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When Hoofbeats Means Zebras: Not All Tachypnea Is Bronchiolitis

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Presentation

A previously healthy full term 30-day-old male infant presented to his pediatrician appointment for a 1-month well child check. Birth history was unremarkable: 37 6/7 weeks gestational age, vaginal delivery, APGARs 9/9, birth weight 3kg, normal prenatal screens except for GBS positive with adequate treatment. Per the infant's mother, he intermittently had tachypnea since birth but otherwise was doing well. He had been gaining weight appropriately despite minor emesis thought to be secondary to reflux, for which he had been to the pediatrician twice in his one month of life. Mom described no cyanosis, sweating, or worsening tachypnea with feeds. Due to the reflux, mom had switched formulas multiple times since discontinuing breastfeeding at several days of life. At the pediatrician appointment on the day of admission, he was noted to be tachypneic with saturations in the mid-80's. He was taken via ambulance with blow-by oxygen to an outside hospital emergency room. There, he was placed on room air with appropriate saturations and mild tachypnea. He was transferred to our Level 4 NICU for further work-up with a sepsis evaluation. On arrival to the NICU, respiratory support was escalated to 5L high-flow nasal cannula for continued tachypnea and retractions. He was otherwise well-appearing with good perfusion in all extremities, no edema, and appropriate capillary refill. No rhinorrhea or upper respiratory infection symptoms were noted on exam. A soft, systolic murmur was auscultated and determined clinically to be a flow murmur. Lower extremity blood pressures were appropriate, and upper extremity pressures were deferred due to IV infusions in both upper extremities. CXR obtained at the outside hospital was reviewed on admission and noted to be concerning for a boot-shaped heart, bilateral perihilar streakiness, and cardiomegaly. Initial screening labs (CBC, CRP, CMP), respiratory viral panel, blood culture, urine culture, and COVID-19 were all drawn. Ultimately, cardiology was consulted, and an echocardiogram revealed the diagnosis.

Discussion

The differential for tachypnea in a 4-week-old infant is broad. The initial differential included viral bronchiolitis (including COVID-19), sepsis, metabolic derangement, and congenital heart disease. Initial laboratory results yielded normal CRP, unremarkable urinalysis, appropriate gas and lactate without acidosis, negative respiratory viral panel, and normal electrolytes. His CBC showed mild leukopenia without bandemia, thrombocytopenia, and a normal hematocrit. Initially, the infant had bilateral upper extremity peripheral IVs actively infusing, so no upper extremity blood pressures were able to be obtained. However, several hours into his admission he lost his right upper extremity IV and a new right upper extremity blood pressure (BP) was obtained with a differential of more than 50mmHg in his systolic from the lower extremity BPs. On repeat examination, it was discovered that there was a significant difference in his femoral and brachial pulses. Perfusion remained appropriate. Cardiology was consulted and an echocardiogram revealed a mild Shone's variant with severe coarctation of the aorta (CoA), bicuspid aortic valve, and mild mitral stenosis. He continued to have no lactic acidosis but was noted to have mild left ventricle dysfunction. He was urgently started on Prostaglandin-E1 and transferred to the CICU, where he ultimately had end-to-end aortic anastomosis, atrial septal defect closure, and patent ductus arteriosus (PDA) ligation with improvement in his left ventricle function and blood pressure differentials. After two weeks in the CICU, he was discharged home on an anti-hypertensive medication and fortified formula to improve weight gain.

Coarctation of the aorta makes up approximately 5% of all congenital heart defects and occurs in 0.4% of live births [1]. It is often an isolated finding, but can occur with other heart defects, such as valve abnormalities (most commonly bicuspid

aortic valve) or septal defects [2]. The exact pathogenesis of CoA is unknown, although it is hypothesized that the defect arises from abnormal development, reduced intrauterine blood flow, or constriction from extra PDA tissue [3,4]. Once suspected, CoA is most efficiently diagnosed with echocardiography. Surgical resection of the narrowed segment is the treatment of choice.

CoA is the most commonly-missed congenital heart disease. Some note it to be the most difficult congenital heart disorder to diagnose because of its timing often after discharge from the newborn nursery and lack of a murmur [1]. Typically, the presentation of CoA occurs in the first two weeks of life when the PDA closes, and there is a significant decrease in blood flow to the lower half of the body. However, even at an age older than two weeks, physicians should be weary of a CoA in a patient with blood pressure and pulse discrepancies. Less than a third of infants who were ultimately diagnosed with CoA were correctly identified by their outpatient referring physician [5]. Thus, it is important to remember that physical exam (specifically upper and lower extremity pulses, upper and lower extremity blood pressures, perfusion, and a systolic murmur) is critical to diagnosing CoA. Although many physicians rely on palpating only the femoral pulses, it is important to compare with the with the upper extremity pulses in order to detect a differential. Most patients with CoA will have decreased femoral pulses, but only 20% will have absent pulses [6]. Thus, palpation of just the lower extremity pulses is not a dependable manner of screening for a CoA. Two blood pressures taken in the upper and the lower extremities is a specific and reliable method to screen for CoA [6]. If discovered late, critical CoA can lead to detrimental effects in the perfusion of the lower extremities and abdominal organs, leading to shock and acidosis. Additionally, late diagnosis and thus repair of CoA is associated with hypertension and premature cardiovascular disease with correlated morbidity and mortality in adults [7]. Continued evaluation of blood pressure is therefore an important aspect of follow-up for patients with CoA.

Lessons for the Clinician

- Coarctation of the aorta continues to be the most commonly-missed congenital heart disease and physicians should have a high level of suspicion, even when presenting in an older infant such as this case. It is important to diagnose as early as possible to avoid cardiogenic shock, loss of perfusion to internal organs and lower extremities, and late hypertension.

- Upper versus lower blood pressure differential is the most specific way to screen for CoA. If evaluating the pulses, it is important to compare both the upper and lower pulses.

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