

Supraventricular tachycardia associated with disseminated intravascular coagulopathy in a neonate in the emergency department setting

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Abstract

A 19-day-old neonate who presented to the emergency department (ED) due to supraventricular tachycardia (SVT) was found to have a coagulation disorder on physical examination. Laboratory work-up was consistent with hepatitis and disseminated intravascular coagulopathy (DIC). The source was later traced to an enteroviral infection. There was no evidence of myocarditis.

The association of SVT with hepatitis and DIC in the neonatal period has not been previously reported. The current work-up recommendations for neonatal SVT in the ED include only the performance of an electrocardiogram and echocardiogram. This case illustrates that a more careful and thorough evaluation may be necessary, as SVT may indicate the presence of a more serious underlying disorder.

MeSH Words: Supraventricular tachycardia, disseminated intravascular coagulation, hepatitis, neonate

Case Report

A 19-day-old female infant was brought to the emergency department (ED) with tachycardia. Maternal history revealed a normal pregnancy and delivery, and both mother and child were apparently healthy in the immediate perinatal period. A week prior to admission, the infant's mother noted a skin eruption on the upper limbs, which later spread to the upper chest. There were no other manifestations of disease. One day prior to admission, the patient became irritable and refused to eat. The next day, she was seen by her pediatrician, who diagnosed tachycardia and referred her to the ED.

There were no symptoms of upper respiratory tract infection, and any diarrhea or fever. On admission, the patient appeared alert, though irritable and tachypneic. Temperature was 36.9°C, heart rate 274 bpm, blood pressure 92/66 mmHg, and respiratory rate was 50/min. Peripheral pulses were good, and capillary refill time was less than 2 sec. The anterior fontanelle was normotensive, and heart sounds were rapid and regular. No murmurs were noted. The lungs were clear. The abdomen was soft, and the liver was palpated 4 cm under costal margin, with no

