Supraventricular Tachycardia in the Pediatric Trauma Patient: A Case Report

abstract

Any injured patient who is cool and tachycardic is considered to be in shock until proven otherwise.1 We describe the diagnostic challenge when evaluating persistent tachycardia in the setting of multiple system trauma with hemorrhagic shock. This is a unique case of a 17-year-old patient with the secondary condition of cardiogenic shock due to supraventricular tachycardia (SVT) complicating ongoing hemorrhagic shock from a facial laceration. She had sustained tachycardia despite aggressive resuscitation and required medical cardioversion 30 minutes after arrival to the emergency department. After successful conversion, she maintained normal sinus rhythm for the rest of her hospitalization. During her follow-up cardiac catheterization, she was found to have a left-sided accessory pathway, consistent with atrioventricular reciprocating tachycardia. This is a unique and rare case of SVT in the traumatic patient. We review causes of tachycardia in the setting of pediatric multisystem trauma, as well as discuss acute SVT evaluation and management in the pediatric emergency department. Pediatrics 2013;131:e1654–e1658

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ABBREVIATIONS
AI—atrioventricular
AVRT—atrioventricular reciprocating tachycardia
BP—blood pressure
ECG—electrocardiogram
ECHO—echocardiogram
ED—emergency department
EP—electrophysiology
IV—intravenous
SVT—supraventricular tachycardia
WPW—Wolff Parkinson White

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Any injured patient who is cool and tachycardic is considered to be in shock until proven otherwise. We describe the diagnostic challenge when evaluating persistent tachycardia in the setting of multiple system trauma with hemorrhagic shock. This is a unique case given the secondary condition of cardiogenic shock due to supraventricular tachycardia (SVT) complicating ongoing hemorrhagic shock.

CASE PRESENTATION

A 17-year-old female patient presented to the pediatric emergency department (ED) with a complicated facial laceration after a multiple system trauma. She was driving restrained in a vehicle without an airbag when she lost control and hit a fence. She lost consciousness for an unknown duration. Upon emergency medical services arrival, she was noted to have a complicated laceration to her nose and partial de-gloving of her forehead with significant ongoing hemorrhage, but she had no other obvious injuries and a Glasgow Coma Scale of 15. Her vitals remained stable en route to the ED with a heart rate of 98 to 110 beats per minute and a blood pressure (BP) of 117 to 120/74 to 78 mm Hg.

Upon arrival she was placed on a monitor, and her initial vital signs in the ED were as follows: heart rate, 219 beats per minute; respiratory rate, 30; BP, 114/51 mm Hg; and oxygen saturation, 100% on room air. She was alert and complaining of pain at her forehead laceration. Her primary survey consisted of a patent and stable airway with c-spine precautions, equal chest rise with clear breath sounds bilaterally, no chest wall tenderness or crepitus, clear heart sounds without murmur, regular rhythm, tachycardic with 1+ distal pulses and 2+ central pulses, active bleeding from her complex facial laceration, Glasgow Coma Scale of 15, and no other wounds or contusions when exposed. Bedside focused abdominal sonogram for trauma examination results were negative. Sonographic views of the heart revealed no effusion. Two normal saline boluses (2 L) and fentanyl 1.5 mcg/kg were infused intravenously. Her secondary survey consisted of an actively bleeding 12 cm forehead laceration with partial scalp de-gloving and nasal laceration with separation of her right nare. The remainder of the survey was negative, with normal chest and pelvis radiographs.

Her heart rate remained between 210 and 220 beats per minute with BP of 114 to 116/51 to 68 mm Hg with good perfusion and mentation during this time of initial assessment. Acute blood loss was considered to be the most likely cause of the continued tachycardia, and rapid transfusion protocol was initiated after the 2 normal saline boluses. Before this, initial arrival bedside hemoglobin/hematocrit was 11.9/35.

