CASE REPORT

Functional atrioventricular block in a preterm infant

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Abstract

A case of functional second degree atrioventricular block is reported in a preterm infant secondary to early onset hypocalcaemia. An infusion of 10% calcium gluconate rapidly corrected the arrhythmia.

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Cardiac arrhythmias in the neonatal period are unusual. Supraventricular tachycardias are more common than bradycardias, and may or may not represent underlying structural cardiac malformations. Bradycardias occurring in a neonatal intensive care unit are usually a response to hypoxia.

Electrolyte imbalances, particularly potassium, are well known to be associated with arrhythmias.

Case report

A baby boy was born by spontaneous vaginal delivery at 30 weeks gestation after an uneventful pregnancy. His birth weight was 1729 g (50th to 75th centile).

The baby developed moderately severe respiratory distress syndrome requiring mechanical ventilation. Initial serum potassium, sodium, calcium, phosphate, and magnesium were within normal limits.

On day 3 of life the baby developed profound bradycardia with a ventricular rate of 75 beats/min. Oxygen saturation, peripheral perfusion, and blood pressure were normal. A chest

radiograph was consistent with improving respiratory distress syndrome with no evidence of cardiomegaly. Examination of arterial blood gas showed mild metabolic acidosis with a bicarbonate level of 17.4 mmol/l and a base deficit of -4.7 mmol/l. Echocardiography showed a normally functioning heart with no structural abnormalities and no pericardial effusion.

At the time, the baby was being intravenously infused with 90 ml/kg/day 10% dextrose in 0.18% saline. Other medication was ampicillin and gentamicin. Electrolytes were normal except total serum calcium which was 1.01 mmol/l with an ionised level of 0.76 mmol/l. The electrocardiogram (ECG) showed a prolonged QT interval with a corrected QT interval (QTc) of 0.53 seconds and a 2:1 atrioventricular block. Alternate non-conducted P waves appeared before the T waves (fig 1A). A 10% calcium gluconate infusion was given with a bolus dose of 0.1 mmol/kg (= 0.5 ml/kg) followed by a maintenance intravenous infusion of 1 mmol/kg/day. During the calcium infusion, the ECG returned to normal, and a repeat ECG four hours later showed a normal sinus rhythm with a heart rate of 140/minute and a QTc of 0.48 seconds (fig 1B). Serum Ca^{2+} rose gradually to 0.886 mmol/l after two hours of the infusion and to 1.025 mmol/l after four hours. Subsequently it remained normal, and the ECG two days later was normal with a rate of 160/minute and a QTc of 0.40 seconds (fig 1C). The ECG of both parents was normal.

The baby was discharged home when 5 weeks old with no further cardiac complications.

Discussion

Depolarisation of myocardial cells is affected by pharmacological and biochemical factors. Hyperkalaemia impairs sodium flux across cell membranes, and hypocalcaemia can lead to prolongation of the plateau (phase 2) of the action potential with resultant prolongation of the QTc interval.¹

Transient physiological hypoparathyroidism is common in neonates, and is associated with a low serum calcium level. Preterm infants, particularly those stressed by asphyxia or anoxia, may have increased secretion of thyrocalcitonin which could be an additional contributing factor to prolonged and persistent hypocalcaemia in this group of neonates.

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Figure 1 Electrocardiogram (lead II rhythm strips) of preterm infant with atrioventricular block. (A) 2:1 atrioventricular block with a QTc of 0.53 seconds; (B) sinus rhythm with a QTc of 0.48 seconds; (C) sinus rhythm with a QTc of 0.40 seconds.

One study by Oppe and Redstone² showed an incidence of early onset hypocalcaemia—that is, within the first 72 hours of life—to be as high as 50%. Ill premature infants, particularly those with respiratory distress syndrome, have a higher incidence of hypocalcaemia because of inadequate intake of calcium and endogenous phosphate secretion from enhanced catabolism.

The association between hypocalcaemia and prolonged QT interval was first described by Carter and Andrus in 1922.³ This was followed by further reports indicating the association between hypocalcaemia and lengthening of the QT or the QTc intervals with possible alteration of the T waves.^{4–6} A study conducted on cattle showed lengthening of the QTc interval with inversion of the T wave during the period of induced hypocalcaemia.⁷ A case report by Fishbein *et al*⁸ showed an association between hypocalcaemia and heart block.

Congenital long QT syndrome may also produce 2:1 atrioventricular block as a result of an underlying myocardial repolarisation disorder. The high mortality from this disorder with neonatal presentation is probably related to the

extremely long QTc, as it has been recognised that a QTc of more than 0.6 seconds is the most predictive of sudden death, with a mortality of 83–91% in untreated cases.⁹

In conclusion, it is important to perform a 12 lead ECG and evaluate the QTc in infants with bradycardia, and to remember that this type of arrhythmia could be a manifestation of hypocalcaemia which is easily treated.

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