Short reports

Recovery from neonatal myocardial dysfunction after treatment of acute hypertension

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SUMMARY Echocardiography showed gross cardiac dysfunction in a neonate with hypertension secondary to renal artery thrombosis. Cardiac recovery was dramatic after control of hypertension with captopril.

Case report

A 2580 g girl was born at 37 weeks' gestation by repeat caesarean section to a 33 year gravida 2, para 1 mother. Apgar scores were seven and nine at one and five minutes. The baby developed progressive respiratory distress that required insertion of an umbilical artery catheter, through which fluids and medications were administered. Her course was complicated by a right sided pneumothorax and finally the need for assisted ventilation at 68 hours of life, after which she was referred to our neonatal intensive care unit.

On admission she had a pulse rate of 126 beats/min and central blood pressure of 86/62. On assisted ventilation, she had good air entry with normal heart sounds and no murmur or gallop. She had no oedema. Liver edge was 2 cm below the right costal margin. The right kidney was palpable. Blood count and serum electrolyte concentrations were normal. Serum creatinine concentration was 109 $\mu\text{mol/l}$. Urinalysis showed proteinuria, haemoglobinuria, and red blood cells. Chest radiograph showed cardiomegaly and pulmonary vascular congestion with the catheter tip at the ninth thoracic vertebra. A renal ultrasound scan initially yielded normal results.

Over the next 48 hours, her systolic blood pressure increased from 85 to 120 mm Hg. She developed increasing heart failure with liver enlargement up to 5 cm below the right costal margin, poor perfusion, and falling urine output. Electrocar-

diography showed ST and T wave changes consistent with ischaemia. Echocardiography at 6 days of age showed left atrial and left ventricular enlargement but no structural abnormality. Left ventricular shortening fraction was only 10%. Cardiac catheterisation confirmed poor left ventricular function with generalised hypokinesis of the left ventricle. The origin of the left coronary artery was normal. On repeat ultrasound scanning the right kidney, although of normal size, showed increased echogenicity with loss of corticomedullary differentiation and normal pyramid architecture. The right kidney was not seen on $^{99\text{m}}\text{Tc}$ -glucoheptonate scan. An aortogram showed thrombosis of the right renal artery.

The umbilical artery catheter line was then removed and the baby heparinised. Infusion of digoxin, furosemide, hydralazine, and even nitroprusside up to 3 $\mu\text{g/kg/min}$ did not affect her blood pressure or clinical condition. Response to diazoxide was transient. Her systolic blood pressure was finally controlled below 80 mm Hg with oral captopril 1 mg every eight hours, with dramatic resolution of heart failure and improved clinical condition within hours.

Repeat echocardiography (30 hours after the first echocardiogram had been taken and 24 hours after the blood pressure had been controlled) showed complete recovery of left ventricular function. Left atrial and ventricular dimensions were normal. Left ventricular shortening fraction was 37%.

Serum creatinine concentration peaked at 136 $\mu\text{mol/l}$, accompanied by mild hyponatraemia (serum sodium concentration 132 mmol/l) before control of blood pressure. Both gradually returned to normal.

Captopril was given for eight days with excellent control of blood pressure, which remained normal after treatment had been stopped. The baby thrived and was discharged at 17 days. Follow up at 2 months revealed a normotensive child with normal biochemical profile and no evidence of heart failure.

A renal scan continued to show a non-functional right kidney.

Discussion

Renal artery thrombosis with renin induced hypertension is a recognised complication of umbilical artery catheterisation.¹ Although renin concentrations were not measured, confirmed renal artery thrombosis with failure to respond to other anti-hypertensives and prompt response to captopril make renin mediated hypertension most likely.

Hypertension in neonates has been described as blood pressure greater than 90/60 mm Hg in term infants and 80/45 mm Hg in premature infants.¹ Cardiorespiratory symptoms and congestive heart failure are the most common symptoms of neonatal hypertension.² The degree of hypertension necessary to produce congestive heart failure may vary with individual neonates. The maximum pressure of 123/81 recorded in our patient was moderate. A very limited myocardial contractile reserve in response to increased afterload has been shown in newborn lambs.³⁻⁴ Inotropes have limited effects on contractility in newborn rabbits and lambs.⁵⁻⁶ Limited myocardial reserve may explain why congestive heart failure is often the dominating picture in neonatal hypertension.

Hypersensitivity to captopril, manifesting in some adults as hypotensive crisis, has not been reported in neonates.⁷

We have shown by echocardiography that ad-

equate treatment of hypertension rapidly reverses severe myocardial dysfunction caused by neonatal hypertension. We suggest that echocardiography may be helpful to document the cardiac effects of neonatal hypertension and to determine the need for and response to treatment.

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Anticonvulsant drugs, growth, and development

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SUMMARY Height and stage of puberty of 67 children with epilepsy were measured before beginning treatment with anticonvulsant drugs and annually to a maximum five years' treatment. Blood concentrations of the drugs used (phenytoin, sodium valproate, carbamazepine, ethosuximide, and phenobarbitone) were monitored throughout. No significant deviation in growth patterns was detected.

Anticonvulsant drugs administered during pregnancy have been postulated to retard fetal growth¹

and head circumference.² In vitro studies have shown that phenytoin and sodium valproate reduce the thickness and cellularity of epiphyseal and articular cartilage in rats.³ Round described a group of children with chronic epilepsy who failed to reach predicted adult height and attributed this to an early growth spurt and lower than normal growth velocity.⁴ Robinson found premature epiphyseal fusion in a group of short children with epilepsy treated medically.⁵

In a study of adults with epilepsy McGowan identified three groups who seemed to be short: (1) males with partial seizures beginning before 18 years of age, (2) females who had taken phenytoin before