In the mean time, further assessment of her facial wound with an attempt at controlling the hemorrhage was performed by the surgery service. Ongoing evaluation of her sustained tachycardia continued. Her medical history included a history of glycogen storage disease type 1 status post liver transplant 4 years ago with a normal echocardiogram (ECHO) and electrocardiogram (ECG) at pretransplant evaluation that same year. She was taking tacrolimus 4 mg twice a day. Hemorrhagic shock remained the most likely etiology of the tachycardia; however, other than the facial laceration, there were no signs of any internal bleeding, and bedside hemoglobin was only mildly decreased. The heart rate of 220 beats per minute was out of proportion to her hemorrhage, and there was no improvement after aggressive volume resuscitation or pain control after 20 minutes. It was difficult to discern discrete p-waves on the telemetry monitor during the duration of her initial evaluation, which was concerning for SVT. The telemetry rhythm printout revealed absent p waves, with narrow complex QRS at a rate of 237 (Fig 1). A 12-lead ECG was obtained, which revealed no beat to beat variability. Cardiology provided a bedside consult to assist in confirming the diagnosis of SVT.

Thirty minutes after arrival (shortly after cardiology arrival and SVT diagnosis), her heart rate continued to be sustained at 220 beats per minute, and her BP fell to 88/52 mm Hg, then 65/34 mm Hg. At this time, she quickly became pale and diaphoretic, while still mentating well. Adenosine was rapidly pushed at an intravenous (IV) dose of 0.1 mg/kg. On the ECG, the SVT was terminated with a pause, followed by ventricular escape beats, then by resumption of a normal sinus rhythm at a rate of 120 beats per minute (Fig 2). She immediately felt better, with marked improvement of her skin color and perfusion, and her BP improved to 120/60 mm Hg, which was maintained throughout the rest of her ED course. A repeat hemoglobin/hematocrit revealed a drop to 6.5/19 after a total of 2 L of normal saline and 3 U of packed red blood cells.

There was no ventricular preexcitation on her resting ECG, providing evidence against Wolff Parkinson White (WPW) syndrome. Hepatology was consulted after her heart rate stabilized during the ED course, confirming the presence of normal liver function and the history of a normal pretransplant cardiac evaluation. They also informed that the tacrolimus should not play a causative role in the SVT, nor should her liver function be a concern regarding metabolism of cardiac medications in general. After repair of her wound and inpatient telemetry observation, cardiology did not recommend any further inpatient work-up for the SVT but did arrange outpatient follow-up since it was triggered by a less than routine event. Her status post liver transplant.
did not contribute to being more susceptible to arrhythmias or cardiac medications because she had a healthy heart before and after liver transplant surgery. No cardiac enzymes or imaging was obtained. The patient was taken to the electrophysiology (EP) laboratory as an outpatient to further delineate the mechanism of her SVT. During her EP study 1 month later, she was found to have a left-sided accessory pathway, consistent with atrioventricular reciprocating tachycardia (AVRT) and was successfully ablated.

DISCUSSION

We present a unique case of pediatric trauma with a diagnostic challenge given the myriad causes of hemorrhagic and cardiogenic shock presenting as tachycardia. There is 1 previous case report of an 8-year-old patient developing SVT after multisystem trauma of motor vehicle collision versus bicycle; however, the runs of SVT did not arise until several hours after arrival, were short with a heart rate 130 to 160 beats per minute, and always terminated spontaneously or with carotid massage. This child had no signs of chest trauma on examination or imaging, similar to our patient. In regards to our case, she had a full cardiac evaluation before her liver transplant (4 years before this event). In a review of her past medical history, type I glycogen storage disease is a deficiency in glucose-6-phosphatase that has no known cardiac effects.

