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MAKING THE QUICK DIAGNOSIS: A CASE OF NEONATAL SHOCK

Mike Gardiner, MD,* Timothy K. Ruttan, MD,† Andrew J. Kienstra, MD,* and Matthew Wilkinson, MD*

*The University of Texas at Austin, Dell Medical School, Dell Children's Medical Center of Central Texas, Austin, Texas and †Department of Emergency Medicine, University of California Davis School of Medicine, Sacramento, California

Reprint Address: Mike Gardiner, MD, The University of Texas at Austin, Dell Children's Medical Center, 4900 Mueller Boulevard, Austin, TX 78723

□ **Abstract—Background: The work-up and initial management of a critically ill neonate is challenging and anxiety provoking for the Emergency Physician. While sepsis and critical congenital heart disease represent a large proportion of neonates presenting to the Emergency Department (ED) in shock, there are several additional etiologies to consider. Underlying metabolic, endocrinologic, gastrointestinal, neurologic, and traumatic disorders must be considered in a critically ill infant. Several potential etiologies will present with nonspecific and overlapping signs and symptoms, and the diagnosis often is not evident at the time of ED assessment. Case Report: We present the case of a neonate in shock, with a variety of nonspecific signs and symptoms who was ultimately diagnosed with tachycardia-induced cardiomyopathy secondary to a resolved dysrhythmia. Why Should an Emergency Physician Be Aware of This?: This case highlights the diagnostic and therapeutic approach to the critically ill neonate in the ED, and expands the differential diagnosis beyond sepsis and critical congenital heart disease. Knowledge of the potential life-threatening etiologies of shock in this population allows the Emergency Physician to appropriately test for, and empirically treat, several potential etiologies simultaneously. Additionally, we discuss the diagnosis and management of supraventricular tachycardia and Wolff-Parkinson-White syndrome in the neonatal and pediatric population, which is essential knowledge for an Emergency Physician.** © 2016 Elsevier Inc. All rights reserved.

□ **Keywords—neonatal shock; resuscitation; cardiomyopathy; supraventricular tachycardia; Wolff-Parkinson-White syndrome**

INTRODUCTION

The evaluation and treatment of a critically ill neonate presents an anxiety-provoking clinical challenge to the Emergency Physician. Although sepsis represents a significant portion of neonates presenting to the Emergency Department (ED) with shock, the differential diagnosis is broad, and common presenting signs and symptoms, such as lethargy, poor feeding, decreased tone, and irritability, are nonspecific. Adding to the challenge, several of the potential diagnoses are life-threatening, requiring emergent recognition and management to prevent significant morbidity or mortality. Due to these factors, it is important for the Emergency Physician to keep a broad differential diagnosis in mind, and to initiate early empiric treatment for several of the potential life-threatening causes, before definitive diagnosis. In this case, we present a neonate in shock with a variety of nonspecific signs and symptoms, and highlight the initial management, work-up, and ultimate diagnosis.

CASE REPORT

A 24-day-old male was brought into a community ED with poor feeding, lethargy, and pallor for 1 day. During the evening before presentation, the patient was reportedly feeding less vigorously, tolerating just 1 ounce of formula per feeding. He was also no longer waking

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spontaneously to feed. On the morning of presentation he became difficult to arouse, had trouble breathing, appeared pale, and was cool to touch. There was no history of vomiting, fever, cough, or nasal congestion, and no known trauma. He was born full term by spontaneous vaginal delivery at 41 weeks gestation after an uneventful pregnancy, which included routine prenatal care.

Upon arrival to the community ED, the patient was found to be ill-appearing, limp, difficult to arouse, and grunting. Initial vital signs included a rectal temperature of 33.2°C, heart rate 128 beats per minute (bpm), respiratory rate of 36 breaths/min, and peripheral oxygen saturation of 100% on room air. A glucose level was found to be low at 14 mg/dL. He was externally warmed, a peripheral i.v. catheter was placed, he was given 5 mL/kg 10% dextrose solution i.v., and a 20 mL/kg normal saline bolus. After these interventions, his rectal temperature was 35°C, glucose improved to 274 mg/dL, and he was noted to be increasingly active, at which point he was emergently transferred by air to a tertiary care pediatric ED (PED).

