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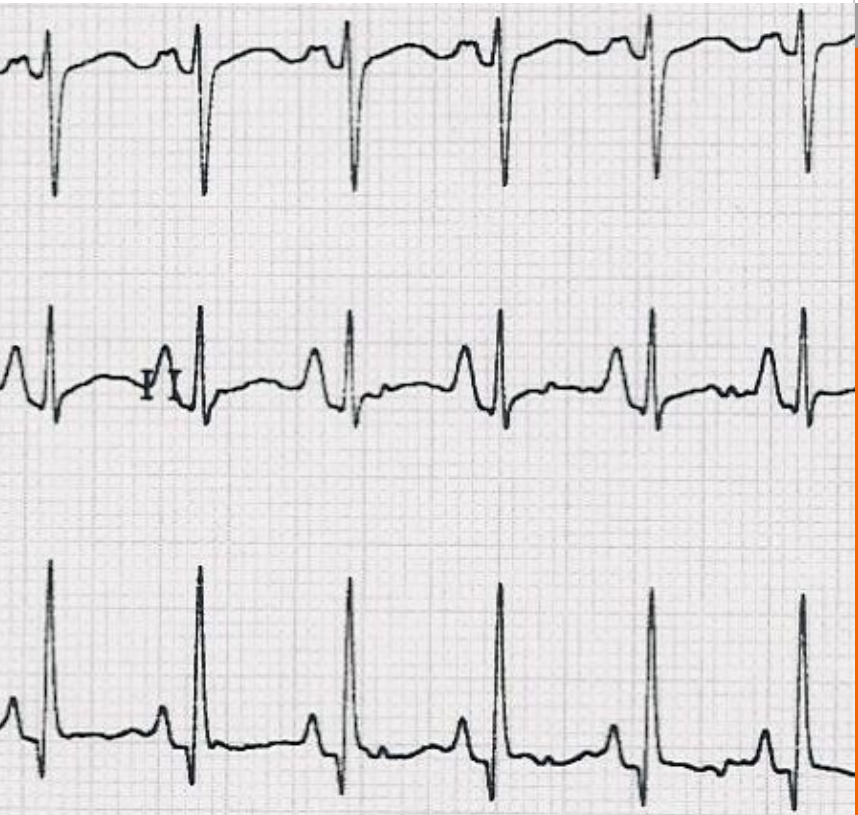
Heart failure caused by propranolol treatment in a newborn infant



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Supraventricular tachycardia developed at 34 weeks of gestation after an uneventful pregnancy. Fetal echocardiogram showed a structurally normal heart and normal ventricular function. There were no signs of hydrops except for minimal pericardial and pleural effusions. The mother was started on digoxin with conversion of the tachycardia into a normal sinus rhythm. Digoxin therapy was continued until delivery.

A baby boy was delivered by Cesarean section at 38 weeks of gestation with Apgar scores of 8, 8, and 8 at 1, 5, and 10 minutes, respectively, and a birthweight of 3150 g (P 25-50). The baby was in sinus rhythm with a heart rate of 120 to 130 per minute. He developed respiratory distress and was admitted to the neonatal unit with an FiO_2 of 60%. At the time of admission he was tachypnoeic (80 breaths per minute), had sternal retractions, nasal flaring and grunting. Chest X-ray showed a cardiothoracic index of 0.55 and hyperinflated lungs compatible with wet lungs. The baby improved rapidly and supplemental oxygen was reduced to 25% by the end of the first day of life. Digoxin concentration in cord blood (0.5 nmol/l) was below the therapeutic level.

On the first day of life cardiac assessment was performed. Auscultation of the heart was normal. ECG (Fig. 1) showed a normal sinus rhythm with a heart rate of 130-140 per minute, tall P waves suggesting atrial hypertrophy, and no evidence of Wolff-Parkinson-

White syndrome; echocardiography showed a 2 mm persistent arterial duct with bidirectional shunting confirming pulmonary hypertension, patent foramen ovale with left-to-right shunt, and an otherwise structurally normal heart. Both atria were enlarged. The measurement of systolic ventricular function was normal but there was mild diastolic dysfunction and ventricular filling appeared to be slightly impaired.

After evaluation of the situation with the pediatric cardiologists, it was decided to put the baby on propranolol, which was started on the first day of life at a dose of 1 mg/kg/day and increased gradually up to 3 mg/kg/day on day three. The baby never showed supraventricular tachycardia but his heart rate slowed to about 90-100 per minute. The baby was noted to be extremely quiet and feeding poorly.

