WHEN LESS IS MORE: THE DELICATE ART OF PATENT DUCTUS ARTERIOSUS STENTING

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A 3kg newborn with antenatal diagnosis of tricuspid atresia, hypoplastic right ventricle, transposed great arteries, subpulmonary VSD and severe valvar and subvalvar stenosis was commenced on Prostaglandin E1 infusion soon after birth. Postnatal echocardiogram confirmed the diagnoses. CT showed a right aortic arch with mirror image branching and a tortuous patent ductus arteriosus (PDA) from the left subclavian artery inserting to the proximal left pulmonary artery (PA) (Figure). Attempts at weaning Prostaglandin E1 resulted significant desaturation to low 50's. MDT Consensus was for PDA stenting.

From initial angiography, the proximal LSCA measured 2.6mm, PDA measured 3 mm with constriction at mid-point. Via a 4 French Flexor sheath, a Cobra Glide catheter was advanced to engage the PDA. A 0.014-inch Choice PT Extra Support (Boston Scientific, Miami, FL, USA) guide wire was gently steered across the PDA and anchored in the right ventricle (RV). Based on angiographic measurements with guidewire across PDA (AP 20mm; LAO cranial 12mm), a 4x15mm length Integrity stent was selected. The stent was expanded to10 atmosphere (stent diameter 4mm) in the PDA. Repeat angiography showed mid-stent waisting (figure 2b), hence further stent inflation to 18atm (stent diameter 4.4mm) was performed. Angiography post second stent expansion showed persistent mild mid stent narrowing and slight distortion of the proximal left subclavian artery (LSCA), related to the stent displacing the vessel downwards. Saturations were in the 80's in 30% oxygen. The result was accepted and the balloon catheter and coronary wire was retrieved. Post extubation, saturation was in the mid 70's in air. Heparin infusion was initiated to maintain stent patency. Two days post procedure, the patient started to have intermittent desaturation to 50's, responding to usual management for acute cyanotic spells. CT (Figure 2) showed more significant narrowing of the proximal LSCA, distorted by the PDA stent. The patient underwent removal of PDA stent and 3.5mm BT shunt insertion between the innominate artery and main PA.

Lessons learnt: Considering the diameter of the proximal LSCA, a smaller diameter PDA stent may be sufficient (3.5mm), particularly in the context of dual pulmonary blood supply. Additionally, the second higher pressure inflation caused a 'diabolo' shape of the stent by increasing the proximal and distal diameter but not the mid stent stenosis, thereby exacerbating the distortion of proximal LSCA. Thirdly, access into PDA should be carefully considered in relation to the guidewire course and direction of blood supply. In this case, both femoral and left axillary access was favourable for engaging the duct. Positioning of stent over a retrograde femoral wire course is more likely to compromise the proximal LSCA in order to fully cover the aortic end of the PDA. In contrast, a guide wire course from left axillary access may have been better to avoid distorting the vessel supplying the duct. Finally, in the case of a long PDA arising from the subclavian artery, two overlapping shorter stents may be more

flexible and provide better conformability to the ductal curvature (especially once guidewire has been removed) compared to a single long stent.

Video 1: Angiographic appearance of PDA and left subclavian artery distortion post PDA stent and mid stent stenosis.

Figure 2: (a,b) CT coronal view showing tortuous PDA originating from left subclavian artery (LSCA) and inserting into proximal left pulmonary artery (LPA). LCCA, Left common carotid artery; RAA, right aortic arch. (c,d) CT Post stent insertion showed distortion of the proximal LSCA (star) and mid stent stenosis (arrow). LPA, left pulmonary artery; RPA, right pulmonary artery.

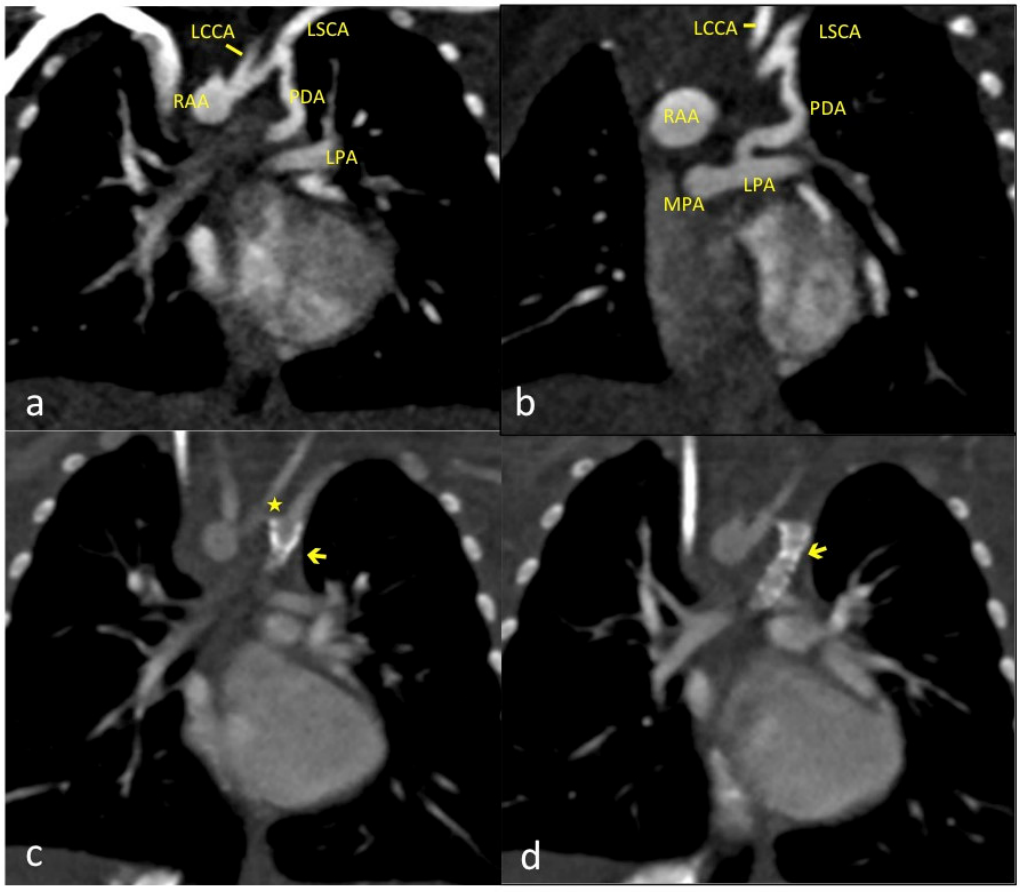

Figure 2