parkinson White (WPW) syndrome with catheter ablation by an electrophysiologist. The ablation was successful in reducing her symptoms of intermittent dizziness, lightheadedness, and palpitations. Key components of the successful outcome included appropriate ECG screening and interpretation by her family physician, appropriate emergent referral to the emergency department, and expedited evaluation by cardiology, resulting in ultimate management via catheter ablation by an electrophysiologist.

DISCUSSION

The patient described above presented to her primary care physician's office with a relatively common chief complaint of pre-syncopal symptoms including dizziness, lightheadedness, and palpitations. These symptoms are frequently brought to the family physician's attention for evaluation by patients of all

ages, gender, race, and with various medical comorbidities. The differential diagnosis for pre-syncope and syncopal symptoms is broad, and includes reflex mediated, cardiac causes, orthostatic hypotension, neurologic causes, endocrinologic causes, psychiatric disorders, and drug induced.[9,10] Of note, patients found to have a cardiac cause of pre-syncope or syncopal symptoms had a higher annual mortality rate.[9,10] Initial evaluation of patients with similar chief complaint of pre-syncope with lightheadedness, dizziness, and palpitations as the presented young female in this case report should include a thorough history and physical exam and ECG in the family physician office setting. Initial screening ECG can aid in assessing for dysrhythmia as source of symptoms. A differential of dysrhythmias or ECG changes that can cause pre-syncope or syncopal symptoms includes atrial fibrillation, atrial flutter, supraventricular tachycardia, pre-excitation syndromes including Wolff-Parkinson White, atrioventricular block, bifascicular block, sinus pause, Brugada syndrome, prolonged QT, ventricular dysrhythmias, bradyarrhythmias, arrhythmogenic right ventricular dysplasia, myocardial infarction, etc.[1,2,11]

WOLFF-PARKINSON WHITE SYNDROME

Wolff-Parkinson White (WPW) syndrome is the cardiac diagnosis responsible for the pre-syncopal symptoms and ECG changes noted of the patient presented in this case report. WPW syndrome is defined as the combination of ventricular pre-excitation pattern on ECG in combination with symptoms of tachyarrhythmia.[12] The characteristic ECG findings of short PR interval, less than 0.[12] seconds, and an associated slurred upstroke of the QRS complex, referred to as a delta wave, are known as the WPW pattern and can be observed in this patient's above ECGs in *Figure 1, 2, and 3*.[1,12,13] This distinct WPW pattern with a delta wave noted on ECG was first identified and documented by Drs. Louis Wolff, John Parkinson, and Paul White in 1930 in a group of patients with the common symptom of intermittent episodes of palpitations or pre-syncope.[13] The prevalence of this distinct WPW pattern is found on ECG in about 0.1-0.3% of the general population.[12,14,15]

WPW PATHOPHYSIOLOGY

The WPW pattern on ECG is the result of conduction via an accessory pathway (AP), known as the Bundle of Kent. This bundle arises from the abnormal differentiation of myocardial tissue during embryonic development.[16] This congenital AP tract forms abnormal conductive cardiac tissue between the atria and ventricles, thus providing an alternative tract for conduction, leading to early ventricular depolarization.[1,16]

In contrast to the AV node, the AP has rapid anterograde and retrograde conduction without rate limitation.[1] This rapid, bidirectional conduction of the electrical stimulus between the atria and ventricles causes ventricular depolarization immediately after atrial depolarization. The result is a pre-excitation pathway that contributes to reentrant tachycardia.[1] This more rapid electrical stimulus conduction over the AP is the reason for the classic ECG findings of the WPW pattern consisting of a short PR interval (less than 0.12 seconds) and slurring of the QRS upstroke (known as a delta wave), and a widened QRS complex lasting more than 0.10 seconds.[1,2,3]

Accessory pathway (AP) and WPW pattern are typically found at random in young healthy patients without structural cardiac abnormalities. However, there have been infrequent case reports of familial WPW and WPW associated with Ebstein's anomaly, as well as structural changes from myocardial ischemia.[17,18]

WPW CLINICAL PRESENTATION

About 60% of patients with WPW pattern on ECG experience symptoms, with the remaining 40% being asymptomatic.[3] Symptoms associated with WPW syndrome include palpitations, episodic lightheadedness or dizziness, pre-syncope, syncope, and, rarely, sudden cardiac death (SCD).[2,3] While SCD can infrequently be the presenting symptom, especially in children or young adults, the overall incidence of SCD in patients with WPW is estimated to be about 0.15-0.39%.[3,19]

For an individual with WPW syndrome, there is an estimated annual 0.25% per year risk of SCD, or about a 4% lifetime risk of SCD.[7] SCD in WPW syndrome occurs in most cases secondary to the rapid ventricular response due to conduction from the AP that deteriorates into ventricular fibrillation. High risk factors for SCD include male sex, age less than thirty years, history of atrial fibrillation, family history of WPW, prior syncope, and presence of congenital heart disease, in particular Ebstein's anomaly.[20]