Hemorrhage is the most common cause of shock after trauma, and virtually all patients with multiple injuries have an element of hypovolemia from bleeding. Early manifestations of shock include tachycardia and peripheral vasoconstriction. A narrow pulse pressure suggests significant blood loss and involvement of compensatory mechanisms. However, a common pitfall is assuming that there is only one cause for shock in the injured patient. Tachycardia from hemorrhagic shock must be differentiated from other causes of tachycardia. The differential diagnosis is wide, with hemorrhage being the most common, but pneumothorax, cardiac injury with structural damage or tamponade, dysrhythmia, and ingestion should be considered. Pain and anxiety also contribute, as well as other nontraumatic causes. Obtaining a chest radiograph, focused abdominal sonogram for trauma to visualize the heart structure on bedside ultrasound, as well as a 12-lead ECG are valuable tools to help delineate the cause quickly.

Before knowledge from the EP study, the most likely trigger of our patient’s SVT was thought to be either blunt chest trauma causing a myocardial injury or contusion with associated arrhythmia, or simply elevated adrenergic tone from stress. Blunt cardiac trauma is more common in children with multiple organ system injuries, with cardiac contusion being the most common final diagnosis. However, this is difficult to diagnose with no defined criteria. A retrospective review from 1983 to 1993 of pediatric blunt cardiac trauma revealed that 95% had an end diagnosis of myocardial contusion. The most common diagnostic tests being ECG, MB band of creatine phosphokinase, and ECHO. ECG and ECHO revealed fair agreement in diagnosis; however, MB band of creatine phosphokinase did not with either test. A study evaluating cardiac contusions in adults revealed that cardiac troponin I is a helpful diagnostic indicator of cardiac injury, with increased levels of cardiac troponin I correlating with increased risk of arrhythmia. Children were not included in this study. However, trauma literature, as well as these studies, support that these laboratory tests rarely assist in diagnosis or treatment of cardiogenic shock in the ED.

In a multiple injured patient who presents with hypotension caused by presumed blood loss, the patient may also have had secondary cardiac injury resulting from poor myocardial perfusion. Our patient did not have cardiac enzymes obtained; however, there are no published normal values in the face of pediatric trauma, so it is difficult to know what utility these serve and how to interpret. It is more likely that increased adrenergic tone from overall stress of the accident or transmitted force of the impact and possible cardiac contusion triggering either premature atrial or ventricular contractions (with an underlying accessory pathway,
which we will mention in detail) that caused our patient’s SVT rather than decreased myocardial perfusion due to blood loss since her BP remained stable for the majority of her presentation and recovered after cardioversion.

Arrhythmias are not common in children,7 regardless of traumatic injury, and presenting to the pediatric ED with first time arrhythmia is also not common. Suspicion for a tachyarrhythmia should remain high when the rate is sustained regardless of interventions such as IV fluids for hydration, fever control, pain control, and calming techniques for anxiety, and evaluation for any structural injury (such as pneumothorax or myocardial injury) with bedside imaging. Patients should be on telemetry monitoring while treatment ensues. SVT is the most common malignant pediatric tachycardia.8 The category of SVT can be divided into 3 major subcategories: reentrant tachycardias using an accessory pathway, reentrant tachycardias without an accessory pathway, and ectopic or automatic tachycardias.9 SVT refers to all tachycardia originating above the ventricles; however, this discussion will refer to reentrant types of tachycardia that are specifically dependent on the atrioventricular (AV) node.10

Classically, SVT has an unvarying heart rate and therefore a fixed cycle length, usually with a narrow complex QRS (it can be wide complex if aberrant conduction, possessing a similar configuration as ventricular tachycardia) with the absence of clearly discernible p waves. The majority of SVT in children is caused by either AV nodal or AV reentry. These reentrant forms of SVT can have abrupt onset and cessation with the only complaint being patient awareness of a rapid heart rate. AVRT, our patient’s ultimate diagnosis, involves an accessory pathway and is the most common mechanism of SVT in infants and children. The most common example of this is WPW syndrome with an accessory pathway that conducts both antegrade and retrograde. However, AVRT can be supported by an accessory pathway, which only conducts in the retrograde direction, a so-called “concealed” accessory pathway due to the absent footprint on a resting ECG. Characteristic ECG findings in WPW include a shortened PR interval and slurred onset of the QRS complex known as the δ wave (Fig 3). This can be found with structurally normal hearts, as well as in hypertrophic cardiomyopathy or Ebstein anomaly. In patients with WPW, syncope is a potential warning sign of rapid ventricular conduction and atrial fibrillation. Atrioventricular node reentry tachycardia is more common in older children and adolescents.9 In this form of SVT, reentry is entirely conducted within the AV node.

