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Case Report

Refractory Neonatal Hypertension: A Challenging Case

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Abstract

Neonatal hypertension is a rare condition that can be asymptomatic or accompanied by nonspecific signs. We describe a case of a 7-day-old female newborn admitted to the Emergency Department with a history of poor feeding and lethargy, presenting with respiratory distress, cardiac murmur, hepatomegaly and weight loss. Sustained hypertension was present at admission. Laboratory evaluation revealed acute kidney injury and transthoracic echocardiogram showed severe left ventricular dysfunction. Despite improvement of cardiac function, antihypertensive therapy was not effective in lowering blood pressure. Etiological investigation included a renal Doppler ultrasound, showing unilateral left renal hypoperfusion, and a renal angiography revealing left renal artery stenosis. A dilatation of this stenosis via percutaneous transluminal angioplasty was performed, followed by normalization of blood pressure and progressive weaning of antihypertensive therapy. Angiography remains the gold standard test allowing both diagnosis confirmation and therapeutic intervention. Prompt diagnosis of neonatal hypertension as well as etiological investigation is essential to assure timely therapeutic institution and prevention of target organ damage.

Keywords: *Neonatal Hypertension (NHT); Blood Pressures (BP); Left Ventricular Dysfunction*

Introduction

Neonatal hypertension (NHT) is defined as persistent systolic and/or diastolic blood pressures (BP) above the 95th percentile for postmenstrual age [1-3]. It is a rare condition with an incidence from 0.2 to 3% [1,2], with 3 to 25% of the cases due to renovascular hypertension (HT) [3,4]. This disorder may be asymptomatic or accompanied by nonspecific signs such as lethargy or poor feeding [1-4]. Rarely NHT can present as acute heart failure and cardiogenic shock [1,2]. We describe the case of a female newborn with refractory HT, presenting as acute heart failure. Written informed consent for patient information and images to be published was provided by a legally authorized representative.

Case Presentation

We present the case of a 7-day-old term newborn female, with an uneventful pregnancy. Routine prenatal screening and obstetric ultrasounds were normal, without history of gestational hypertension. She was born at 38 weeks via eutocic delivery, 3225gr birthweight. Postnatal course was unremarkable. There was a family history of hypertension in adult age.

On the 7th day of life the child presented to the Emergency Department due to poor feeding and lethargy. Parents denied other symptoms. On examination, she had generalized jaundice, hydrated mucosa but a 5% weight loss; significant tachypnea (75-80 cycles/minute), grunting and chest retractions, with normal pulmonary auscultation; ongoing sinus tachycardia even when calm (150-160 beats/minute) and HT (left arm 115/72 mmHg, above the 99th percentile for postmenstrual age), without differential between upper and lower limbs; grade II/VI heart murmur; and 3 cm palpable hepatomegaly; femoral pulses present and symmetric; reactive to stimulus but lethargic, with little spontaneous movement.

Initial investigation revealed cardiomegaly (cardiothoracic ratio of 0.62), mild metabolic acidosis with high lactate level (venous blood gas with pH 7.34, pCO_2 35 mmHg, HCO_3 18.9 mmol/L, lactate 53 mg/dL), acute kidney injury (serum creatinine 1.2 mg/dL, glomerular filtration rate 21 mL/min/1.73m^2, blood nitrogen urea 49 mg/dL) and indirect hyperbilirubinemia (total bilirubin 21 mg/dL), with mild hematuria in urinalysis. Serum ionogram was normal and inflammatory parameters were negative. Urine and blood cultures were collected.

Neonatal sepsis was presumed, and she was started on empiric intravenous antibiotic therapy with ampicillin and cefotaxime, although repeat inflammatory parameters and cultures remain negative. A transthoracic echocardiogram was performed, that revealed an ostium secundum-type atrial septal defect, left cardiac chambers dilation, interventricular septum hypertrophy (4.4 mm zs+2.4) and severe left ventricular dysfunction (ejection fraction of 27%). The 12-lead electrocardiogram was normal.

