

Ductus arteriosus thrombosis extending into the pulmonary artery- case reports

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INTRODUCTION

Thrombosis of ductus arteriosus and pulmonary arteries is a rare new-born's condition. Few data concerning its pathogenesis exist; however ductal aneurysm is recognized as potential cause of thrombosis and prenatal closure. Although thrombotic risk factors may play a role, many cases occur in their absence. Currently, no specific guidelines regarding treatment are available. We describe two cases of ductus arteriosus and pulmonary arteries thrombosis diagnosed in our department.

CASE 1

Male, GA 41, emergency C-section because of cardiotocographic alterations. Apgar score 5-8. Therapeutic hypothermia for 3 days. Concomitant thrombocytopenia required platelets transfusions.

In the first day of life echocardiography showed right ventricular hypertrophy and dysfunction and signs of pulmonary hypertension. No flow was detectable in the ductus arteriosus.

At second check, a thrombus (6 x 9 mm) within the ductus was clearly visible, extending into the pulmonary artery, just before the origin of left branch, without significant Doppler flow changes.



Pulmonary artery thrombus of 6x9 mm, just before left branch's origin.

Since he was clinically stable, no urgent invasive intervention was necessary: unfractionated heparin was started. The thrombus decreased in size and, after 10 days, low molecular weight heparin replaced unfractionated heparin. Thrombophilia screening resulted negative. The patient continued therapeutic low molecular weight heparin administrations for other 2 months, when he switched to aspirin 5 mg/Kg per day. There were no symptoms and no irregular growth. A calcific thrombus attached to the wall of the pulmonary artery persisted.

CASE 2

Male, GA 35, Apgar score 7 at 1' and 8 at 5'.

Neonatal course was uncomplicated.

At 5 days of life, physical examination revealed a systolic murmur.

Echocardiography showed the presence of ostium secundum atrial defect and thrombotic obliteration of the ductus arteriosus extended into the pulmonary trunk, at the origin of the left pulmonary artery, its size was 3,5x2,9 mm.



Pulmonary artery thrombus of 3,5 x 2,9 mm, just before left branch's origin.

Right ventricle was mildly enlarged and hypertrophic; systolic function was preserved and no indirect signs of pulmonary artery hypertension were detected. The patient was completely asymptomatic without signs of respiratory or hemodynamic failure. Due to hemodynamic stability and major risk of bleeding of late preterm condition, we started prophylactic dose of low molecular weight heparin. Thrombophilia screening was negative. The following echocardiographic evaluations documented a mild reduction on thrombotic dimensions and development to calcific thrombus attached to the wall of the pulmonary artery.

CONCLUSIONS

Patients affected by ductus arteriosus thrombosis extended to pulmonary arteries may present with symptoms of hemodynamic decompensation, likely due to intrauterine ductus arteriosus closure as possible cause of perinatal asphyxia, or may be totally asymptomatic. Many factors, still unknown, may interact in its pathogenesis. In both cases, the thrombus dimensions did not grow thanks to the use of heparin. As most cases may be asymptomatic, this condition may frequently remain underdiagnosed.