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Acute modified Blalock-Taussing shunt obstruction successfully treated with urokinase and heparin

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Abstract

Acute modified Blalock-Tuassig shunt obstruction due to thrombosis may be life-threatening. We report non-invasive relief of shunt obstruction with thrombolysis and heparinisation, a potentially life-saving intervention that is applicable in all settings, including outside tertiary paediatric cardiology centres.

MeSH: Abnormalities, Multiple/*surgery/ultrasonography, Constriction, Pathologic/ultrasonography, Echocardiography, Doppler, Color/*methods, Heart Defects, Congenital/*surgery/ultrasonography, Pulmonary Circulation

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Introduction

The modified Blalock-Tuassig shunt continues to be widely used as a palliative procedure, and allows staging for further cardiac surgery by controlling pulmonary blood flow.[1] Shunt morbidity is not insignificant[2,3] and shunt obstruction due to thrombosis, stenosis or kinking may be life threatening due to hypoxia. Various modalities have been employed in relieving such obstruction but the options are severely limited in settings outside a tertiary center. We report an acutely obstructed shunt that was relieved in an exclusively non-interventional, pharmacological way, a viable alternative in virtually all settings.

Patient

Our patient (female) was born in mid-2000 with a birth weight of 2.4 kilograms. She was noted to be cyanosed a few hours after birth and an echocardiogram revealed situs solitus, concordant atrioventricular connections, discordant ventriculoarterial connections, a large ventricular septal defect, pulmonary stenosis and a patent arterial duct. A 4 millimetre Gore-Tex right modified Blalock-Taussig shunt was implanted in the early neonatal period at a tertiary center and the duct was closed with a ligaclip.

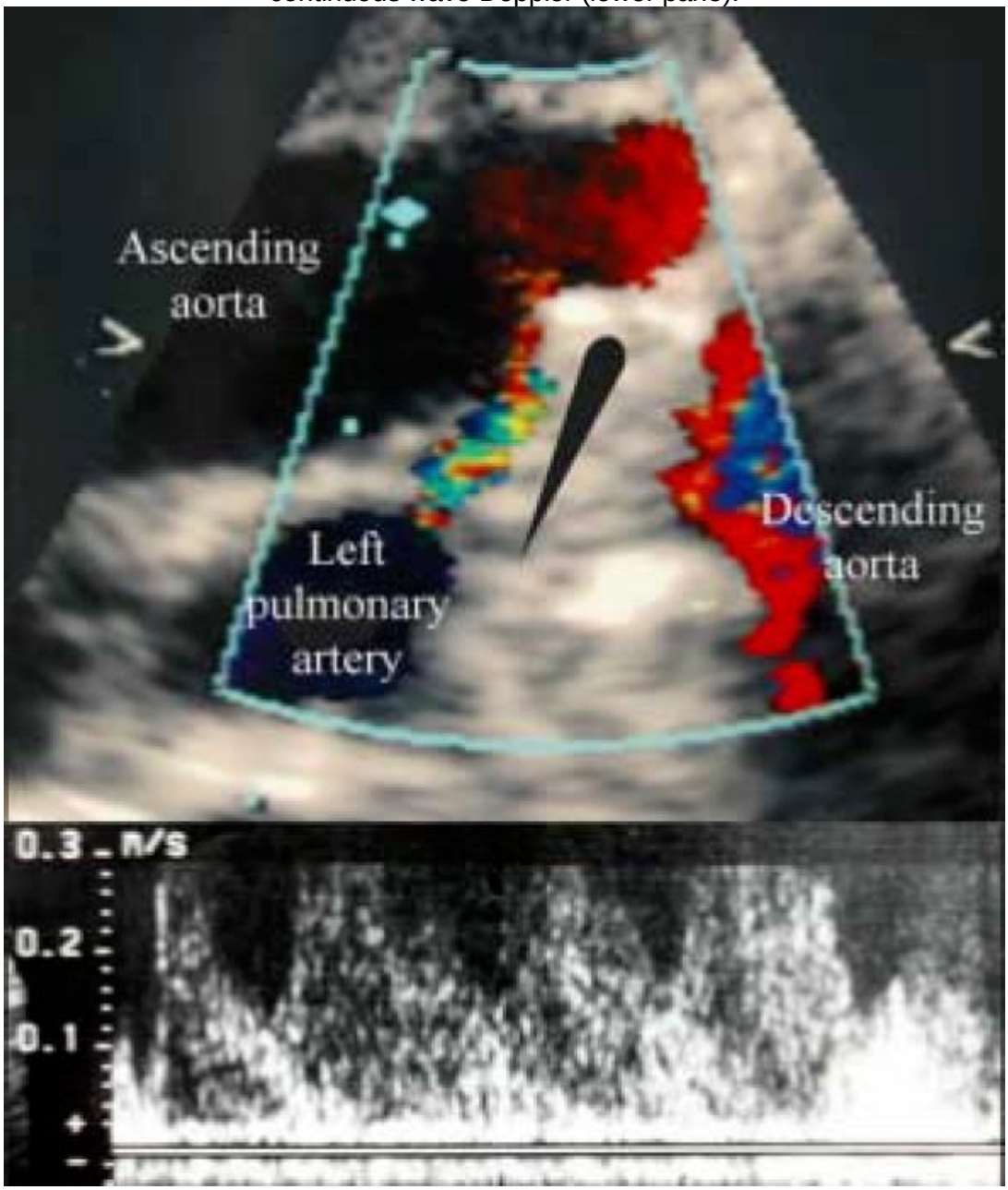
She remained well after surgery with oxygen saturations in the high 70s to mid 80s. Her only problem was failure to thrive (growing below and parallel to the third centile). She had been started on aspirin post-operatively, but this was stopped by the parents.

