A Rare Cause of Vomiting in a 5-Year-Old Girl

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Abstract

A 5-year-old previously healthy girl presented to the emergency department (ED) with vomiting for 6 days. Her activity level was normal but her parents described progressive exercise intolerance with frequent recreational breaks approximately 2 days prior to presentation. In addition, the child complained of mild abdominal discomfort that began 24 hours prior to her presentation. She had no diarrhea or changes in her diet or oral intake; she had no dysuria or changes in urination. She had no cough, shortness of breath, dizziness, vision changes, falls, or decreased coordination. [Pediatr Ann. 2015;44(3):e62-e66.]
A 5-year-old previously healthy girl presented to the emergency department (ED) with vomiting for 6 days. On arrival in the ED, her blood pressure was 74/53 mm Hg, heart rate was 203 beats per minute (bpm), respiratory rate was 30 breaths per minute, temperature was 97.9.6°F, and oxygen saturation was 98% while breathing room air. On examination, she was pale, lying quietly on the exam table but interactive when spoken to. Her oropharynx was clear and her lips were dry and crusty. Her cardiac examination revealed a quiet precordium, normal point of maximal impulse, tachycardia, single S1 and S2, no murmurs, no S3/S4, and no other abnormal heart sounds. She had clear breath sounds bilaterally with no crackles or wheezes. Her abdomen was soft with mild tenderness at the umbilicus. She had normal bowel sounds, and there was no hepatomegaly, splenomegaly, or masses. Examination of her extremities was remarkable for slightly mottled hands and feet, 1+ distal pulses, and capillary refill time of 3 to 5 seconds. Her skin was pale with a petechial rash at the right forearm below the blood pressure cuff. She had no cyanosis or clubbing.

**DISCUSSION**

The patient was determined to be in shock because of the clinical findings of hypotension, tachycardia, pallor, and delayed capillary refill. However, the etiology or type of shock had yet to be determined. Septic shock, perhaps from a pneumonia or urinary tract infection (which can both present with vomiting in a young child) was considered. Hypovolemic shock was considered, although the patient’s history of vomiting included relatively normal fluid intake and urine output. Cardiogenic shock was also considered. When questioned about the patient’s increased heart rate on intake, her mother acknowledged feeling a rapid heartbeat several days prior but she was uncertain if it had continued. There was no family history of sudden death, irregular heart rhythms, or children with congenital or acquired heart disease.

To treat presumptive hypovolemic shock, the patient was given two intravenous (IV) fluid boluses of 20 mL/kg of normal saline (NS). Her heart rate decreased from over 200 bpm to approximately 180 bpm with variability, and her blood pressure increased to 99/71 mm Hg. The initial heart rate decrease and blood pressure response to fluids suggested a sinus tachycardia in response to hypovolemia. However, after the third IV NS bolus of 20 mL/kg, her heart rate increased to over 200 bpm. She was given an IV dose of antibiotics to empirically treat septic shock, one of the diagnoses on our differential diagnosis. A complete blood count and complete metabolic panel were ordered and returned within normal limits. A chest X-ray (Figure 1) showed a slightly enlarged cardiac silhouette. An abdominal X-ray was normal.

Her electrocardiogram (ECG) showed a narrow complex tachycardia with a P wave preceding every QRS complex (Figure 2). However, the P wave was noted to be upright in lead I but (abnormally) inverted in lead II and aVF. A pediatric cardiologist was consulted and the diagnosis of a long R-P tachycardia, consistent with persistent/paroxysmal junctional reciprocating tachycardia (PJRT), was made.

Further cardiac studies showed a B type-natriuretic peptide of 1996 pg/mL. An echocardiogram done shortly thereafter showed normal cardiac anatomy, but left ventricular function was severely depressed (Figure 3), with a shortening fraction of <10%. The right ventricular function was likewise impaired.

**DIAGNOSIS**

**Supraventricular Tachycardia and Persistent Junctional Reciprocating Tachycardia**

Supraventricular tachycardia (SVT) is the most common tachyarrhythmia in children. It is usually nonsustained. The majority of patients have structurally normal hearts, although patients with congenital heart disease are more likely to have SVT. Gastrointestinal symptoms, especially vomiting, can be a presenting symptom of SVT in children.

It is important for the general pediatrician to understand the unique properties of PJRT and to differentiate this re-entrant SVT from sinus tachycardia. PJRT is an orthodromic, narrow QRS, long RP SVT. The atrial impulses are conducted down the atrioventricular (AV) node, and the ventricular impulses are conducted up an accessory pathway near the AV node. The conduction properties of the ac-

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**Figure 1.** Patient’s chest X-ray showing an enlarged cardiac silhouette.

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**Figure 2.** ECG showing a narrow complex tachycardia with a P wave preceding every QRS complex.

