

Hemodynamic Echoguided Management in Neonatal Cardiogenic Shock

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Received: 01 November 2020; **Accepted:** 16 December 2020

Citation: Baquedano Lobera I, Rite Gracia S, Palanca Arias D. Hemodynamic echoguided management in neonatal cardiogenic shock. J Pediatr Neonatal. 2020; 2(2): 1-4.

ABSTRACT

Introduction: Neonatal cardiogenic shock is an uncommon condition whose detection and management continues to be a major challenge in the NICU.

Clinical case: We present a case of neonatal cardiogenic shock in a 25 days old newborn admitted in the NICU due to renal failure, where the point-of-care ultrasound was the main tool for hemodynamic management. After progressive deterioration of renal function (creatinine 5.67mg/dl, urea 96mg/dl), she needs peritoneal dialysis during 6 days. Two weeks after the extrarrenal assistance withdrawal, and simultaneously with a blood transfusion, she suffers a clinical descompensation with maintained blood pressure >p99, tachycardia, oligoanuria and respiratory distress needing CPAP. Point-of-care ultrasound is practiced showing systolic dysfunction and left ventricle dilatation (left ventricle ejection fraction 23%), moderate mitral and tricuspid insufficiency, mild aortic insufficiency and acute pulmonary edema findings. The clinical signs supported by the echographical findings presuppose an increased systemic postcharge within nephrogenic hypertension and hypervolemia due to blood transfusion, triggering a left heart failure with postcapillary pulmonary hypertension, acute pulmonary edema and, secondarily, right heart overload. Diuretics are started (furosemide=1mg/kg/h) and inodilators (milrinone=0.75mcgr/kg/min and dobutamine=5mcgr/kg/min), monitoring the hemodynamic response with point-of-care ultrasound, adding four hours later vasodilators (nitroprusside=0, 75 mcgr/kg/min). Early heart failure improvement is observed, withdrawing all hemodynamic support after 36 hours, with recovery of myocardial contractility, normalisation of the valve insufficiencies, and resolution of pulmonary edema.

Conclusions: Point-of-care ultrasound guided the physiopathology of the cardiogenic shock as well as the therapeutic decisions allowing optimizing the hemodynamic management, minimizing therapeutic delays and early reverting the life-threatening descompensation.

Keywords

Cardiogenic shock, Neonate, Point-of-care ultrasound, Hemodynamic management, NICU.

period whose detection and management continues to be a major challenge in the NICU. When it occurs, it usually affects newborns with congenital heart diseases that trigger the shock condition [1].

Introduction

Cardiogenic shock is an uncommon condition in the neonatal

The shock scenario comprises hemodynamic instability characterized by tissular hypoperfusion. There are different types

of neonatal shock depending on the physiopathological cause that unleashes the hemodynamic instability (hypovolemic, distributive, cardiogenic, obstructive, multifactorial), although these causes are not selective, and it is not uncommon to find it hard to promptly and correctly identify the etiopathogenic cause.

Therefore, we have an accessible and non-invasive tool which can be very helpful in the diagnosis and management of these patients, the point-of-care ultrasound (POC-US). POC-US has been gaining space among the neonatal intensive care units (NICU) during the last years, and it allows to perform a functional evaluation of the patient in order to establish the diagnosis, as well as monitor response to treatment and optimize management and evolution [2,3].

It is a safe tool, non-invasive, and it provides very important information and complementary to the rest of the guide datum of shock (vitals, physical examination, laboratory results). Furthermore, it allows obtaining frequent information in a very accessible way.

Functional evaluation and parameter measurement such as the left ventricle ejection fraction (LVEF) and the inferior vena cava (IVC) collapsibility can guide the cause of the shock and help optimizing its management, as well as monitoring the therapeutic response [4].

Clinical case

We present a case of cardiogenic shock in a 25 days old newborn admitted in the NICU due to renal failure, where the point-of-care ultrasound (POC-US) was the main tool for the hemodynamic management.

