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Brief Report

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Author for correspondence:

O. Mingas, MD, Department of Pediatric Cardiology, Santa Cruz Hospital, Centro Hospitalar de Lisboa Ocidental, Av. Prof. Dr Reinaldo dos Santos, 2790-134 Carnaxide, Lisbon, Portugal. Tel: +351-965595763; Fax: +351 214188095. E-mail: tch.uca@hotmail.com

Spontaneous thrombosis of the arterial duct in a newborn with alloimmune thrombocytopaenia

Odete Mingas (), Natália Noronha (), Graça Sousa and Rui Anjos

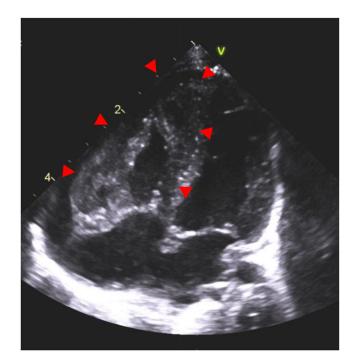
Pediatric Cardiology Department, Hospital de Santa Cruz, Centro Hospitalar de Lisboa Ocidental, Lisbon, Portugal

Abstract

We present an uncommon challenging case of spontaneous thrombosis of the arterial duct and with alloimmune thrombocytopaenia in a full-term newborn who presented with respiratory distress, hypoglycaemia dispersed petechiae on the trunk, and significant haemorrhage of the umbilical venous catheter.

Case report

A 15-day-old full-term newborn presented with respiratory distress and hypoglycaemia on the first day of life. Her mother had a background of stroke as a child and three previous abortions. The baby was admitted to the neonatal ICU and was started on non-invasive ventilation and supplemental oxygen. The chest X-ray was normal. On the following day, physical examination revealed dispersed petechiae on the trunk and significant haemorrhage of the umbilical venous catheter. Laboratory investigations showed severe thrombocytopaenia (platelet count 23×10^9 /L) and abnormal coagulation (PT 25 s, aPTT > 320 s, fibrinogen 221 mg/dl). She was started on a 3-day course of intravenous immunoglobulin. Repeat laboratory investigation 2 days later, laboratory investigation showed some improvement on the platelet count and coagulation (platelets 32×10^9 /L, PT 20 s, aPTT 72.5 s). Haematological investigation revealed class I anti-HLA antibodies, suggesting the diagnosis of alloimmune thrombocytopaenia. On her fifth day of life, she had a Paediatric Cardiology consultation due to a heart murmur, tachypnoea, and hypoxaemia. Transthoracic echocardiogram showed a patent foramen ovale with bidirectional flow, right ventricular hypertrophy (Fig 1) and a thrombus (Fig 2) in the ductus arteriosus extending to the left pulmonary artery (a) and main pulmonary artery (c) and colour flow mapping showing a reduction of flow in the left pulmonary artery (b). The peak gradient in the left pulmonary artery was 35 mmHg and the arterial duct was closed. Ventricular function was normal and there was no pericardial effusion. Transfontanellar ultrasound was normal. He was started on one heparin infusion with an improvement of the platelet count to 32×10^9 /L. There was also



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Figure 1. Transthoracic echocardiogram, apical four-chamber view, demonstrating significant right ventricular and septal hypertrophy (arrow heads).

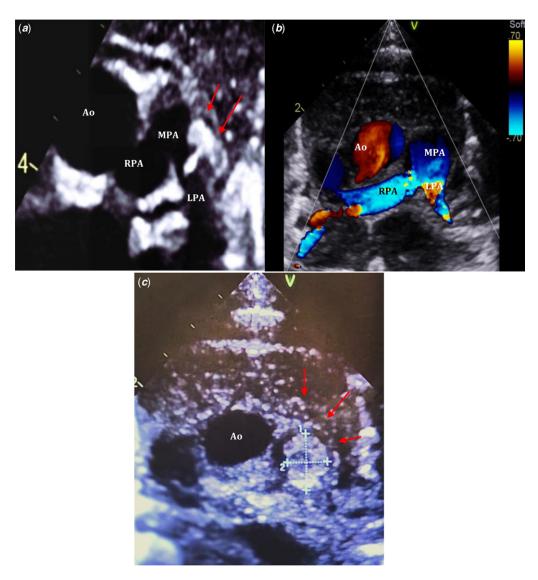


Figure 2. Transthoracic echocardiogram, parasternal short axis, demonstrating: (*a*) and (*b*) thrombus protuding to the left pulmonary artery end of the arterial duct (*c*) throumbus in the main pulmonary artey. Ao = Aorta; LPA = left pulmonary artery; MPA = main pulmonary artery; RPA = right pulmonary artery; arrows = thrombus.

progressive normalisation of the coagulation (PT 10.4 s, aPTT 28.5 s, fibrinogen 4.3 mg/dl). Repeat transthoracic echocardiogram performed on the 13th day of life showed no evidence of thrombus and a peak gradient of 12 mmHg in the left pulmonary artery. Given these findings, heparin infusion was stopped and she was started on acetylsalicylic acid (5 mg/kg/day). She was discharged 2 days later.

This case depicts an association of two diseases (neonatal alloimmune thrombocytopaenia and spontaneous thrombus of the arterial duct) or a complication of alloimmune thrombocytopaenia with thrombus of the arterial duct with deleterious effects on the coagulation profile. Neonatal alloimmune thrombocytopaenia is the most frequent cause of severe thrombocytopaenia in the full-term newborn. It is caused by immunoglobulin G class antibodies against platelet antigens present in the newborn with consequent platelet destruction.¹ Immune thrombocytopenia is linked to both bleeding and thrombosis.² Spontaneous thrombus of the arterial duct, on the other hand, is a rare condition and a

potential cause of fatal thromboembolism. The combination of these two entities may result in haemorrhage and/or thromboembolism of difficult management.²

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Conflicts of interest. None.

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