Pediatr Cardiol (2013) 34:1868–1870
DOI 10.1007/s00246-012-0417-2

CASE REPORT

Successful Treatment of a Newborn With Acute Myocardial Infarction on the First Day of Life

S. Cesna · A. Eicken · H. Juenger · J. Hess

Received: 11 March 2012 / Accepted: 21 June 2012 / Published online: 21 July 2012

Abstract Cardiogenic shock occurring after acute neonatal myocardial infarction (MI) due to coronary artery thrombosis is very rarely encountered. Acute neonatal MI typically presents suddenly with usually a fatal outcome. Treatment options in patients with this condition are limited. There are previous case reports in the literature advocating the use of extracorporeal membrane oxygenation for hemodynamic support. In this report, we present a newborn with severe MI secondary to thrombus formation within the left anterior descending coronary artery. There also proved to be a Factor V Leiden heterozygotic mutation. The patient initially presented with cardiogenic shock. After resuscitation and thrombolytic therapy were administered, coronary artery patency was restored resulting in myocardial revitalization and recovery of left-ventricular function within 4 weeks.

Keywords Newborn · Acute myocardial infarction · Cardiac catheterization · Thrombolytic therapy

S. Cesna (✉)
Vilnius University, Vilnius, Lithuania
e-mail: sigitas.cesna@santa.lt

A. Eicken · J. Hess
Department of Paediatric Cardiology and Congenital Heart Disease, Deutsches Herzzentrum München, Technische Universität München, 80636 Munich, Germany

H. Juenger
Department of Pediatrics, Klinikum Rechts der Isar, Technische Universität München, 81675 Munich, Germany

Introduction

Myocardial infarction (MI) in the perinatal period is rarely encountered. Acute neonatal MI usually is fatal. Perinatal or neonatal MI has received little attention in the literature. Previous reports have focused on treatment with extracorporeal membrane oxygenation (ECMO) [11, 12]. We report a patient who presented with cardiogenic shock. After resuscitation and thrombolytic therapy were administered, coronary artery patency was restored resulting in myocardial revitalization and recovery of left-ventricular function within 4 weeks.

Case Report

A newborn boy (weight 4.4 kg, length 50 cm) needed primary resuscitation immediately after birth. On admission at our centre, the ventilated patient showed severely decreased left-ventricular (LV) function (ejection fraction (EF) 33 %, fractional shortening (FS) 15 %), increased echogenity of the LV myocardium and the mitral papillary muscles, good right-ventricular function, patent ductus arteriosus with bidirectional shunt, and an open foramen ovale with a mean pressure difference of 12 mm Hg between the left and right atriums. Electrocardiogram (ECG) showed signs of acute anterolateral MI (Fig. 1). Laboratory findings were as follows: troponin I 7.1 μg/l (normal <0.032), creatine kinase 5,188 U/l (normal <100), and B-type natriuretic peptide 206 ng/l (normal <100). On cardiac catheterization, a nearly occluded (thrombus) left anterior descending coronary artery was diagnosed (Fig. 2). Through a 4F left coronary catheter, 2 mg recombinant tissue plasminogen activator (r-tPA) were injected directly into the left anterior descending coronary

(LAD) artery. On the next day, another dose of 1 mg r-tPA was repeatedly administered into the LAD artery, and the vessel showed improved patency. Because a thrombus was still visible in the LAD artery, thrombolytic therapy was continued for another 48 h (Fig. 3). On the sixth day of life, cardiac magnetic resonance imaging (cMRI) was performed in which late gadolinium enhancement was seen within the LV myocardium and the anterior papillary muscle of the mitral valve (Fig. 4). The patient was weaned from the respirator. In addition, Factor V Leiden heterozygotic mutation was diagnosed. Eight weeks later, the patient's LV function improved to nearly normal values (EF 51 %, FS 26 %).

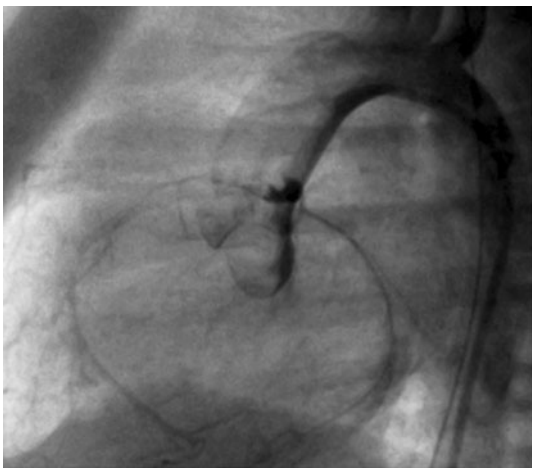

Fig. 2 Selective angiogram into the left coronary artery (*lateral view*) shows a thrombus that nearly occludes the LAD artery

Discussion

In this report, we present a newborn with acute MI due to thrombotic occlusion of the left coronary artery. Thrombolytic management resulted in revitalization of the LV myocardium. The LV function improved significantly to nearly normal values.