She had history of prematurity (34+6 weeks of gestational age), Apgar 4/7 and 7-day-long amniotic membrane rupture. During the early neonatal period she presents anemia, thrombocytopenia,

oligoanuria, macroscopic hematuria, hyperuricemia and high serum creatinine. Hemolytic uremic syndrome is ruled out.

After progressive deterioration of renal function (creatinine 5.67mg/dl, urea 96mg/dl), she needs peritoneal dialysis during 6 days. Two weeks after the extra renal assistance withdrawal, and simultaneously with a blood transfusion, she suffers a clinical descompensation with maintained blood pressure >99 (115/80 mmHg), lethargy, tachycardia, oligoanuria and respiratory distress needing CPAP.

POC-US is practiced showing systolic dysfunction and left ventricle dilatation (LVEF 23%, DDVI 23mm (Z score +2.8)), moderate mitral and tricuspid insufficiency (Figure 1a), mild aortic insufficiency, diastolic dysfunction (Figure 2a) and acute pulmonary edema findings (Figure 3a).

The clinical signs supported by the echographical findings presuppose an increased systemic postcharge within nephrogenic hypertension and hypervolemia (dilated IVC with low collapsibility) due to blood transfusion, triggering a left heart failure with postcapillary pulmonary hypertension, acute pulmonary edema and, secondarily, right heart overload. With those findings inodilator support is started with milrinone (0.75 mcgr/kg/min), combining with diuretics (furosemide 1 mg/kg/h).

The hemodynamic response is monitored with POC-US, in order to value and support therapeutic decisions, adding four hours later vasodilators (nitroprusside 0, 75 mcgr/kg/min).

Early heart failure improvement is observed, withdrawing all hemodynamic support after 36 hours, with recovery of myocardial contractility (LVEF $>70\%$), normalisation of the valve insufficiencies and pulmonary hypertension, and resolution of pulmonary edema (Figures 1b, 2b, 3b).

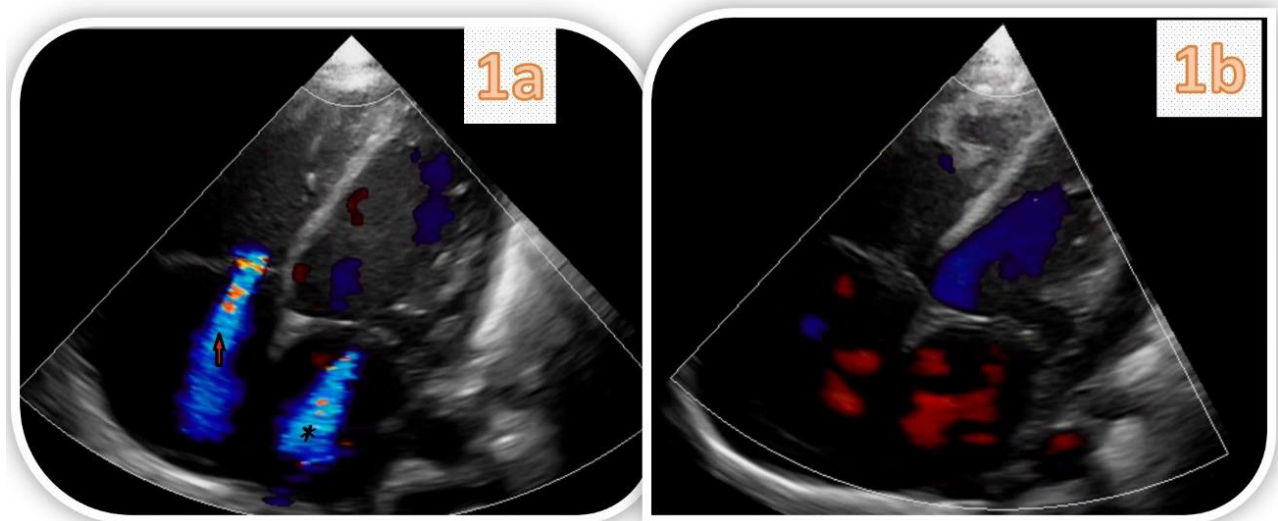


Figure 1: Apical four-chamber plane with colour-doppler. **1a.** Moderate mitral insufficiency reaching the left atrium ceiling (*) and moderate tricuspid insufficiency (arrow). **1b.** Normalisation of both valve insufficiencies after 36 hours of inotropic therapy.

