CASE REPORT

Paradoxical embolism causing fatal myocardial infarction in a newborn infant

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Abstract

A neonate who presented with circulatory collapse was found to have myocardial infarction caused by thrombotic occlusion of the left main coronary artery. At autopsy, a thrombus was found in the ductus venosus making paradoxical embolism through the foramen ovale the most likely mechanism of coronary occlusion.

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Case report

A female infant weighing 4.04 kg was born at term by spontaneous vaginal delivery. The delivery was complicated by shoulder dystocia. The Apgar scores were 7 and 10 at one and five minutes respectively. At 12 hours of age, the infant became cyanosed and tachypnoeic with grunting respiration. An inspired oxygen concentration of 80% was required to maintain oxygen saturation at 91%. Peripheral pulses were weak, there was a tachycardia of 180 beats/min, and, on auscultation, a grade 2/6 systolic murmur was heard at the left sternal edge. A chest radiograph showed cardiomegaly and pulmonary plethora.

Profound metabolic acidosis developed and she was therefore ventilated and started on prostaglandin E and inotropes before transfer

to our centre with a provisional diagnosis of critical left heart obstruction.

On cardiological review at 20 hours of age, the clinical findings were unchanged. The electrocardiogram (ECG) showed sinus rhythm 140 beats/min, QRS axis of 45°, Q waves and ST elevation in leads I and aVL, and T wave inversion in leads V₄₋₆ (fig 1). An echocardiogram confirmed normal cardiac connections. The left ventricle was mildly dilated with an end diastolic dimension of 26 mm (normal range 20–25 mm) and an end systolic dimension of 24 mm (normal range 15–18 mm). Left ventricular function was very poor, with a fractional shortening of 8% (normal 28–40%). The aortic valve did not open, but was of normal size at 8.9 mm (normal range 7–14 mm). The right ventricle supported the systemic circulation with right to left shunting across the arterial duct. Cardiac catheterisation was performed to exclude anomalous origin of the left coronary artery. The left coronary artery origin was patent but it then ended abruptly (fig 2) confirming left main coronary artery occlusion. The right coronary artery was normal. An infusion of tissue plasminogen activator was started, and plasma levels of cardiac specific creatinine phosphokinase and lactate dehydrogenase at 24 hours were grossly elevated: 544 units/l (normal range 5–30 units/l) and 7365 units/l (normal range 40–120 units/l) respectively. The baby's condition continued to deteriorate and she died at 36 hours of age.

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Figure 1 Electrocardiogram of newborn infant with complete left coronary artery occlusion.

Figure 2 Chest radiograph showing that the left coronary artery origin was patent but then ended abruptly. RCA, Right coronary artery; LCA, left coronary artery.

Post mortem examination showed almost a full thickness haemorrhagic infarction of the left ventricle. There was complete occlusion of the lumen of the left coronary artery by a fresh thrombus. The coronary artery itself was not stenosed and had a normal origin and wall. The ductus venosus had a thrombus attached to the intimal surface. It was not possible to obtain a prothrombotic screen from the infant because of prior administration of blood products. Both parents, however, had normal levels of protein C, protein S, and antithrombin III.

Discussion

Early circulatory collapse of cardiac origin is usually due to critical left heart obstruction, obstructed pulmonary venous return, or, more rarely, myocardial ischaemia. The last of these can result from hypoperfusion of the myocardium when demand outstrips supply, such as in perinatal hypoxia. Some previous reports have inferred myocardial infarction on the basis of indirect evidence from ECG or echocardiography. Our case provides clear evidence that myocardial infarction may result from thromboembolic coronary artery occlusion. The finding of a thrombus in the ductus venosus at post mortem examination makes paradoxical embolism from this site, through the foramen ovale, the likely mechanism of coronary occlusion. Although in situ thrombosis cannot be completely excluded, the normality of the coronary wall and lack of organisation of the thrombus provide supportive evidence of an embolic mechanism.

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 With regard to closure of the ductus

venosus, a number of mechanisms have been proposed, but thrombotic occlusion was not noted in two ultrasonographic studies.^{4,5} Other causes of occlusion of the coronary artery include anomalous origin, stenosis of the ostia, and intrinsic abnormality of the coronary artery wall. Contributing factors to thrombosis include prothrombotic conditions such as antithrombin III deficiency.⁶ Some cases of neonatal myocardial infarction have been associated with a "difficult" delivery potentially leading to circulatory stasis and thrombosis,⁷ thus the occurrence of shoulder dystocia may have been relevant in our case. Mortality from neonatal myocardial infarction is reported to be above 80%.⁷ Supportive measures include treatment of arrhythmias, diuretics, inotropes, and after-load reducing agents. Prostaglandin E1 may be used to maintain ductal patency if this is supporting the systemic circulation. Thrombolytic agents, administered intravenously⁸ or intracoronary,⁹ have been used for coronary artery thrombosis in two neonatal case reports and in children with Kawasaki disease.¹⁰ Alternatively, extracorporeal membrane oxygenation^{11,12} 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 our case.

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 guide management, but, despite supportive measures and fibrinolytic agents, mortality remains high.

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