Neonatal MI is rarely encountered and is a potentially life-threatening condition with an approximated mortality rate of 90 % [5, 12]. MI, when seen in newborns, is associated with intrauterine asphyxia and thromboembolic coronary occlusion [2, 7]. Other possible causes for early MI are anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA), congenital coronary artery stenosis, inflammatory disease or medial calcification of the coronary arteries, Kawasaki disease, endocarditis, myocarditis, or neonatal thrombosis [3, 5–7, 11]. A paradoxical embolus usually arises from thrombotic material within the ductus venosus, umbilical cord, or renal veins [1, 2, 4]. Possible etiologies for neonatal thrombosis are antithrombin III deficiency, protein S and/or protein C deficiency, viral myocarditis, and erythroblastosis fetalis. Our patient had heterozygotic Factor V Leiden mutation. It remains unclear if the neonatal asphyxia was the cause or a symptom of the coronary arterial occlusion.

In our patient, the acute onset occurred with cardiogenic shock [9, 10]. First, ALCAPA was suspected. However, an anomalous coronary artery could not be seen on echocardiography. The diagnosis was established by cardiac

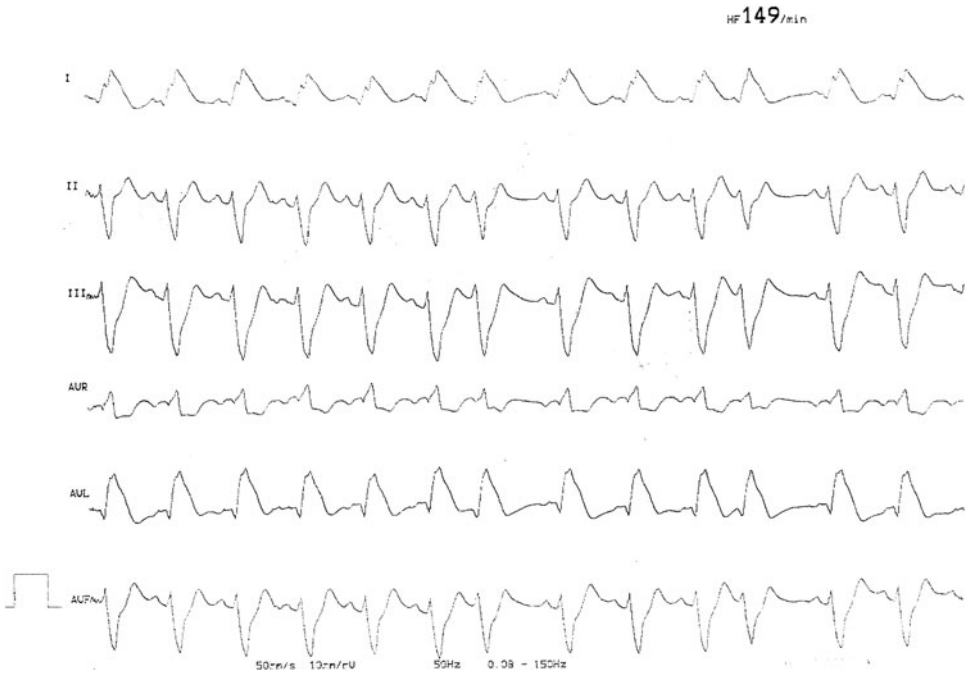

Fig. 1 First ECG in the emergency room shows signs of acute anterolateral MI

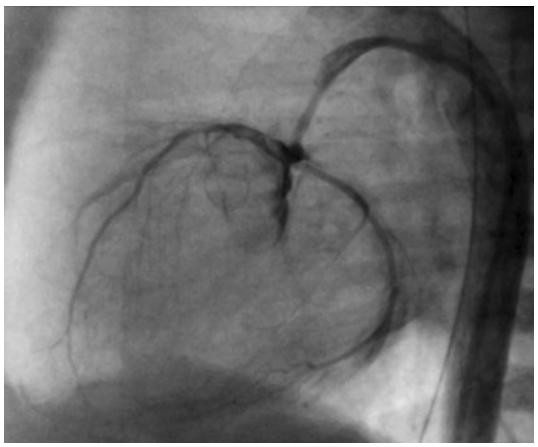

Fig. 3 Visible thrombus is seen in the LAD artery 24 h after continuous thrombolytic therapy

