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Acute Myocardial Infarction in A Premature Baby with Respiratory Distress Syndrome and Cardiogenic Shock Early Recognition and Successful Treatment

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Abstract

Acute myocardial infarction is a life-threatening condition rarely recognized in premature babies with respiratory distress syndrome [17]. The patients usually present with poor left ventricular function or cardiogenic shock. Clinical management of these premature babies is extremely challenging it requires a high index of suspicion and early recognition for intensive care management to be successful. Furthermore, there is a great potential for myocardial regeneration to occur in surviving neonates with myocardial infarction. The present report describes a two-day old premature baby born at 33 weeks of Gestational age with an anatomically normal heart and normal coronary arteries who developed clinical, laboratory, electrocardiographic, and angiographic features of myocardial infarction secondary to thromboembolic occlusion of the left circumflex coronary artery. Coronary artery patency restoration was performed by selective intracoronary lysis with recombinant tissue plasminogen activator (r-TPA) and nitroglycerin.

Introduction

Cases of myocardial infarction in premature neonates with or without congenital heart disease are sporadic. Mortality rate has been estimated between 50 to 90% [1,11] with causes being attributed to Congenital Heart disease [5,15], perinatal asphyxia [2,6], Coagulopathy [5], hydrops fetalis [9], thromboembolism, and double exchange transfusion [13]. The present

report describes a premature baby with normal heart and normal coronary arteries who experienced an acute myocardial infarction on day 2 of life and was successfully treated with SIMV, surfactant, fluid resuscitation, inotropes, vasodilators and intracoronary injection of nitroglycerin and r-TPA.

Case Report

Premature female infant product of a 33-week gestation, born via cesarean section to a 20 year old mother who was G1 P2 at the time of delivery. Mother's prenatal labs were unremarkable. Pregnancy was complicated by Di-Chorionic Di-amniotic Twin pregnancy and preterm labor and fever up to 101.5 F during labor. Late decelerations during labor. APGAR score was 4 and 8 at 1 minutes and 5 minutes respectively. The baby required PPV for poor respiratory effort and desaturations. Was subsequently transferred to the NICU and placed on NCPAP of 6 with 35% FiO2 with O2 Saturations: 94%, infant was intermittently grunting and retracting. Ampicillin and Cefotaxime, CBG obtained 7.15/66/-7.0, umbilical lines placed, Chest-x-ray: mild RDS, initial improvement on NCPAP but 24 hours later acute clinical deterioration with a capillary blood gas CBG: PH: 7.1/ 42/-10, CXR: Moderate cardiomegaly and bilateral pulmonary venous congestion with good lung expansion. Echocardiogram showed: Moderate size PDA with all left to right shunting, mild mitral regurgitation, flattened IVS motion RV hypertension, Moderate to severe diminished left ventricular systolic shortening, EF: 37% with LV dilation and normal origin of both coronary arteries. The Patient was intubated and started on SIMV. Milrinone and Epinephrine drips were started. Surfactant given, despite been able to wean the FiO2, the respiratory distress continued to worsen, an EKG was done and showed: Figure # 1: Normal sinus rhythm, right axis deviation, pathologic Q waves 1 and AVL suggesting a lateral wall infarct. Cardiology was consulted, and the baby was taken for a Cardiac catheterization that showed: Normal coronary artery anatomy with multiple filling defects on the left circumflex artery due to multiple microthrombi, intracoronary nitroglycerin was given at 2 mcg/kg/dose followed by TPA at 0.1 mg/kg/dose this resulted in improved flow on the left circumflex artery. Post cardiac catheterization she was started heparin 10 units/kg/h and was transferred back to the neonatal intensive care NICU, the baby was extubated 7 days later and gradually weaned off epinephrine and transitioned from milrinone to enalapril, low dose heparin was maintained for 48 hours. The Echocardiogram done four days after the cardiac catheterization demonstrated normal Doppler color flow in the left main, coronary artery and Left anterior descending and, proximal portion of the circumflex, right ventricular systolic was normal, left ventricular ejection fraction: bullet: 50% and qualitatively normal systolic shortening fraction. Twenty days later showed: Patent foramen ovale, PFO, normal Right ventricular size and qualitatively normal systolic shortening, Normal LV size and qualitatively normal systolic shortening, Left ventricular Ejection fraction: bullet: 61%, Biplane: 58%, Right ventricular ejection fraction: within normal limits and at the same time the EKG showed: (Figure 2) Normal sinus rhythm, Q wave resolved, mild ST elevation inferior leads. The troponin and lactate that were initially elevated were back to normal and the baby clinically was doing well, ready to go home.

