

Case Presentation

- A 15 year-old female with iron deficiency anemia, vitamin D deficiency, and dysfunctional uterine bleeding presented to the ED for acute onset palpitations while swimming. Associated symptoms include chest pain, shortness of breath, dizziness, headache, and nausea.
- Heart rate was in the 220s on a smart watch. She also felt as if she passed out for a couple of minutes (unwitnessed).
- On arrival to the ED heart rate was 220 bpm. All other vital signs were within normal limits.
- Patient reported similar episodes of palpitations in the past, most recently after having an energy drink. Prior thyroid workup was negative and her symptoms were previously attributed to anxiety/panic attacks.
- Family history was unremarkable.
- Initial cardiac exam was benign with regular heart sounds, no murmurs, clicks, rubs or gallops.
- Troponin and BNP were elevated at 1.4 ng/mL and 134.2 pg/mL, respectively.
- EKG showed a wide complex tachycardia with a left bundle branch block and a superiorly orientated QRS complex [Fig.1].
- She was hemodynamically stable throughout the episode and spontaneously converted to sinus rhythm without evidence of ventricular pre-excitation [Fig. 2].

Evaluation/Management

- She was admitted to the PICU for continued observation and initiation of atenolol 25 mg twice daily.
- Patient remained in sinus rhythm with stable vitals on atenolol. Repeat EKG during admission demonstrated normal sinus rhythm.
- She was discharged home on hospital day 3 with a 30-day event monitor.
- Patient was referred to the pediatric electrophysiology service.
- An electrophysiology study was performed and identified a concealed right-sided accessory pathway [Image 1] and a typical atrioventricular nodal reentry tachycardia (AVNRT) [Image 2].
- She underwent successful radiofrequency (RF) ablation of the concealed right-sided accessory pathway and cryoablation of typical AVNRT.
- She has continued to do well clinically without documented recurrence of her SVT.

Figures and Images

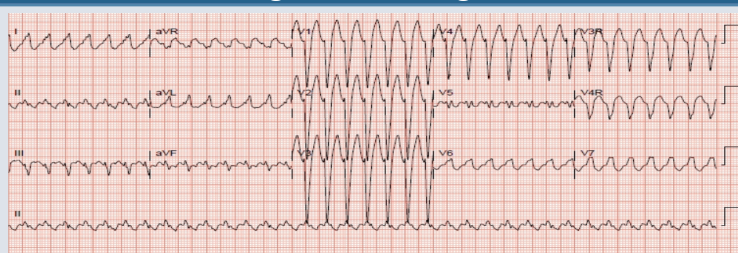


Figure 1. Electrocardiogram on presentation in ED.

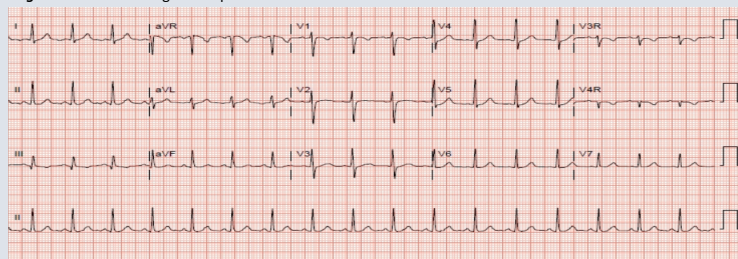


Figure 2. Electrocardiogram after spontaneous conversion to sinus rhythm.

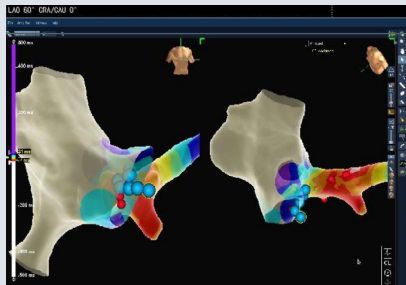


Image 1. Activation patterns with V-pacing to help localize the accessory pathway. Blue dots were used for cryoablation. Red dots were used for RF ablation.



Image 2. Electrophysiology lab intracardiac electrogram showing typical AVNRT.

Discussion

- Differential diagnosis of wide complex tachycardia commonly ranges from genetic causes such as long QT syndrome, Brugada syndrome, and cardiomyopathies to acquired conditions such as infectious myocarditis.
- In the adolescent population, ingestion of stimulants such as amphetamines, cocaine, excess caffeine, and nicotine can also be potential contributors.
- Supraventricular tachycardia can also present with a wide QRS complex due to an aberrant accessory pathway.
- Palpitations are often the presenting symptom of atrioventricular reentrant tachycardia and can often be mistaken for anxiety or panic attacks.
- While conditions such as Wolff-Parkinson-White syndrome are known to have predisposition to ventricular fibrillation, other etiologies of SVT, such as AVNRT, EAT, and concealed accessory pathway with AVRT have a lower risk to develop ventricular tachyarrhythmia.
- Clinical suspicion for underlying cardiac etiology should be high in patients presenting with palpitations, especially associated with exertion or suspected syncope.
- Prompt recognition, expert consultation, and medical treatment are a cornerstone to management of SVT. Invasive electrophysiology study with curative ablation of SVT substrate offers a potentially permanent solution.

References

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