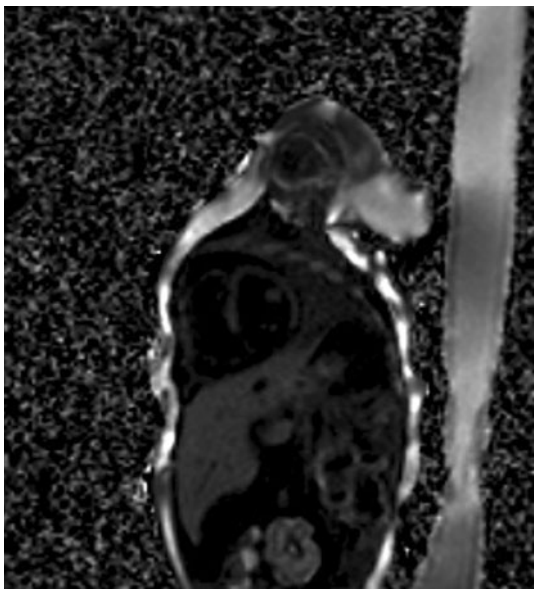

Fig. 4 On the sixth day of life, cMRI shows late enhancement within the LV and the anterior papillary muscle of the mitral valve (*short-axis lateral view*)

catheterization, which carries a significant risk in a hemodynamically instable neonate [12].

Acute MI in neonates is usually followed by cardiogenic shock. This is usually fatal. There are some reports in the recent literature on using ECMO to support the circulation during the acute and post-MI periods [11, 12]. In our patient, the initial goal of thrombolytic therapy was restoration of myocardial perfusion. After r-tPA infusion, the patient stabilized clinically without any need for catecholamine infusion or ECMO.

Global ventricular dysfunction usually occurs after acute MI. Newborns with MI due to coronary vessel occlusion are a high-risk group because usually there is no well-established coronary collateral flow to the myocardium [8, 11]. Ischemic ventricular dysfunction is a reversible physiological process and has been termed "myocardial stun" [8]. The mechanism of injury remains unclear, but with myocardial reperfusion there may be a breakdown of membrane phospholipids by way of oxygen free radicals. There are data in the literature that the stunned neonatal myocardium may have an increased potential for recovery compared with the adult myocardium [11].

In conclusion, we report the successful emergency treatment of a newborn with acute MI due to a thrombus in the LAD RTER. Thrombolytic therapy with r-tPA seemed to be the only possible treatment option in this very young and hemodynamically instable patient.

References

1. Arthur A, Cotton D, Evans R, Spencer H (1968) Myocardial infarction in a newborn infant. J Pediatr 73:110–111
2. Bernstein D, Finkbeiner WE, Soifer S et al (1986) Perinatal myocardial infarction: a case report and review of the literature. Pediatr Cardiol 6:313–317
3. Bor I (1969) Myocardial infarction and ischaemic heart disease in infants and children. Arch Dis Child 44:268–281
4. Fletcher MA, Meyer M, Kirkpatrick SE et al (1976) Myocardial infarction associated with umbilical cord hematoma. J Pediatr 89:806–807
5. Franciosi RA, Blanc WA (1968) Myocardial infarcts in infants and children. I. A necropsy study in congenital heart disease. J Pediatr 73:309–319
6. Gault MH, Usher R (1960) Coronary thrombosis with myocardial infarction in a newborn infant. Clinical, electrocardiographic and post-mortem findings. N Engl J Med 25:379–382
7. Kilbride H, Way GL, Merenstein GB et al (1980) Myocardial infarction in the neonate with normal heart and coronary arteries. Am J Dis Child 134:759–762
8. Kloner RA (1993) Does reperfusion injury exist in humans? J Am Coll Cardiol 21:537–545
9. Lucas VW Jr, Burchfield DJ, Donnelly WH Jr (1994) Multiple coronary thromboemboli and myocardial infarction in a newborn infant. J Perinatol 14:145–149
10. Murugan SJ, Gnanapragasam J, Vettukattil J (2002) Acute myocardial infarction in the neonatal period. Cardiol Young 12(4):411–413
11. Saker DM, Walsh-Sukys M, Spector M, Zahka KG (1997) Cardiac recovery and survival after neonatal myocardial infarction. Pediatr Cardiol 18:139–142
12. Tometzki AJ, Pollock JC, Wilson N et al (1996) Role of ECMO in neonatal myocardial infarction. Arch Dis Child Fetal Neonatal Ed 74:F143–F144