Old Before His Time – A Case of Neonatal Myocardial Infarction

Andrea Pisesky[1], Anne I Dipchand[1], Edward Hickey[2], Emilie Jean-St-Michel[1], Aamir Jeewa[1]
1. Labatt Family Heart Centre 2. Division of Cardiovascular Surgery; The Hospital for Sick Children. Toronto, Canada
Disclosures: None

Introduction

- We present a rare case of cardiogenic shock due to neonatal myocardial infarction (MI) secondary to coronary artery occlusion.
- The need for orthotopic heart transplantation (OHT) in our case underscores the malignant nature of this pathology and the current lack of management strategies effective in altering the natural history.

Case – Presentation

- A 3.9 kg term male was born following an uneventful pregnancy and delivery.
- APGARS were 9 and 9 and he required no initial resuscitation.
- At 1 hour of life, he was tachycardic and tachypneic with severe desaturation and metabolic acidosis.
- Initial management included intubation, antibiotics, inotropes, prostaglandin and transfer to our centre.
- CXR showed normal cardiac silhouette and mild bilateral peripheral peribronchial thickening.
- ECG (Image 1) showed acute ischemia with anterolateral ST changes, lateral q waves and reciprocal changes.

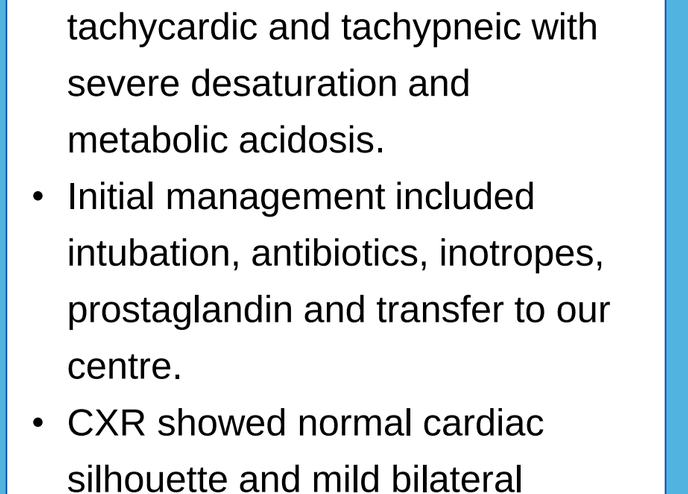

Image 1 – Admission ECG showing sinus tachycardia with ST depression in V1-V3, ST elevation and q waves in V4-V6 and reciprocal changes in the inferior leads.

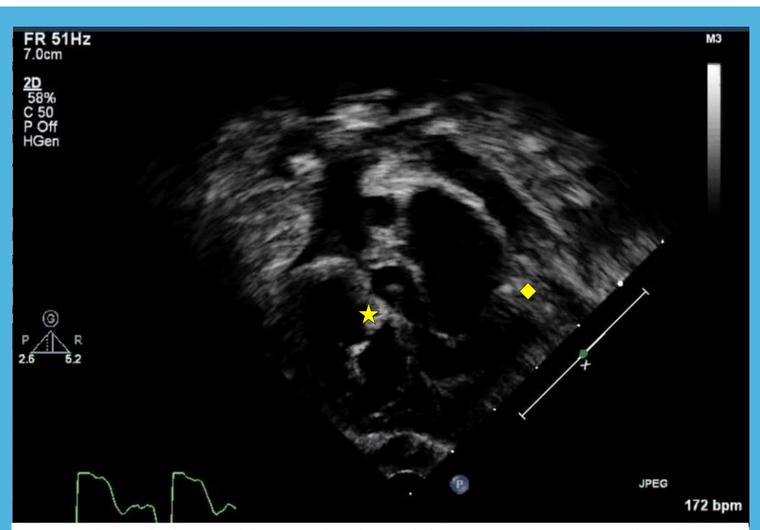

Image 2A – Subcostal long axis view of the LV outflow tract showing a hyperechoic mass, consistent with a thrombus, at the sinotubular junction and a dilated left atrium. ◇ = mitral valve annulus; ☆ = aortic valve.

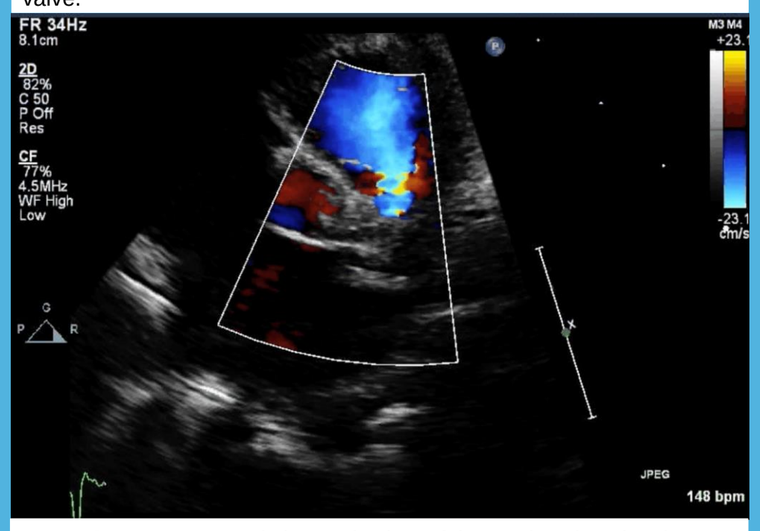

Image 2B – Parasternal short axis view of the aortic valve showing a thrombus at the origin of the left main coronary artery and no flow in the left coronary system.

