Myocardial Infarction in Newborn Infant: A Case Report

Khalid M. Al Harbi*, CABP

Department of Pediatrics, King Fahd Hospital of the University
Al Khobar, Saudi Arabia
drkharbi@yahoo.com

Abstract. A neonate with Ellis-van Creveld Syndrome who presented with circulatory collapse was found to have myocardial infarction caused most probably by air embolism to coronary artery. Milking of the umbilical cord after delivery causing paradoxical air embolism through the atrial septal defect is the most likely mechanism of coronary occlusion.

Keywords: Myocardial infarction, Coronary artery air embolism.

Introduction

Myocardial infarction is rare in the neonate period with a high mortality rate of 80%. This study describes a newborn that developed myocardial infarction soon after birth and the possible cause, in addition to a literature review pertaining to the case.

Case Report

A newborn male infant weighing 2.23 kg was born at term by spontaneous vaginal delivery. Apgar scores were 9 and 10 at one- and five-minutes, respectively. The patient had dysmorphic features: short sternum, short upper and lower limbs, short trunk, polydactyly, and hypoplastic nails. These features were consistent with the diagnosis of Ellis-van Creveld Syndrome. At 2 hours of age, the infant became tachypneic with a respiratory rate above 60 per

*To whom all correspondence & reprint requests: P.O. Box 4007 MBC (J-16), Jeddah 21589 Saudi Arabia.
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minute. An inspired oxygen concentration of 10 liters per minute via face mask was required to maintain oxygen saturation at 92%. Septic work up screen was completed and chest roentgenogram showed normal heart size and normal pulmonary vascular markings. The patient was started on ampicillin and gentamicin as an empiric antibiotic coverage to rule out sepsis.

At 10 hours of age, the patient started to become duskier requiring more oxygen. Peripheral pulses were weak, capillary refill was delayed > 2 seconds, there was tachycardia of 178 beats/min, active right ventricle, and, on auscultation, a grade 2/6 systolic murmur was heard at the left-sternal edge. The liver was palpable 3 cm below the right-costal margin. A repeated chest roentgenogram showed moderate cardiomegaly and pulmonary plethora. Profound metabolic acidosis was evident in arterial blood gas with a pH of 6.9. The patient at that time was put on mechanical ventilation for further support.

At 18 hours of age, the patient was more stable with improved capillary refill. An electrocardiogram (ECG) obtained at that time showed normal sinus rhythm at a rate of 120 beats/min, QRS axis of - 20°, ST segment elevation in leads II, III, aVF, V1, V2, V3, V4 (Fig. 1). An echocardiogram showed a large atrial septal defect, almost single atrium with very poor left ventricular function, fractional shortening was 15% (normal 28 - 40). Hypokinesia of inferolateral wall of the left ventricle was recorded along with good right ventricular function with mild pericardial effusion.

Plasma levels of troponin, creatinine kinase M B fraction at 20 h were grossly elevated 6.72 µg/l (normal range: 0 - 0.5 µg/l) and 79.7 iu/l (normal range: 0.3 - 0.6 iu/l), creatinine kinase 423 iu/l (normal range: 23 - 232 iu/l), lactate

![Fig. 1. Electrocardiogram of newborn infant with myocardial infarction.](image)
dehydrogenase 665 µ/l (normal range 100 - 190 µ/l). Protein C, Protein S, and antithrombin III were all normal. Cardiac catheterization was performed to exclude anomalous origin of the left coronary artery and it was normal.

The patient was started on Lasix 2 mg i.v. Q 12 h, Digoxin 5 mcg i.v. Q 12 h, Heparin 100 u/l/kg loading dose followed by maintenance 20 u/kg/h. General condition began to improve gradually and on the second day the patient had good pulses in his four limbs with a heart rate of 130 beat/min. Electrocardiogram showed normalization of ST segment (Fig. 2). The patient's general condition continued to improve and he was discharged home after 15 days of admission.

**Discussion**

Early circulatory collapse of cardiac origin is usually due to critical left heart obstruction, obstructed pulmonary venous return, or more rarely, myocardial ischemia. The latter of these can result from hypoperfusion of the myocardium when demand outstrips supply, such as in perinatal hypoxia, or from embolic coronary occlusion. The myocardial infarction diagnosis was made on the basis of indirect evidence from echocardiography (ECG): clinical presentation, ECG findings, and elevated cardiac enzyme.
In the neonate, various sites of primary thrombus formation leading to paradoxical embolism have been implicated, including the ductus venosus\[1\], umbilical vessels\[2\], and renal veins\[3\]. Other causes of occlusion of the coronary artery include; anomalous origin, stenosis of the ostia, and intrinsic abnormality of the coronary artery wall. In this case, cardiac catheterization showed normal coronary arteries origin. Contributing factors to thrombosis include prothrombotic conditions such as antithrombin III deficiency, Protein S deficiency, Protein C deficiency\[4\]. This case report showed normal level of antithrombin III, protein S and protein C. Some cases of neonatal myocardial infarction have been associated with difficult delivery potentially leading to circulatory stasis and thrombosis\[5\]. After excluding the most likely causes of myocardial infarction in neonate, milking of the umbilical cord in the delivery room after the delivery of the newborn raises the possibility of an air embolism originating in the umbilical cord, then migrating to the right atrium then to the left atrium via the large atrial septal defect and eventually to the left ventricle and to the coronary artery causing transient air embolism and myocardial infarction.

Mortality from neonatal myocardial infarction is reported to be above 80\[5\]. Early detection in the reported case and appropriate management led to saving the life of our patient. Supportive measures included treatment of arrhythmias, diuretics, inotropes, and after load reducing agents. Thrombolytic agents, administered intravenously\[6\], or intracoronary\[7\], had been used for coronary artery thrombosis in two neonatal case reports and in children with Kawasaki Disease\[8\]. Alternatively, extracorporeal membrane oxygenation\[9,10\] has been successfully used to support the circulation. In those cases, however, the coronaries were actually patent, permitting perfusion of the myocardium, which contrasts with the findings in this case report.

In summary, neonatal myocardial infarction may result from embolic occlusion of the left main coronary artery. This may lead to circulatory collapse in the newborn period, which can be difficult to differentiate from critical left heart disease. Prompt cardiac investigation is indicated to confirm the diagnosis and to guide management which can improve the outcome of myocardial infarction.

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References


احتشاء عضلة القلب في طفل وليد

خالد متحم محمد الحربي
قسم طب الأطفال، أمراض القلب
مستشفى الملك فيصل التخصصي ومركز الأبحاث
جدة - المملكة العربية السعودية

المستخلص. تستعرض حالة احتشاء عضلة القلب في طفل وليد عمره يوم واحد، ويعاني من متلازمة إيلبيس فان كريفلد. وتعتقد أن عصر الحبل السري، وبالتالي الضغط على الأوردة السرية أدى إلى اندفاع صمامات هجائية دخلت إلى الدورة الدموية ومن ثم عبرت الشبكة بين الأذينتين إلى الأذينية اليسرى ثم البطين الأيسر، ومنه إلى السريان الإكليلية.