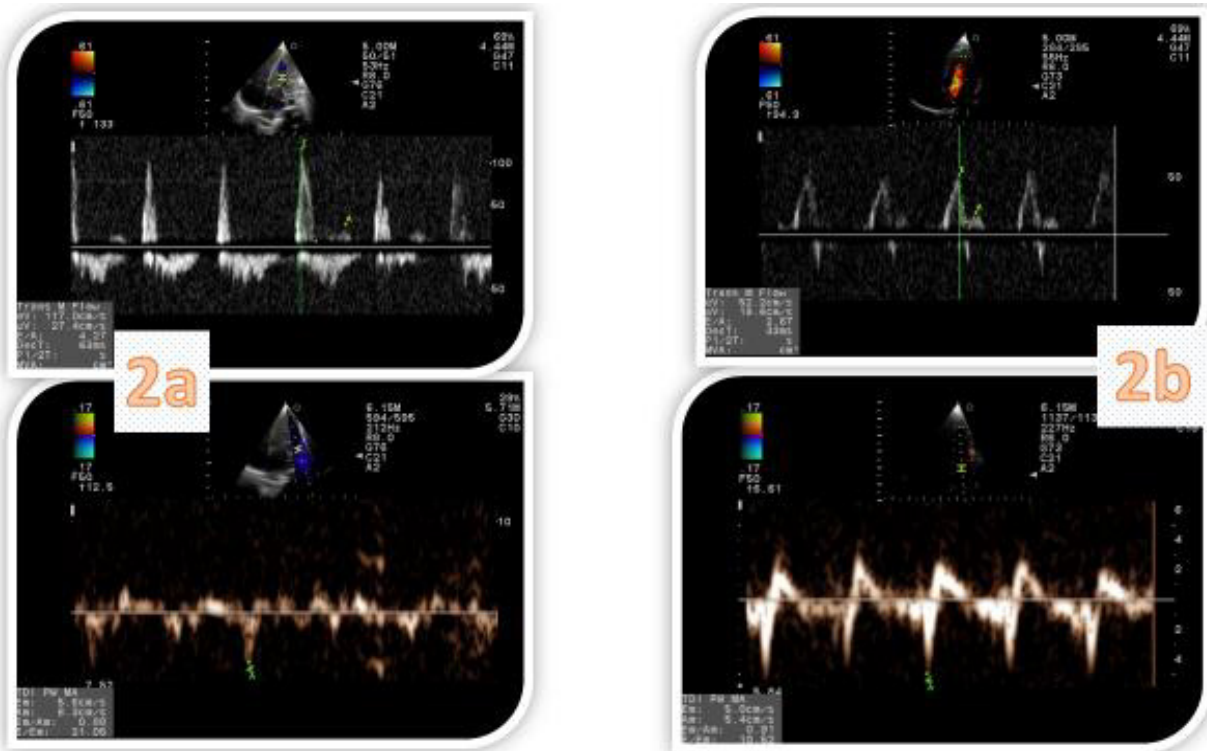


Figure 2: Apical four-chamber plane (transmitral pulsed doppler – upper images, and lateral wall tissue doppler – lower images). **2a.** Dyastolic dysfunction with restrictive pattern, with E/A index=4.7 and E/E' index=21 suggesting very high pulmonary capillary pressure. **2b.** Recovery of dyastolic dysfunction with E/A index=2.6 and E/E' index=10.5.