Case – Management

- Echocardiogram on admission revealed normal segmental anatomy, severely reduced left ventricular (LV) function with akinesia of the anterior and lateral LV walls, preserved right ventricular (RV) function and severe mitral regurgitation (MR).
- There was a large thrombus in the aortic root, extending into the left main coronary artery with no demonstrable flow in the left coronary system (Image 2A and 2B).
- No other thrombi were found after screening with ultrasound.
- Thrombophilia work-up was negative and there was no relevant family history.
- Following a normal CT head, systemic thrombolysis with Alteplase was administered at 21 hours of life.
- There were no complications following thrombolysis.

Case – Outcome

- 6 hours post-thrombolysis, there was resolution of the thrombus by imaging and improvement in the ECG abnormalities.
- There was no recovery of LV function and the infant remained on full supports with ongoing evidence of severe end organ dysfunction.
- He was listed for cardiac transplantation on day of life 17 and underwent an ABO-compatible OHT on day of life 18.
- Histopathology of the explanted heart showed an extensive transmural myocardial infarction of the anterior, lateral and anteroseptal walls of the LV and the anterior wall of the RV (Image 3).

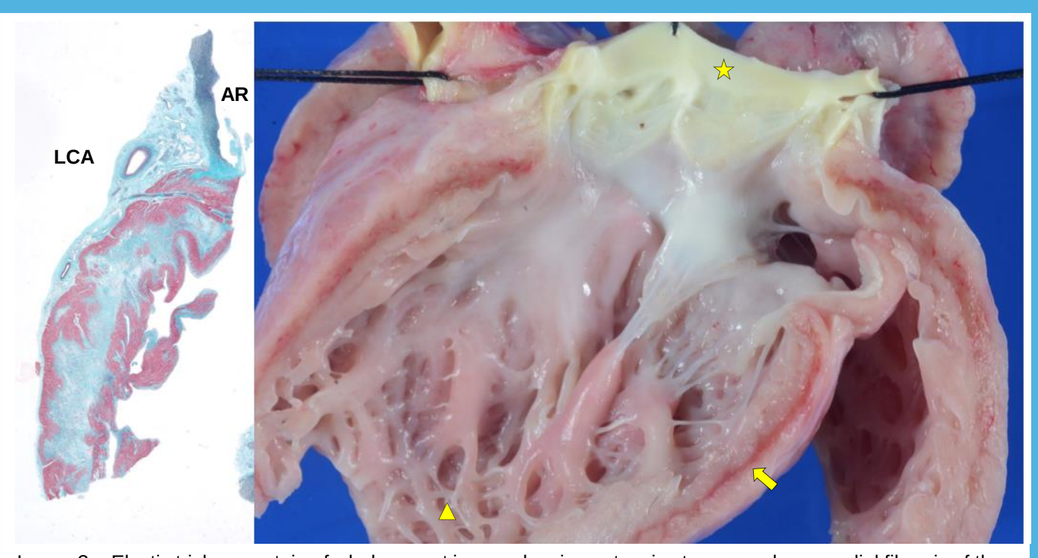

Image 3 – Elastic trichrome stain of whole mount image showing extensive transmural myocardial fibrosis of the LV outflow tract (left). Gross specimen showing a red-brown band (arrow) of vascularized fibrosis from the base to the apex of the LV lateral wall (right). There was no residual or recurrent thrombus in the aorta or proximal coronaries. LCA = left coronary artery; AR = aortic root; ☆ = aortic valve; △ = LV apex.

Discussion

- Neonatal MI should be considered in a newborn with cardiogenic shock, ischemic ECG changes, regional wall motion abnormalities and/or unexplained MR[1].
- Since first described in 1947[2], neonatal MI has been characterised by its fulminant presentation and striking mortality, reported as high as 90%[3].
- Mortality appears to be shifting with the use of aggressive targeted and supportive therapies, such as systemic and intracoronary thrombolysis and mechanical circulatory support[1].
- The evidence is limited. There is thus a need to standardise reporting and an opportunity to study the immature myocardium's response to injury, for example its regenerative capacity and mechanisms.
- Despite prompt diagnosis and management, extensive myocardial injury is possible and may warrant early consideration for OHT.

References:
1. Molkara D, Sepulveda J, Do T, Davis C, Goldstein G, Moore J, El-Said H. Tissue plasminogen activator for neonatal coronary thrombosis presenting with mitral regurgitation and impaired ventricular function. Cong Hrt Ds. 2017;12:270-274
2. Ravich R, Rosenblatt P. Myocardial infarction in the newborn infant. J of Ped. 1947;31 (3): 266-273.
3. Boulton J, Henry R, Roddick LG, Rogers D, Thompson L, Warner G. Survival after neonatal myocardial infarction. Pediatrics. 1991;88:145-50.