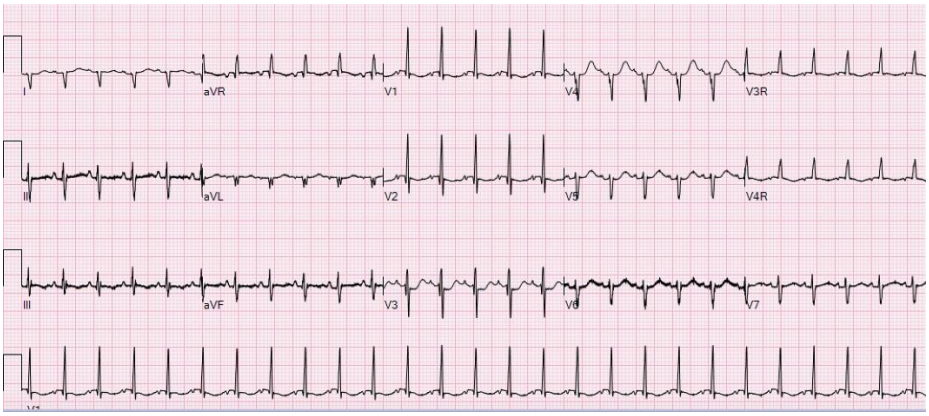

Figure 1: Pre- treatment: EKG shows pathologic Q waves in I and AVL lateral infarct.

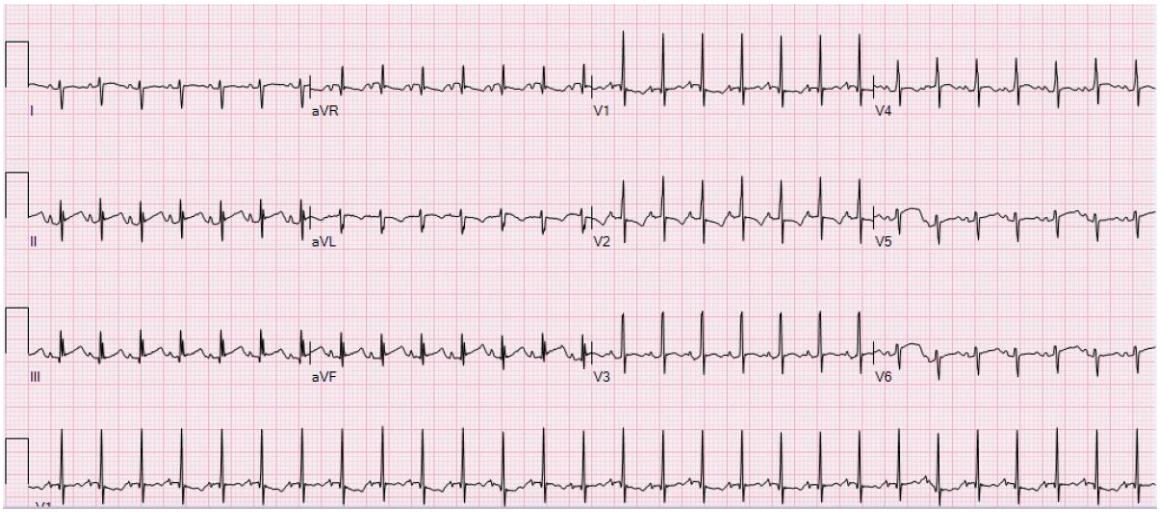

Figure 2: POST treatment: EKG has normalized Q waves no present anymore in 1 and AVL.

Discussion

Myocardial infarction in premature babies is a recognized uncommon entity, but the incidence and broad spectrum of the disease is unknown and likely underestimated due to limited reporting [6,9,18]. The challenges involve clinical diagnosis which masquerades in the early phase as non-specific symptoms and signs that are commonly found in a large variety of neonatal disorders [7]. One the most common causes in patients with structurally normal hearts are thromboembolism: secondary to placement of an umbilical venous catheter and thrombus formation in the ductus venosus or in the umbilical vein followed by paradoxical thromboembolism [2,13,16] sometimes associated to delayed cord clamping , milking of the cord, polycythemia, genetic hyper-coagulable states [5,8], or abnormal vessel communications or thrombi in the placenta that in this case were ruled out by clinical testing and pathologic examination of the placenta [9]. The most common congenital anomalies associated with myocardial infarction are aortic stenosis or atresia, hypoplastic left heart syndrome, pulmonary atresia, total anomalous pulmonary venous return, stenosis of the coronary ostium and

anomalous left coronary artery [15], ruled out in this case by echocardiography. Ductal steal phenomenon secondary to Persistent Ductus Arteriosus (PDA) has been described in the past with ST segment depressions on the EKG in babies with RDS and PDA that disappeared after surgical ligation [4]. Recent transthoracic Doppler study showed decreased left anterior descending coronary artery flow as a result of PDA; coronary artery perfusion improved after coil closure of the PDA [3]. We speculate that in our patient a PDA-associated ductal steal phenomenon in diastole probably led to decrease coronary perfusion and the slow flow during that period led to thrombi formation in the left circumflex artery that caused, severe LV dysfunction that was recognized early and successfully treated. Thrombolysis was used in this case intracoronary due to the high risk of intracranial bleeding in a premature baby with good results.

Conclusion

Acute myocardial infarction in the neonatal period in premature babies with respiratory distress syndrome is exceptional and represents a considerable diagnostic and therapeutic challenge for neonatologists and pediatric cardiologists. Early diagnosis via electrocardiography, echocardiography and cardiac catheterization is of upmost importance for adequate treatment and to promote coronary reperfusion because these patients often lack adequate collateral circulation. Prompt treatment could avoid the irreversible myocardial necrosis that culminates in death or the best of scenarios in heart transplantation [10,14,19].

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