WPW MANAGEMENT

Treatment of WPW is dependent on hemodynamic stability, severity of symptoms, assessed risk for sudden cardiac death, and location of patient presentation.[2] If a patient presents as an outpatient to the non-acute setting of a primary care physician office, is hemodynamically stable, and has intermittent mild symptoms or if the patient is asymptomatic with incidentally discovered WPW pattern on ECG, a history and physical with non-invasive testing are useful for initial risk-stratification of SCD. Supplemental non-invasive testing includes ECG, ambulatory electrocardiography monitoring, echocardiogram, and exercise stress testing. Electrophysiology study should also be considered to risk-stratify for potential arrhythmic events. The loss of conduction over the AP during exercise testing, or sporadic loss of pre-excitation during ambulatory monitoring indicate lower risk of arrhythmias.[2,3] Asymptomatic WPW pattern patients are determined to be at higher risk for SCD if the shortest pre-excited RR interval is less than 250 milliseconds in AF, if the patient has a history of symptomatic tachycardia, if the patient is found to have multiple AP during an electrophysiology study, or if the patient has a history of Ebstein's anomaly.[2,3,12]

In WPW pattern patients deemed to be at low risk for SCD, close monitoring and observation as an outpatient without further treatment is reasonable.[2,8] In patients with chronic WPW syndrome, or frequent cumbersome symptoms, catheter ablation of the accessory pathway is the current first-line approach to decrease frequency and severity of symptoms as well as to prevent SCD.[1,2,3,8] Catheter ablation is indicated in asymptomatic patients both at higher risk for SCD and in low-risk asymptomatic patients with WPW pattern whose employment requires the treatment of pre-excitation, such as pilots.[2,3] As in this case report, referral of the

patient with WPW syndrome who presents to the primary care office to the emergency department can expedite the evaluation and testing and determination of treatment plan.

In the acute setting, such as in the emergency department, if a patient with known WPW pattern presents with a tachyarrhythmia with a pulse but is hemodynamically unstable, cardioversion is indicated.[2] In hemodynamically stable patients with acute tachyarrhythmia such as supraventricular tachycardia (SVT) or atrioventricular reentrant tachycardia (AVRT) caused by the AP in WPW, pharmacologic agents designed to slow ventricular heart rate and cease arrhythmias are commonly used in the acute setting.

Vagal maneuvers such as the Valsalva maneuver or carotid massage and/or intravenous (IV) adenosine are recommended initial treatments. If vagal maneuvers and IV adenosine are unsuccessful, then IV verapamil, diltiazem or beta blockers are indicated in a hemodynamically stable patient with symptomatic tachyarrhythmia.[2] If these pharmacologic treatments fail to control the ventricular heart rate or cease the arrhythmias, synchronized cardioversion is indicated.[2] It should be noted that in patients who have atrial fibrillation (AF) with pre-excitation, IV or oral verapamil, diltiazem, beta-blockers, IV adenosine, IV amiodarone, and IV digoxin are contraindicated. The reason: these AV nodal blocking agents could enhance conduction over the AP and cause increased ventricular contraction, thus increasing the risk to the patient for developing ventricular fibrillation or SCD.[2] Instead, IV ibutilide or IV procainamide are used to restore sinus rhythm in hemodynamically stable patients with pre-excited AF.[2]

Once the tachyarrhythmia of the symptomatic patient is restored to sinus rhythm and stabilized in the acute setting, the ultimate treatment of WPW syndrome is catheter ablation of the accessory pathway.[1,2,3,8]

CONCLUSION

Although rare, Wolff-Parkinson White pattern and WPW syndrome patients can initially present in the primary care setting. Thus, it is important to be aware this differential diagnosis when evaluating patients with a chief complaint of palpitations, lightheadedness, dizziness, pre-syncope, or syncope. It is also crucial that primary care physicians are able to recognize the characteristic ECG findings for WPW pattern consisting of a short PR interval, less than 0.12 seconds, and slurring of the QRS upstroke (delta wave), resulting in a widened QRS complex.

Family physicians can and should assess for these ECG findings, especially since approximately 40% of patients with WPW pattern are asymptomatic.

Further vigilance to these ECG patterns are especially important in young patients with a history of intermittent palpitations or pre-syncope, in patients with a history of Ebstein's anomaly, and in patients with a familial history of WPW. Referral of WPW pattern or WPW syndrome patients by primary care physicians to cardiology/electrophysiology should be prompt for further evaluation and treatment planning.

AUTHOR DISCLOSURES:
No relevant financial affiliations or conflicts of interest.

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