These findings were consistent with NHT with acute heart failure, and the patient was transferred to a paediatric intensive care unit (PICU). At admission on the 8th day of life, milrinone (0.5 mcg/kg/min) and non-invasive ventilation with continuous positive airway pressure (nasal CPAP) were started to support left ventricular function, along with antihypertensive treatment with furosemide, amlodipine, and captopril. Due to ongoing HT above 95th percentile, captopril was stopped, and labetalol perfusion was initiated on the 9th day of life, with no sustained BP response (maximum dose 3 mg/kg/h). Nonetheless, there was a progressive recovery of cardiac function with an ejection fraction that improved to 50% on the 10th day of life, allowing for weaning milrinone to 0.2 mcg/kg/min and withdrawing CPAP support.

Etiologic investigation included a renal Doppler ultrasound that revealed an irregular left renal artery with severe reduction of left renal perfusion. Laboratory studies showed elevated NT-proBNP and troponinT (max 85758 pg/mL and 354 ng/L, respectively), elevated serum renin and aldosterone (> 1.000 µUI/mL and > 2.000 pg/mL, respectively), with a normal endocrinological evaluation (cortisol 10.3 ug/dL, adrenocorticotropic hormone 12.3 pg/mL, free thyroxine 2.49 ng/dL, thyroid stimulating hormone 6.62 uU/mL, dehydroepiandrosterone 199 ug/dL). On the 11th day of life she was submitted to a diagnostic angiography that showed a critical stenosis of the proximal third of the left renal artery with distal hypoperfusion (Figure 1), followed by angioplasty with multiple dilatations (high-pressure balloon to maximum of 24atm, Figure 2). Post procedure angiography showed notable improvement of left renal artery's calibre and perfusion, followed by quick normalization of BP and progressive normalization of renin, NT-proBNP, troponinT, BUN and creatinine levels.

Antihypertensive therapy was stopped on the same day and heart failure medication was weaned and stopped on the 13th day of life. After two days the patient was transferred to the paediatric ward with normal BP (P50 for postmenstrual age), adequate weight

progression and normal neurologic evaluation. Serial transcranial Doppler were normal. She was discharged home on the 20th day of life. On re-evaluation 3 weeks after discharge she presented with normal clinical examination and a good weight gain (30g per day).

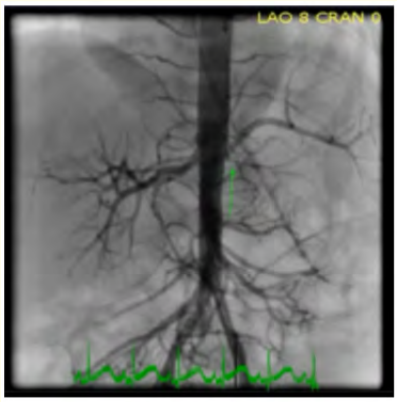

Figure 1

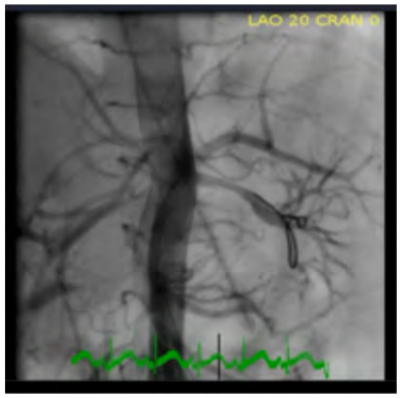

Figure 2

Figure 1 and 2: *1: Left renal artery stenosis with distal hypoperfusion (green arrow); 2: Renal vasculature after angioplasty.*

Discussion

NHT is a rare disorder in neonatal population, with variable incidence depending upon the clinical setting. In otherwise healthy term infants, hypertension is exceedingly uncommon with a reported rate of 0.2% [5]. In infants admitted to a neonatal intensive care unit (NICU), the incidence is higher with reported rates ranging from 0.7 to 3% [1,2].

