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Severe myocardial injury and extracorporeal membrane oxygenation following perinatal asphyxia

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ABSTRACT

Perinatal asphyxia is a common cause of morbidity and mortality in the newborn and is associated with myocardial injury in a significant proportion of cases. Biomarkers, echocardiography, and rhythm disturbances are sensitive indicators of myocardial ischemia and may predict mortality. We present a case of severe myocardial dysfunction immediately after delivery managed with extracorporeal membrane oxygenation (ECMO) and discuss the role of cardiac biomarkers, echocardiography, electrocardiography, and ECMO in the asphyxiated newborn.

Perinatal asphyxia is a common cause of morbidity and mortality in the newborn period with an incidence between 0.5 and 2% [1,2]. The newborn myocardium is preferentially spared during the early phase of newborn hypoxemia; however, evidence of myocardial injury is present in 29–78% of newborns diagnosed with hypoxic-ischemic encephalopathy (HIE) [3–5]. Global oxygen deprivation, which is suggested by a 5 min Apgar score less than 7, underlies the pathogenesis of myocardial injury in the newborn period [6,7]. Additionally, biomarkers of myocardial injury (i.e. troponin and CK-MB), echocardiographic abnormalities, and rhythm disturbances are sensitive indicators of myocardial ischemia and may predict mortality [7]. We present a case of an infant who developed severe myocardial dysfunction following emergent Caesarian delivery for fetal decelarations and was managed with extracorporeal membrane oxygenation (ECMO). We discuss the role of cardiac biomarkers, echocardiography, electrocardiography (ECG), and ECMO in the asphyxiated newborn.

1. Case presentation

A 22-year-old G_1P_0 mother presented to a regional hospital with symptoms of labor at 35 weeks gestation. The perinatal history was significant for insulin-dependent diabetes mellitus and chronic hypertension. A normal anatomic ultrasound was noted, however fetal echocardiography was not performed. Following a trial of labor and poor progression, late decelerations were noted and the infant was delivered by Caesarian section. Upon delivery, the 2,700 g infant appeared floppy without spontaneous respiratory effort and persistent bradycardia. Resuscitative efforts in the delivery room included intubation, ventilation, chest compressions (15 min), multiple fluid boluses, and epinephrine. APGAR scores were 1, 3, and 5 at 1, 5, and 10 min, respectively and an initial arterial blood gas provided evidence of severe metabolic acidosis with a pH of 6.81 and base deficit of −23. The patient was transferred to the in-hospital Level III NICU and was noted to have spontaneous respirations and movement with significant hypoxemia (70–80% saturations on FiO2 of 1.0). However, the patient experienced prolonged bradycardia (60–70 b.p.m.) and a corresponding rhythm strip revealed ST segment elevation. A continuous infusion of epinephrine was initiated and the infant was transferred to our institution for further care at 2 h of life.

Active whole-body cooling was initiated on arrival to our NICU. A chest X-ray following admission revealed a normal cardiac silhouette and mild interstitial markings and echocardiography demonstrated a thickened interventricular septum, left ventricular ejection fraction of 21% and severe pulmonary hypertension. A continuous infusion of milrinone at 0.5 mcg/kg/min was initiated and multiple fluid boluses and inotropic support with dopamine and dobutamine were necessary for adequate perfusion. Epinephrine was discontinued following stabilization of blood pressure and

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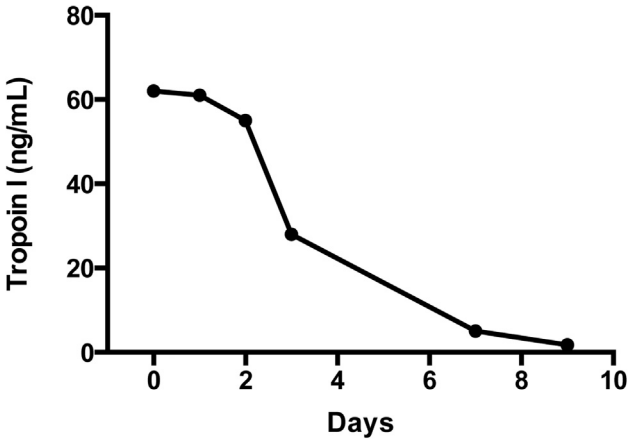

Fig. 1. Troponin I levels (ng/dl) over the first 10 days of life. Initial value was 62 ng/mL (normal 0.02–0.06 ng/mL).

