Short reports

Atrial flutter in the newborn resulting from maternal lithium ingestion

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SUMMARY We report a case of isolated atrial flutter in a neonate, attributable to maternal lithium treatment, and suggest that the assessment of all infants born to mothers on lithium treatment during pregnancy should include an electrocardiogram.

Maternal lithium ingestion has been associated with various structural cardiac abnormalities in the baby.¹ As yet, however, no clear cause and effect relation has been established. Atrial flutter is an exceedingly rare arrhythmia in the newborn that is sometimes associated with structural heart disease. We report a case of isolated atrial flutter in the early days of life attributable to maternal lithium treatment.

Case report

A 29 year old primigravid mother who was physically well, suffered from a manic depressive psychosis and was maintained on lithium for the duration of her pregnancy. She took 800 mg twice a day for 6 months, increasing to 800 mg three times a day in the seventh month. Serum concentrations were 0.57 mmol (mEq)/l and 1.5 mmol (mEq)/l respectively. (Adult therapeutic range 0.6-1.2 mmol/l).

She went into spontaneous labour at term and was delivered normally of a baby girl weighing 3.2 kg, who was subsequently bottle fed. On the fifth day a tachycardia was noticed with an apical rate of 190/minute at rest. Examination showed no murmurs, a respiratory rate of 90/minute, a liver edge palpable 1 cm below the right costal margin, but no other abnormal physical signs. Her electrocardiograph (ECG) showed atrial flutter with variable 2:1 and 3:1 atrioventricular block, and an atrial rate of approximately 400/minute (Fig 1a). Chest x-ray film showed a minor degree of cardiomegaly and pulmonary venous congestion. At this time her electrolyte values were normal, with a serum lithium concentration of 0.25 mmol (mEq)/l. Forty eight hours later she reverted spontaneously to

Fig. 1a Lead II—atrial flutter with variable 2:1 and 3:1 block. Atrial rate approx 400/min.

Fig. 1b Lead I—sinus rhythm 130/min.

sinus rhythm and tachypnoea and pulmonary venous congestion resolved. Subsequent echocardiography was normal. At her last review at age 3 months she was well and in stable sinus rhythm (Fig 1b).

Discussion

A direct cause and effect relation between maternal lithium ingestion and atrial flutter is not proved but seems likely in this patient and is supported by the rapid and permanent resolution of the arrhythmia after clearance of the drug from the plasma. Clearance is known to be considerably prolonged in the neonatal period² and a concentration of 0.25 mmol/l at 5 days of age is consistent with a maternal concentration of 1.5 mmol (mEq)/l at the time of delivery.

Although the cardiovascular actions of lithium are poorly understood, the drug does participate in cation exchange across the cell membrane. This can cause intracellular hypokalaemia and extracellular hyperkalaemia through reduction of the inward potassium current during repolarisation.³ This mechanism is thought to account for T wave flattening of the ECG in adults during chronic treatment. Despite modest serum concentrations neonates may be particularly sensitive to the drug

because of relative hyperkalaemia and reduced cellular potassium content.⁴ Potassium loss from the atria has been suggested as an aetiological factor in atrial arrhythmias⁵ and may explain the development of atrial flutter in this patient. We suggest that the assessment of all infants born to mothers on lithium treatment during pregnancy should include an electrocardiogram.

References

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Lithium toxicity in a neonate

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SUMMARY Severe transplacental lithium toxicity in a neonate is described. There were gross functional lesions of the cardiovascular, renal, and neuromuscular systems with no structural abnormalities. At 1 year of age cardiovascular and renal function is normal, but there is developmental delay.

Case report

The baby's mother suffered from manic depression and had been treated with lithium for 7 years. Throughout this pregnancy (her fourth) the mother was maintained on lithium carbonate 1200 mg/day, chlorpromazine 50 mg 3 times per day and orphenadrine 50 mg 3 times per day. At 35 weeks' gestation she developed polyhydramnios. Two weeks later she developed signs of acute lithium toxicity—serum lithium 2.6 mmol (mEq)/l, sodium 133 mmol (mEq)/l, urea 11 mmol/l (66 mg/100 ml), creatinine 159 mmol/l (1.8 mg/100 ml)—and all drugs were stopped. The following day she went into labour and delivered a girl, weighing 2.78 kg.

The baby's Apgar score was 2 at 1 minute. Resuscitation resulted in spontaneous respiration by 15 minutes. At 3 hours of age a severe apnoeic episode occurred, necessitating continuous assisted ventilation and at this stage the infant was transferred to the regional cardiothoracic unit. On arrival she was shocked with an aortic blood pressure of 35/25 mm Hg and central venous pressure of 10 mm Hg.

Heart sounds were normal with a soft parasternal mid systolic murmur. Hepatomegaly was present and generalised hypotonia was pronounced.

Radiography confirmed gross cardiomegaly with normal lung vascularity. Electrocardiography showed sinus bradycardia (105/minute), right atrial hypertrophy, and a prolonged Q-T interval and T wave inversion in the left chest leads. Cross sectional echocardiography showed a structurally normal heart with marked left and right atrial enlargement, a poorly functioning left ventricle, and increased left and right ventricular wall thickness. These echocardiographic features suggested a primary cardiac muscle dysfunction.

The results of biochemical investigations (Table 1) showed a high serum lithium value, hyponatraemia, uraemia, a high serum creatinine value, and hypocalcaemia. Acid base balance and cardiac enzymes were normal. There was thrombocytopenia (platelets $50 \times 10^5/l$) and prolonged clotting times (prothrombin time 34 s, kaolin cephalin time 78 s, thrombin time 31 s).

A cardiac inotrope—isonrenaline—was infused (6 µg/hour) and produced instant improvement of left ventricular function monitored by 2-D echocardiography. The cardiac output remained stable during isoprenaline treatment, which was continued for 54 hours. With improving cardiac output, murmurs suggestive of tricuspid and mitral regurgitation became apparent. Echocardiography showed persistent poor left ventricular function at