Upon arrival at the PED, the patient was found to be minimally responsive, mottled, and dusky-appearing. Rectal temperature on arrival was 29.5°C, heart rate was 120 bpm, lower-extremity blood pressure was 49/26 mm Hg, and his peripheral oxygen saturation was 87% while receiving bag-valve-mask ventilation with 100% oxygen by emergency medical services. His initial physical examination revealed a well-developed infant with an anterior fontanelle that was open, soft, and flat. His head was without signs of trauma. His pupils were equal and reactive, and the remainder of his head, eyes, ears, throat, and neck examination was unremarkable. He had poor respiratory effort, a midline trachea, and slightly coarse breath sounds. Cardiovascular examination revealed no murmur, rub, or gallop. His brachial pulses were weak and femoral pulses were not palpable. His abdomen was soft, nontender, and distended, with decreased bowel sounds noted. A firm liver edge was palpated 5 cm below the right costal margin and his spleen was not palpable. He had normal-appearing Tanner I male genitalia, and both testes were palpable in his scrotum.

A normal saline bolus of 10 mL/kg was administered, and the patient's oxygen saturation improved to 100% without improvement in his blood pressure. Rapid sequence intubation was performed with fentanyl, midazolam, rocuronium, and atropine. He was treated empirically with i.v. hydrocortisone, ampicillin, cefotaxime, and acyclovir. Initial venous blood gas was significant for a pH of 6.9, pCO_2 of 37 mm Hg, a base deficit of 25 mmol/L, and an elevated lactate of 15.9 mmol/L. White blood cell count was 27,300 cells/mm^3. Initial chemistry panel was significant for a HCO_3 of 6 mmol/L, anion gap of 24, an elevated blood urea nitrogen at 28 mg/dL, and normal creatinine of 0.7 mg/dL, aspartate aminotransferase and alanine aminotransferase levels of 263 U/L and 241 U/L, respectively, and a glucose level of 87 mg/dL. A chest radiograph showed mildly increased perihilar and peribronchial markings with a normal cardiothymic silhouette. Repeat blood pressure was 87/33 mm Hg in the right upper extremity, with a simultaneous right lower-extremity blood pressure of 40/23 mm Hg. Given the concern for ductal-dependent critical congenital heart disease (CHD), a prostaglandin infusion was initiated at 0.1 μg/kg/min.

After initial resuscitation, the patient clinically improved with stabilization of his vital signs with a temperature of 35.5°C, a heart rate of 153 bpm, blood pressure of 94/60 mm Hg, and a peripheral oxygen saturation of 100% while mechanically ventilated. The discrepancy between upper- and lower-extremity blood pressures persisted on repeated measurements. He was admitted to the pediatric intensive care unit (PICU) for further management.

On arrival in the PICU, the patient was found to have intermittent hypotension despite fluid resuscitation, and was started on milrinone and epinephrine infusions. An electrocardiogram (ECG) showed sinus tachycardia and nonspecific T wave abnormalities (Figure 1). A head computed tomography showed findings consistent with early ischemic changes, without evidence of acute trauma. An echocardiogram demonstrated normal cardiac anatomy and poor left ventricular contractility with an ejection fraction of 35%.

After the echocardiogram results, the prostaglandin infusion was discontinued. The patient continued on antimicrobials and additional infectious studies were sent, all of which were ultimately negative. Overnight and into the following morning, the patient remained hemodynamically stable on inotropic support and repeat echocardiogram showed improved ventricular contractility. Approximately 12 h after admission to the PICU, the patient developed persistent tachycardia with a heart rate of 270 bpm. An ECG showed narrow QRS complexes with no discernable P-waves (Figure 2). The patient was diagnosed with supraventricular tachycardia (SVT). Adenosine 0.1 mg/kg was given with successful conversion to a normal sinus rhythm.

CASE RESOLUTION

After resolution of the patient's SVT episode, a follow-up ECG was performed that showed evidence of ventricular pre-excitation with delta waves (Figure 3). The patient was diagnosed with Wolff-Parkinson-White (WPW)