At the age of three years, she presented acutely, deeply cyanosed and tachypnoeic (respiratory rate 46/minute). The shunt murmur was not audible and there was a faint systolic murmur at the upper left sternal edge. Echocardiography was performed and the shunt could not be visualized at all. Colour doppler showed some forward flow across the right ventricular outflow, with good ventricular function.

The provisional diagnosis was acute shunt obstruction. The clinical condition deteriorated dramatically. She become confused, deeply cyanosed with saturations in the high 30s and bradycardic. She was given pulmonary embolism loading doses of urokinase (4400 units per kilogram intravenous bolus) and heparin (75 units per kilogram intravenous bolus), followed by a maintenance doses of both (4400 units per kilogram per hour; 20 units per kilogram per hour). This, combined with a fluid bolus, led to improved saturations. Aspirin at a dose of 150 milligrams daily was also commenced. She was never acidotic and gases at this point were: pH 7.334, pCO2 29.2 millimetres of mercury, PO2 28.5 millimetres of mercury, BD –7.9, HCO3 15.7 millimoles per litre.

Oxygen saturation climbed to the 60s over the next four hours. A morphine infusion was started to calm her down as the saturations dropped to the 40s with struggling. Shunt flow became evident on echocardiography, with an unimpressive and only systolic murmur (figure 1). Heparin was continued and the urokinase was stopped. She was transferred to a tertiary center where she underwent a bi-directional Glenn procedure. She has remained well after surgery.

Figure 1 Shunt flow recommencing: colour flow Doppler (arrow - top pane) and continuous wave Doppler (lower pane).



Discussion

In suspected partial or complete shunt obstruction, echocardiography is a reliable diagnostic tool[4] but even in the absence of echocardiographic confirmation, the clinical diagnosis of acute shunt obstruction with absent shunt murmur and cyanosis is should prompt immediate action. Acute obstruction may be treated surgically,[4,5] by catheter ballooning,[6,7] with the option of simultaneous intervention on the pulmonary arteries that may also be stenosed and require balloon dilatation,[8] and also by balloon and stenting.[9] Acute shunt blockage after cardiac catheterization using thrombolytic agents has also been described.[10] Our case highlights the possibility of relieving acute shunt obstruction due to thrombosis by solely pharmacological means, using drugs that are widely available even in district general hospitals, an intervention that may be life-saving until transfer to a tertiary center is arranged. This

report also highlights the importance of low dose antiplatelet agents in the prevention of shunt obstruction.[3]

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