Pulsus Paradoxus in a Neonate with Interrupted Aortic Arch

Pulsus paradoxus is an exaggerated inspiratory fall (>10 mmHg) of systolic blood pressure (BP). It has been reported with cardiac tamponade, asthma, pericarditis, croup, hemorrhagic shock and cardiomyopathy [1-3]. We report a novel association with interrupted aortic arch [left ventricular outflow tract (LVOT) obstruction].

A full-term, 2.8 kg, 8-day-old male neonate presented with congestive cardiac failure and shock (heart rate-152/min, respiratory rate 80/min, cold/pale extremities, right upper limb BP 31/12 mmHg, chest retractions, and hepatomegaly). His femoral pulses were feeble and were disappearing during inspiration. His right brachial pulse was constant throughout respiratory cycle. His lower limb BP was unrecordable. The respiratory variation in pulses was also appreciated on pulseoximetry (Fig. 1a). His cardiac examination was unremarkable except increased precordial activity. Echocardiography and computed tomography angiography (Fig. 1b) confirmed interrupted aortic arch. With Prostaglandin E1 (PGE1) infusion and supportive care, his lower limb pulses and BP improved [45/28 mmHg (mean 34)]. The respiratory variation in pulseoximetry gradually disappeared. Disappearance of pulses during inspiration and respiratory variation in pulse waveforms was consistent with pulsus paradoxus [4,5].

The possible explanation of pulsus paradoxus in our neonate is as follows. Blood flow in descending aorta is compromised in LVOT obstruction. With onset of PDA closure, congestive cardiac failure develops and hence aortic blood flow gets further compromised. Such neonates develop respiratory distress and thus generate ‘strong negative’ intrathoracic pressure. As a result, LV diastolic filling is compromised and right ventricular (RV) filling is relatively increased. This differential ventricular filling results in increased RV end diastolic volume, which augments LV transmural pressure (LV wall stress) and further decreases LV diastolic filling [4]. Due to these reasons, aortic blood flow can decrease markedly during inspiration leading to manifestation of pulsus paradoxus. Additionally, blood is likely to shunt from left to right ventricle across VSD, which could have also contributed to development of pulsus paradoxus. Pulsus paradoxus in LVOT obstruction is likely to manifest in post-ductal circulation during impending PDA closure. Our hypothesis is strengthened by disappearance of pulsus paradoxus after PGE1 infusion. We postulate that pulsus paradoxus in postductal circulation can detect impending ductal closure, in LVOT obstruction. Whereas, differential pulses suggest LVOT obstruction, pulsus paradoxus in such setting might indicate impending ductal closure and is thus an ominous sign. This case underscores the importance of thorough clinical examination of pulses and pulse waveforms in any neonate with suspected LVOT obstruction.

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