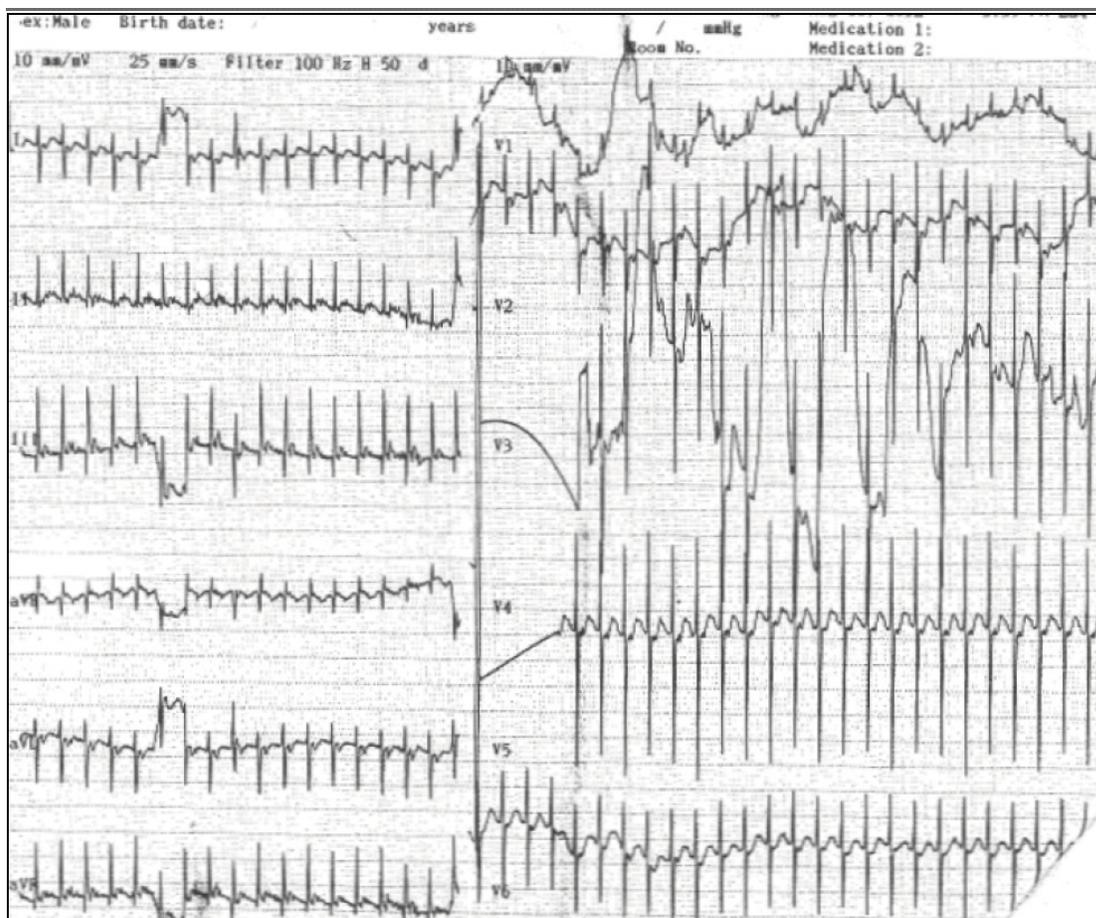


Figure 1: ECG demonstrates SVT

apparent splenomegaly or lymphadenopathy. Muscle tone was normal. A maculopapular rash was apparent on the upper chest and arms, with no purpura or petechiae. The rhythm strip on the monitor was consistent with supraventricular tachycardia (SVT) (Figure 1).

As the baby was hemodynamically stable, various vagal maneuvers were attempted, but they failed to convert the rhythm. Intravenous adenosine 0.1 mg/kg proved ineffective, but a second dose of 0.2 mg/kg, administered a few minutes later successfully converted the rhythm to sinus at 130 bpm. An electrocardiogram performed at that time was normal, aside from a short P-R, with no delta wave.

During the attempts at insertion of the intravenous line, we observed constant bleeding from the puncture sites, which prompted a request for coagulation studies, in addition to a complete blood count and blood chemistry profile. The results are shown in Table 1. The combination of thrombocytopenia and

noncoagulable blood led to a diagnosis of disseminated intravascular coagulopathy (DIC), and the patient was

transferred to the pediatric intensive care unit (PICU) for further treatment and evaluation.

The clinical picture on admission to the PICU was consistent with DIC and hepatitis. Full sepsis work-up, blood serology studies for hepatitis and TORCH viruses, viral cultures and tests for metabolic inborn errors of metabolism were all negative or normal. Interestingly, polymerase chain reaction (PCR) studies were positive for enterovirus in the stool but negative for herpesvirus and enterovirus in the cerebrospinal fluid. Myocarditis was ruled out on further evaluation using electrocardiograms, echocardiograms and measurements of blood creatine phosphokinase (CPK) and troponin levels.

Laboratory variable	On admission	6 hours later	3 days later
Complete blood count			
WBC	9300	7230	6330
Hgb	13.6	12.1	10.5
Platelets	62000	258000	134000
Coagulation tests			
PT % (INR)	NC	21(4.66)	66(1.43)
PTT (sec)	NC	40.7	24.4
Fibrinogen	NC	69	355
Blood chemistry			
Glucose (mg/dl)	78	76	77
Urea (mg/dl)	48	49	12
Creatinine (mg/dl)	0.6	0.6	0.6
Na (meq/L)	132	136	138
K (meq/L)	5.6	4.4	3.7
Albumin (gr/dl)	3.7	3.7	3.4
Bilirubin (T/D) (mg/dl)	9.2/1.1	8	4.2
Alk phosphatase (u/l)	1013	656	403
GOT (AST) (u/l)	490	451	134
GPT (ALT) (u/l)	291	217	141
GGT (u/l)	64	55	35

Table 1: Lab Results: SVT with DIC

Treatment in the PICU consisted of multiple blood products and digoxin, later changed to propranolol, in addition to supportive therapy. The infant was discharged from the hospital on day 9.