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**Figure 3.** Echocardiogram showing normal cardiac anatomy with severely depressed left ventricular function.
CME

Accessory pathway are what make PJRT a unique form of SVT. The accessory pathway exhibits the electrophysiological property of decremental conduction. In decremental conduction, transmission of electrical pulses slows with increasingly premature stimuli. The faster the pathway is stimulated, the slower it actually conducts. This property explains the variability of the heart rate compared to typical reentrant SVT. It also explains the long RP interval of PJRT (Figure 4). Typical SVT is referred to as a short RP tachycardia because the P wave is immediately after the QRS complex. In typical SVT, the impulse is conducted rapidly from the ventricle to the atria. Therefore, the surface ECG shows a P wave either buried in the QRS or just after the QRS complex. However in PJRT, because of the relative slowness of the decrementally conducting fibers, the depolarization of the atria is delayed and the atria are depolarized closer to the following QRS complex. This pattern can be confusing because atrial depolarization appears to be preceding ventricular conduction as it does in normal conduction. It is important to note that the inverted P wave in SVT, whether the RP is short or long, is associated with the previous QRS and represents the retrograde depolarization of the atria via the accessory pathway (hence, the P waves were inverted in leads II and aVF in our patient, but would normally but upright in these leads). Additionally, the slowly conducting decremental pathway makes this a relatively stable tachycardia that can be sustained and lead to tachycardia-induced cardiomyopathy. 

PJRT frequently presents with tachycardia-induced cardiomyopathy. This form of cardiomyopathy most closely resembles dilated cardiomyopathy (DCM), whose cardinal feature is low cardiac output leading to cardiogenic shock. However, because DCM can itself cause tachyarrhythmias, it can sometimes be difficult to distinguish cause from effect.

This case illustrates the variability in presentation in children with dilated cardiomyopathy and even severe left ventricular (LV) dysfunction. Typically, infants and small children with poor LV function will present with poor feeding, vomiting, fatigue, irritability, and respiratory distress. Older children and adolescents will typically present with exercise intolerance, chest pain, anorexia, or abdominal pain. Examination findings can include lower extremity edema, jugular venous distention, pulmonary congestion, murmurs (tricuspid or mitral valve insufficiency), gallops, or hepatomegaly.

The treatment for tachycardia-induced cardiomyopathy is to terminate the offending rhythm (or at least slow the rate) and support the patient until there is recovery of cardiac function. Because of the difficulty in controlling the rhythm of PJRT, most centers initially focus on rate control. Recovery of function depends on time in the tachyarrhythmia, degree of tachycardia, and age of presentation. Partial recovery can be noted within days of treatment, but full recovery may take weeks. In a study by Vaksmann et al., congestive heart failure resolved after successful PJRT treatment in all children. This same study found that it was challenging to convert PJRT to sinus rhythm or to control the rate of the tachyarrhythmia. Some patients were prescribed up to 9 different antiarrhythmic drugs to attain rhythm and/
or rate control.\textsuperscript{1} With the challenge of attaining lasting rhythm conversion on antiarrhythmic drugs, definitive therapy is often sought with radiofrequency catheter ablation of the accessory pathway. Although more curative perhaps than antiarrhythmic therapy, catheter ablation has obstacles as well.

In young children, the small size of the patient and the heart itself can make the procedure more difficult. Other risks of ablation are anesthesia/sedation, the need for re-ablation if the initial procedure is unsuccessful, and inadvertent atrioventricular conduction block secondary to the ablation.\textsuperscript{7}

A key point is that when evaluating and treating a patient, in this case with suspected shock, it is important to continue to re-evaluate after giving treatments, such as an NS bolus. Furthermore, it is important to reconsider the diagnosis when a patient does not respond as expected to the given treatment. It is also important to remember that hypotension is a late finding of shock in children. Because of their faster resting heart rate, children need a larger baseline increase in their systemic vasoconstriction than would be seen in adults with various forms of shock. Therefore, the finding of tachycardia and hypotension in a child is a potentially ominous sign of impending cardiovascular collapse.\textsuperscript{8}

As mentioned earlier, it is instructive to differentiate PJRT from sinus tachycardia. The ECG of PJRT differs in appearance from the more typical causes of SVT. Sinus rhythm (Figure 5) and by extension sinus tachycardia, is defined as upright P waves preceding a QRS complex in leads I, II, and aVF. In typical re-entrant SVT, whether through the atrioventricular node or an accessory pathway, the P wave morphology, if visible, will always be different than the usual sinus morphology.\textsuperscript{7} Furthermore, sinus tachycardia will usually exhibit some degree of variability in the heart rate, whereas in typical SVT it is likely to be a fixed rate. The rate can sometimes be helpful to take into account as well. A rate of more than 180 bpm may suggest an increased likelihood of SVT; however, it is important to note that children with heart rates of more than 180 bpm are still more likely to be in sinus tachycardia than SVT. Response to fluids, fever reducers, pain relievers, vagal stimuli, and adenosine can also help to differentiate sinus tachycardia from SVT, with sinus tachycardia usually responding to the latter two interventions.

Another interesting point is the differential diagnosis of vomiting alone. Without diarrhea, it may not represent simple gastroenteritis. Other diagnoses to think of, although less common, include gastrointestinal obstruction, increased intracranial pressure due to bleeding or mass, pneumonia, urinary tract infection, and, as in this case, SVT or heart failure. In most cases, not all of these possibilities will be high on the differential diagnosis, but they are important to consider in assessing the patient’s whole clinical picture.

**CONCLUSION**

This case illustrates a patient with a common presenting symptom and an uncommon diagnosis. Her initial presentation suggested hypovolemia, but as evaluation continued, this diagnosis could not account for all of her clinical features. The diagnosis of PJRT,
a rare but well-documented form of SVT, was made based on specific ECG criteria in addition to a consistent history, physical examination, and initial ED treatment course.

REFERENCES