An intervention that interrupts the reentrant circuit in will interrupt the tachycardia for both AVRT and atrioventricular node reentry tachycardia.8 Treatment choice of SVT depends on the clinical stability of the patient. A confirmatory 12-lead ECG should be obtained before treatment. Vagal stimulation is the first-line treatment. If unsuccessful or not applicable (as with our trauma patient), the pharmacologic agent of choice for the rapid conversion is adenosine, breaking the reentry circuit by slowing or blocking conduction in the AV node and will terminate the SVT in 72% to 77% of cases.11,12

In the evaluation of sustained tachycardia occurring with blunt trauma management and ongoing hemorrhage, there has to be a high level of clinical suspicion for arrhythmia. It is best to use a step-wise approach to evaluate sustained tachycardia with blood loss being the most common likely etiology. This explains the clinical rationale for our incremental approach as the patient continued to show adequate perfusion and acceptable BP and mentation. In accordance with advanced trauma life support (ATLS) guidelines for the trauma patient, resuscitation of ongoing hemorrhage with rapid IV crystalloid infusion in the first 10 to 15 minutes (our patient receiving 2 boluses of normal saline during this short time), then the initiation of the rapid transfusion protocol to compensate for the ongoing bleeding (moving to colloid administration when no response is seen with crystalloid per ATLS) was performed. While giving this therapy time to work, evaluation for any cardiac tamponade

**FIGURE 3**
ECG revealing WPW.
on ultrasound as well as pneumo/hemothorax and treating the pain/anxiety component were also completed. This left the more rare entity of a concurrent arrhythmia. As this patient remained well perfused with a stable BP, there was enough time to consult cardiology. Adenosine was drawn up in the meantime, and the defibrillation/cardioversion equipment was appropriately set up and attached to the patient. As seen in the final hemoglobin, ongoing blood loss was also a contributing factor to her problem list, adding to the complexity of the clinical situation and acuity.

In terms of the administration of adenosine for SVT, continuous ECG should be performed to confirm the effects on the rhythm. Adenosine can uncover other arrhythmias such as primary atrial tachycardia or flutter. It is best to have cardioversion equipment available in case of acute decompensation to a nonperfusing rhythm or external pacing equipment for unexpected sustained bradycardia from prolonged AV block. Adenosine side effects include nausea/vomiting, chest pain, bronchospasm, apnea (in young children typically), arrhythmia (other atrial or ventricular), or long asystolic pause. Adenosine is contraindicated in high grade heart block and should be used with care in WPW with rapid antegrade conduction over the pathway due to the possibility of evolution to atrial or ventricular fibrillation (again, have cardioversion equipment available). Admission and treatment with an esmolol drip is also an option for refractory yet clinically stable SVT. Synchronized cardioversion is reserved for refractory or unstable SVT.

After conversion to sinus rhythm, an ECG should be obtained looking for the presence of WPW syndrome. If WPW is present, β-blockers can be started for prevention. In the absence of WPW, either a β-blocker or digoxin is generally the treatment of choice for chronic preventative therapy. Expectant management guided by pediatric cardiology is reasonable if older aged children, in the absence of cardiac dysfunction or structural heart disease, and after the first episode of SVT. It is important to use all resources to achieve the best outcome for the patient, which cannot be emphasized enough. This is a perfect example of excellent teamwork between the ED staff and the consulting cardiologist, as well as trauma evaluation and ED management in the pediatric area in a complicated and rare case.

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