Following the initial improvement on the first day of life, the baby deteriorated steadily and developed signs of heart failure. On the fourth day of life, he presented suddenly with marked tachypnoea of 120 breaths per minute. He was hypercapnic, required 40% of supplemental oxygen and was placed on nasal CPAP. In addition, he had several episodes of apnea and bradycardia. Further examination revealed a pale infant with diminished peripheral perfusion; clinical signs of right-sided heart failure like edema, puffy eyelids, or enlargement of the liver were absent. Heart auscultation was still normal without gallop rhythm; blood pressure was

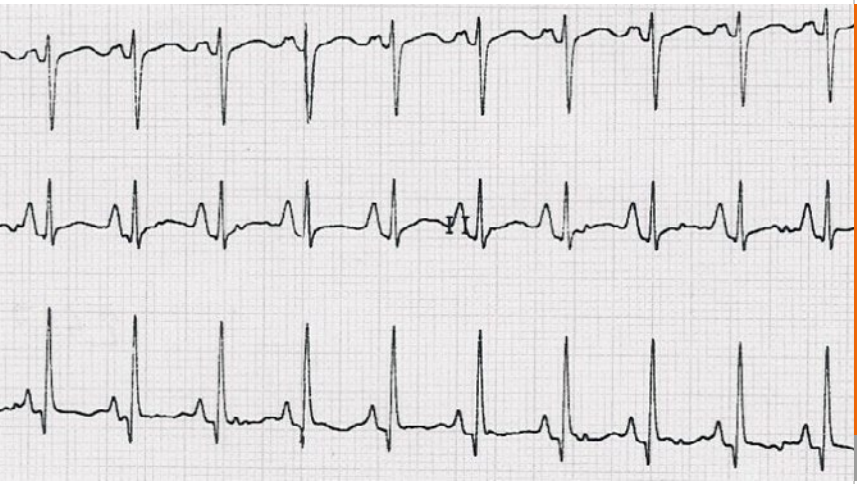

Fig. 1

ECG on the first day of life: normal sinus rhythm with tall p-waves.

normal. ECG was not performed at this stage, but on chest X-ray there was cardiomegaly (cardiothoracic index 0.65) and signs of pulmonary venous congestion. Echocardiography showed cardiomegaly with ventricular and atrial dilatation and mildly reduced ventricular contractility; the arterial duct had closed spontaneously.

Our hypothesis was that the heart failure was the consequence of propranolol treatment. The drug was discontinued and a low dose of diuretics was given. The baby improved rapidly and supplemental oxygen as well as nasal CPAP could be stopped within 12 hours. He started to feed and was increasingly active. The diuretics were stopped after 2 days of treatment. The antibiotics started at the time of clinical deterioration were stopped after obtaining negative blood cultures.

On follow-up echocardiography at the age of seven days, systolic ventricular function was normal. Propranolol was restarted at the age of ten days beginning with a small dose of 0.5 mg/kg/d and was increased daily. This time, it was well tolerated without development of heart failure. Holter ECG was normal and the baby was discharged from hospital at the age of 15 days. Ambulatory follow-up at the pediatric cardiology unit showed an uneventful course with no further episodes of tachycardia.

Intrauterine supraventricular tachycardia can lead to heart failure and hydrops within a short period of time and therefore requires rapid treatment. Among the many possibilities of prenatal treatment, digoxin given to the mother is often the first choice and was successful in the present case with rapid conversion into sinus rhythm.

It is well established that newborns having had fetal supraventricular tachycardia in utero should be treated prophylactically during the first year of life. Beta blockers are popular drugs and propranolol is usually used as the first line of treatment because it is easily applicable, usually well tolerated, and has a low incidence of complications. Side effects have been described, among them the negative inotropic and chronotropic effect which can accentuate heart failure. Other side effects are other arrhythmias, arterial hypotension, diminished peripheral perfusion, apneic episodes, bronchospasms, allergic skin reactions, and reduced activity.

Our patient presented postnatally with sinus rhythm and a normal systolic function on echocardiogram. Despite the normal shortening fraction, ejection fraction and cardiac output, echocardiogram showed diastolic dysfunction, which led to the suspicion of mild heart failure during the tachypnoeic episode. In this patient, the measurement of cardiac function was normal but the cardiac reserve was poor and this was un-

derestimated. Furthermore, the baby had respiratory distress after birth, which accentuated his cardiac overload. Propranolol was started at that stage and led to a deterioration of the cardiac function within four days. The negative chronotropic and inotropic effect of propranolol accentuated the reduction of cardiac output and led to congestive heart failure, which could not be compensated by an elevation of the heart rate as this adrenergic response was blocked by propranolol.

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