It is usually secondary and related to a variety of risk factors, including low gestational age and birth weight, specific diseases (e.g. bronchopulmonary dysplasia, cardiac disease and acute kidney injury), use of umbilical artery catheters, and severe neonatal illness [1,2,5]. Most cases of NHT are due to renovascular pathology [1-3,6]. Stenosis occurring in the main or segmental renal artery, as in this case, causes renal hypoperfusion that will induce secretion of renin by the juxtaglomerular apparatus, with rising levels of angiotensin II. This will cause vasoconstriction in renal efferent arterioles, elevating glomerular filtration pressure and BP in more proximal vessels [3]. The raised BP and glomerular hyperfiltration will subsequently stimulate pressure natriuresis by the intact contralateral kidney, preventing volume expansion and sodium retention. The nonstenotic kidney tends to counteract the elevation of BP, maintaining the reduced perfusion of the stenotic kidney and leading to continuous increased renin release [3].

Regarding diagnosis, initial evaluation of HT requires the following laboratory tests: serum creatinine, electrolytes, blood urea nitrogen, urinalysis, complete blood count, and fasting plasma glucose [3]. In addition to monitoring BP, cardiac evaluation with an echocardiogram is of utmost importance to rule out structural heart disease and possible systolic dysfunction with a reduction in ejection fraction when acute heart failure occurs, as in the present case [2]. When possible, an ophthalmological evaluation with fundoscopy should be requested to detect macular alterations [6].

Renal ultrasonography with Doppler evaluation is widely used as a screening test for renal artery stenosis due to its low cost, widespread availability, and lack of ionizing radiation exposure, sedation, or intravascular contrast. However, there are considerable limitations in identifying the accessory renal artery, branch renal artery, and intrarenal renal artery stenosis [3]. Despite these limitations, this exam allowed a presumptive diagnosis in our case, which was later confirmed by angiography. Angiography remains the gold standard test for diagnosis [1], allowing therapeutic intervention at the same time as confirming the diagnosis of renal artery stenosis.

Therefore, prompt recognition and treatment for underlying systemic HT are crucial to provide the best possible outcomes [2]. There are no specific guidelines for the therapeutic approach in this age range, so the recommended treatment is mainly based on experts' opinion [6,7]. The initial therapeutic approach should be correction of iatrogenic and reversible causes, followed by thorough consideration of pharmacological treatment with one single anti-hypertensive medication, including angiotensin-converting enzyme (ACE) inhibitors, diuretics and beta-blockers; if there is not control of BP, the medication's doses should be increased or it should be combined with a different anti-hypertensive drug [7,8].

In this case, there was a multidisciplinary discussion with Paediatric Nephrology and Paediatric Cardiology to decide the best therapeutic approach, and regarding the sustained BP presenting as acute heart failure it was decided to start a combination of different anti-hypertensive medications, including a diuretic due to the clinical presentation with heart failure. However, the angiography was essential to the treatment of this patient, with sustained control of BP. NHT due to renal artery stenosis usually requires a surgical intervention, and percutaneous or surgical revascularization is recommended, as performed on the present case. This procedure is essential to preserve renal function and prevent target organ damage, and sometimes it also avoids the need for anti-hypertensive medication, as in this case.

There are few studies regarding the follow-up of these patients. Despite this, it is important to maintain periodic surveillance to control BP and medication adjustments, as well as to identify complications [6]. Available data suggest that long-term outcomes are usually good, with resolution of HT in most infants [1,2,5,8].

Conclusion

This clinical report is a rare case of HT in a newborn presenting as an acute heart failure, with complete resolution after angiographic dilatation of unilateral renal artery stenosis. Prompt diagnosis and etiological investigation are essential to assure timely therapeutic institution and prevention of target organ damage. There is a great need for further research to support efficacy, safety, and standardization of therapeutic interventions for NHT to provide the best clinical outcomes.

Conflict of Interest

The authors declare that there is no conflict of interest.

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