resolution of bradycardia. Arterial blood gas sampling on FiO_2 of 1.0 and high frequency oscillatory ventilation was 7.1/57/28/17/-13. An initial lactic acid was measured at 11.2 mmol/L and troponin I was measured at 62 ng/mL (normal 0.02–0.06 ng/mL). Inhaled nitric oxide therapy was initiated for persistent pulmonary hypertension with minimal improvement in blood oxygen content. Acceptable perfusion and blood pressure were obtained and maintained with inotropic support. The infant was placed on veno-venous (VV) ECMO at 10 h of life. The collaborative decision to place the infant on VV ECMO was based on recent center-specific experience of patients with cardiomyopathy, which demonstrates that these patients can be effectively supported on VV ECMO with continued inotropic support. Recently, we successfully supported two infants of diabetic mothers with cardiomyopathy on VV ECMO and were discharged home. Our decision included a plan to convert to VA ECMO if necessary. The ability to wean inotropes can be used as an indirect marker of improved cardiac function and, in fact, dopamine and dobutamine were weaned and discontinued at 48 h of life and milrinone was continued for afterload reduction.

Corresponding with the patient's ability to wean inotropes, serum tropoinin and lactic acid levels improved (Fig. 1). Serial ECHOs failed to demonstrate improvement in cardiac function (EF ~ 20%); however, the infant was clinically stable with improved acid/base balance and adequate oxygenation. On DOL 7, infant was weaned to 30% oxygen and 0.4 L/min of sweep gas via ECMO with improved oxygenation and ventilation. On DOL 9, the infant deteriorated rapidly and an ECHO demonstrated absent right ventricular function and cor pulmonale. Following discussion about prognosis with the family, the parents elected to withdraw ECMO and ventilator support. The infant expired following discontinuation of ECMO.

2. Discussion

The etiologic event leading to myocardial failure in our patient was likely perinatal asphyxia resulting in global hypoxia and cardiac ischemia, which was superimposed on predisposing maternal risk factors including diabetes mellitus. Other common etiologies of sudden myocardial failure in the newborn period include paradoxical coronary embolism, thrombosis, enteroviral myocarditis, erythroblastosis fetalis, and congenital cardiac anomalies [8]. The diagnosis of myocardial failure with associated pulmonary hypertension secondary to perinatal asphyxia was suggested by patient history, ECG, echocardiography, and biomarkers.

ECG abnormalities suggestive of ischemia have been noted in 19% of neonates with asphyxia [3], but are only reliable after the first 24 h of life [9]. Jedeiken et al. proposed a 4-stage grading system of myocardial ischemia based on a comprehensive assessment of ECGs from "at risk" infants [9]. Validation studies of the grading system suggested that infants with ECGs meeting criteria for grade 3 and 4 corresponded with evidence of myocardial ischemia and were associated with higher mortality in the setting of perinatal asphyxia [2,7,10]. Our patient's initial ECG revealed diffuse ST segment elevation suggestive of global ischemia

Table 1
Summary of EKG, ECHO, and cardiac biomarkers for patients with perinatal myocardial ischemia who were managed with ECMO.

Year	Author	Arrest	EKG	Echocardiogram	Cardiac biomarkers
2014	Ham	Yes	Diffuse ST elevation	EF 21%, severe pulmonary hypertension, thickened interventricular septum, poor biventricular function	Troponin I 62.45 ng/mL
2014	Deutsch	No	ST elevation in the anterior leads and ST depression in the inferior leads.	EF 27%. Anterolateral, apical, and inferior wall akinesis. Moderate AV valve regurgitation	Troponin T 24.72 ng/mL
2012	Farooqi	No	Q-waves in Leads I and II, with ST segment changes in the precordial leads	EF 43% and left ventricular wall motion abnormalities	Troponin I 2.73 ng/mL
2009	Ferns	Yes	Q waves and ST elevation in lateral leads, T wave inversion in leads V4–V6, and poor R wave progression with ST depression in V1–V4.	Severe supravalvularaortic obstruction and extremely poor biventricularfunction	Troponin T 1.9 ng/mL
2009	Ferns	No	Anterolateral myocardialinfarction	Fractional shortening of 15%, dilated left atrium, impaired LV posterior wall function, and severe mitral regurgitation	Troponin T 0.3 ng/mL
2009	Ferns	No	Anterolateral Q wave myocardial infarction	Dilated left ventricle with marked septal and posterior wall dyskinesia	Troponin T 10.5 ng/mL
1997	Saker	No	Q waves in leads I, V5, and V6	Fractional shortening 21%, Poor function, AV valve regurgitation, PDA, no regional wall motion abnormalities.	CK 4478 U/L CK-MB 4 U/L CPK 4478 U/L LDH 2043 U/L
1996	Tometski	No	ST elevation in V4–V6	Fractional shortening 22%, left ventricular wall and apex akinesis.	CK = 663 IU/L

History of cardiac arrest, EKG and ECHO findings as well as serum cardiac enzymes are listed.