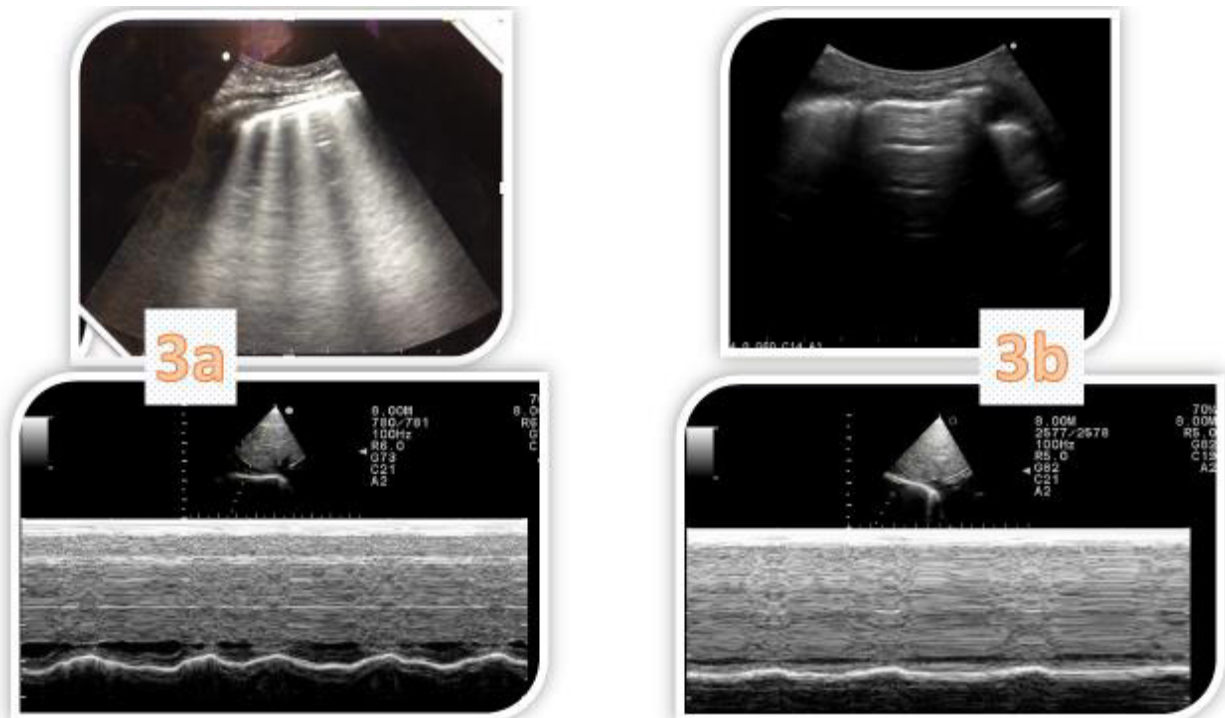


Figure 3: Pulmonary ultrasound and evaluation of inferior vena cava (IVC) collapsibility. **3a.** Billaterally thickened B lines pattern (upper left image) and subcostal M mode showing dilated IVC with low collapsibility (lower left image) suggesting acute pulmonary edema. **3b.** Billateral normal pulmonary pattern with A lines (upper right image), and subcostal M mode with normal IVC collapsibility (lower right image).

The patient was discharged home after several days and follows periodic check-ups in the pediatric cardiology department. The patient is currently asymptomatic, with minimum mitral insufficiency in the last echocardiographic reviews, and does not need any medication.

Discussion

NICU are a complex environment where detection of neonatal shock continues to be a challenge. POC-US provides complementary information to clinical and analytical management of shock, and helps to monitor treatment response.

POC-US has proved to be a useful tool but not universally. Its use for cardiovascular, pulmonar and transfontanelar evaluation in certain circumstances and diseases has a grading quality of evidence from A to D [2].

Besides the already existing validity of some parameters whose values are indicative of shock, some other parameters have also been described to help distinguish the different etiopathogenic possibilities. Thus, the IVC collapsibility index and the left ventricle area at the end of diastole, help to differentiate between cardiogenic and septic shock, finding the first one decreased and the second one increased in the cardiogenic shock [4].

In our case, POC-US guided the physiopathology of the cardiogenic shock as well as the therapeutic decisions allowing optimizing the hemodynamic management, minimizing therapeutic delays and early reverting the life-threatening descompensation.

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