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# Neonatal Pneumonia with Heart Failure: Beware of ALCAPA

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### **Clinical Image Description**

Anomalous origin of the Left Coronary Artery (ALCAPA) can be revealed in the early infancy through signs of decreased perfusion or sweating and poor feeding. Unfortunately, pain or distress can be misdiagnosed as food intolerance or gaseous colic. Only early diagnosis and surgery to restore two normally arising coronary arteries can be life-saving, possibly leading to progressive myocardial recovery1. Sometimes, ALCAPA can be precipitated by intercurrent airway infection: a viral or bacterial lung infection may coexist thus disclosing the clinical picture of heart failure. Bronchiolitis and acute airway infection can have a catastrophic effect on the underlying cardiovascular condition: the infection per se or some treatments (as inhaled epinephrine)

can be very detrimental for the baby2-5. However, severe lung infections revealing LCA origin anomaly during the neonatal period have been reported unfrequently.

1)A 20-day male was admitted having signs of respiratory infection (leukocytosis, polypnea and poor feeding): the clinical conditions improved while on antibiotic treatment, though both sustained polypnea and abnormal chest X-ray required a chest CT scan (Figure 1). During the diagnostic work-up a newonset systolic murmur prompted a pediatric cardiology assessment, disclosing marked left heart chambers enlargement and moderate-severe mitral regurgitation. LVEF was 45% and Tnl level was 0.743. On Doppler examination, a reverse flow pattern was evidenced in the descending LCA. The baby was re-



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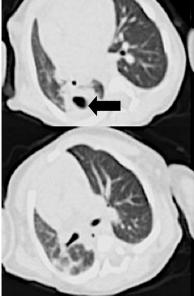
ferred to PICU, and vasodilator/diuretic treatment started. The anomalous origin of the LCA from the pulmonary trunk was evidenced by a contrast-coronary CT (Figure 1a,b), which was performed under dexmedetomidine infusion. An escavative lesion (pneumatocele) was evidenced in the right basal lung, together with a venous malformation (right pulmonary artery agenesia, right lung hypoplasia, bronchial arteries enlargement), possibly a concause of the severe lung disease. The baby was referred for LCA reimplantation.

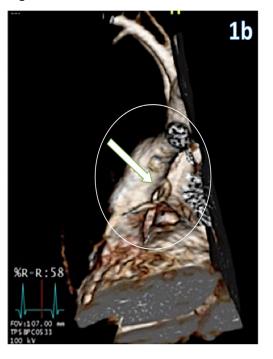
2)A 22-day male was admitted to the NICU from the neonatal ward due to worsening clinical status, possibly after an aspiration episode. He was born at 32 wks gestation, BW 1650 gr. A bacterial lung superinfection was hypothesized. Over the following 12 hours, the respiratory and clinical conditions markedly deteriorated, the baby was intubated and high ventilatory setting was required to achieve oxygenation. His chest film whitened with poor peripheral perfusion (Figure 2), tachycar-

dia and slight hypotension (195 bpm, BP 48/28). Both 10 mL/kg 5%albumin and light analgosedation achieved HR reduction until 140-145 bpm. Ischemic signs were present (TnI 1.250). A quick US assessment evidenced enlarged left cardiac chambers and papillary muscles involvement. Dobutamine and then milrinone infusion were introduced and the baby was referred to the Cardiac Surgery unit. The eventual diagnosis was ALCAPA.

In the neonatal period, in the presence of ECG and heart ischemia markers, differential diagnosis should be done between ALCAPA and myocarditis/cardiomyopathy, while the lung infection severity may hide or delay the underlying diagnosis; however, even if the echocardiographic evidence of anomalous LCA origin may be unclear, rapidly progressing left cardiac enlargement with hypoperfusion/low cardiac output and heart ischemia should arise the suspicion of LCA anomaly- requiring timely surgical advice - even if pneumonia has been previously diagnosed.







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