Supraventricular Tachycardia in a Neonate Repeatedly Induced by Ectopic Ventricular Couplet During Breast-feeding

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To the Editor: A male infant weighing 3495 g was born at term by emergency section after fetal tachycardia (250 bpm) was noted. Initially well, on day 2 he had an episode of supraventricular tachycardia (SVT; 270 bpm), and was treated with adenosine. On day 3 he had further episodes, was started on propranolol 1 mg/kg TDS and transferred to our cardiac unit. Echocardiography showed patent ductus arteriosus, and structurally and functionally normal heart. Twelve-lead ECG was normal whilst ECG in SVT was consistent with atrio-ventricular re-entry tachycardia (AVRT). He continued to have episodes which were noted every time he was fed. Telemetry revealed that each episode was triggered by a ventricular ectopic couplet, whilst single ectopy did not trigger SVT. He was therefore started on flecainide 2 mg/kg TDS. His subsequent course was uneventful, and at discharge on day 8 of life SVT had not occurred for >36 h.

Supraventricular tachycardia is the most common sustained arrhythmia in the pediatric population, often requiring emergency cardiac care in neonates [1]. Management includes correction of hypoxia, acidosis and electrolyte imbalance. Vagal manoeuvres or intravenous adenosine (0.1 to 0.3 mg/kg) are used for termination of acute episodes of SVT, whereas hemodynamically unstable SVT may require synchronized electrical cardioversion [2]. Pharmacological treatment of SVT includes beta-blockers as first-line therapy; flecainide, alone or in combination, is demonstrated to be safe and effective [3].

Breast-feeding is recognised to be associated with apnea and increased vagal tone in some neonates. Swallowing induced SVT has been observed in adults [4], whilst only one previous neonatal case has been reported, however with no suggestion as to the electrical events that may initiate the SVT [5]. We describe the first case worldwide of a neonate in whom SVT occurred recurrently with breast-feeding, and was reproducibly triggered electrically by a ventricular couplet.

We suggest that increased vagal tone during swallowing plays a key-role, and speculate that the resulting negative dromotropic effect leads to spontaneous ventricular activity (couplets), which could trigger the AVRT.

Once the diagnosis was confirmed and propranolol was seen to fail, flecainide was added as it would not only slow conduction in the accessory pathway but also suppress the trigger.

Compliance with Ethical Standards

Conflict of Interest None.

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