Eight days post discharge, she was readmitted with irritability and refusal to feed. Again, an SVT at a rate of 260 bpm was recorded. The baby was afebrile and hemodynamically stable. Four attempts to convert the rhythm with

adenosine at doses of up to 0.2 mg/kg were unsuccessful. Sinus rhythm was achieved more than 12 hours later, after a loading dose of procainamide and another dose of adenosine. No other clinical or laboratory abnormalities were noted. The child was discharged 3 days later on digoxin and propranolol treatment.

Discussion

SVT is not uncommon in children, with a reported incidence of 1 in 250-1000 (1). In 60% of cases, the arrhythmia is idiopathic, but in the remainder, there is an underlying disorder – such as Wolff-Parkinson-White syndrome (1, 2) or a structural defect of the heart, such as Ebstein's anomaly. Metabolic abnormalities, infections, and drugs may also cause SVT (2). The pediatric cardiology and emergency medicine literature recommends evaluation with electrocardiograms and echocardiograms only, after the SVT converts to sinus rhythm.

As the clinical manifestations of neonatal infection vary, and might be very subtle, most pediatricians advocate thorough evaluation and treatment at any hint of infection or systemic inflammatory response syndrome (3). Nevertheless, in infants who present with SVT, sepsis work-up is not recommended especially if there are no signs of sepsis, even though SVT has been reported to be a manifestation of an infection (2). In our case, a comprehensive laboratory work-up was performed because of the clinical observation of a coagulation disturbance, which could have been a sign of sepsis.

The positive PCR finding for enteroviruses in the stool in our patient was suggestive of an enteroviral infection. Enteroviral infection in neonates is usually asymptomatic (4), but if a disease develops, it might be followed by a severe clinical course and may be fatal. The main manifestations of disease, especially in fatal cases, include myocarditis, meningoencephalitis, and hepatitis (5).

SVT may be the preliminary manifestation of myocarditis (6,7). Two reports have described neonatal myocarditis due to enteroviral infection presenting as supraventricular arrhythmia (8,9). However, in our patient, there was no evidence of myocarditis: the electrocardiogram done after reversion to sinus rhythm was normal, blood

CPK and troponin levels were within normal range, and heart contraction was normal on the echocardiogram. However, we cannot rule out the possibility of a focal myocarditis in the atrium serving as a re-entry mechanism (8).

The combined finding of hepatitis with DIC usually suggests fulminant hepatitis. In enteroviral infections, the prognosis is grave, especially if pronounced jaundice, myocarditis, or meningoencephalitis develops (5,10). In our patient, the jaundice was only mild, and there was no evidence of meningoencephalitis, neither clinically nor in the CSF.

We believe this child had an enteroviral infection. The diagnosis of enterovirus infection requires growth in a viral culture (5) or a positive PCR finding in usually sterile locations (7). CSF PCR is more accurate than CSF culture for diagnosis. Our patient's viral cultures were negative, and PCR was positive only in the stool. However, in neonates, the presence of an enterovirus in the stool, in the clinical setting of arrhythmia, hepatitis and DIC, is highly suspicious - if not diagnostic - of an enteroviral disease.

The etiology of the SVT in our patient remains unclear. Was it a manifestation of an enterovirus infection, caused by focal myocarditis or sepsis? Or was it idiopathic? Did the evaluation for SVT in the hospital lead to the chance recognition and early treatment of the other potentially life-threatening condition of DIC and hepatitis? On the one hand, the second hospitalization for SVT, when there was no evidence of concurrent infection, could suggest that the SVT was idiopathic. On the other hand, recurrent SVT could also be residua of focal myocarditis.

In summary, we describe a neonate with SVT whose laboratory evaluation in the ED, performed because of a bleeding problem, was consistent with hepatitis and DIC, most probably due to an enterovirus infection. Although SVT is usually idiopathic in childhood, a more careful evaluation in affected neonates presenting to the ED might be in order, as SVT in this age group could indicate a more serious underlying disorder

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