Vitamin D deficiency and cardiac failure in infancy

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Dilated cardiomyopathy is an important cause of heart failure in children. Often it requires transplantation, but on rare occasions it is curable by micronutrient supplements.

CASE HISTORIES

Case 1
A girl aged 4\frac{1}{2} months was referred in shock with acute heart failure and respiratory distress. She was of West African descent but had been born in the UK and breastfed from birth. Echocardiogram revealed a poorly functioning dilated heart (fractional shortening 6.5%; Table 1). She was admitted to the paediatric intensive-care unit where treatment included inotropic agents, furosemide and captopril. Intravenous antibiotics were also administered for suspected chest infection. Laboratory investigations revealed a severe normocytic anaemia (haemoglobin 6.8 g/dL), abnormal clotting (international normalized ratio 2.04) and a blood picture typical of vitamin D deficiency (low calcium [1.37 mmol/L], low phosphate, low vitamin D, raised parathyroid hormone and raised alkaline phosphatase). Radiographs showed bony changes consistent with rickets. Her dilated cardiomyopathy was therefore attributed to nutritional vitamin D deficiency.

In addition to supportive treatment for her cardiac failure and transfusion for the anaemia she received ergocalciferol and calcium. By day 11 serum calcium was within normal limits (2.52 mmol/L); phosphate remained low. At the time of her discharge on day 17 the echocardiogram showed persisting left ventricular dilatation, although ventricular function was improving (fractional shortening 17.8%). Subsequently all her biochemical results became normal, and fifteen months after discharge her left ventricular dilatation had completely resolved (fractional shortening 34%). She is now well without medication.

Case 2
This boy, aged 8 months and likewise of West African descent, presented in a similar way with shock, acute heart failure and respiratory distress; he also had a low blood glucose. Echocardiogram showed a dilated heart with fractional shortening 7.6% (see Table 1). Blood tests showed a normocytic anaemia (haemoglobin 8.3 g/dL), metabolic acidosis and deranged clotting (international normalized ratio 2.9). The biochemical picture was similar to that in case 1, with low calcium (1.55 mmol/L, vitamin D and raised parathyroid hormone). This infant had originally been breastfed at night and formula-fed during the day. However, from the age of 4\frac{1}{2} months his diet had consisted mainly of African meats and rice with very few dairy products.

Serum calcium did not improve with calcium supplementation, so he was started on 1-alpha-calcidol, later changed to ergocalciferol. His anaemia worsened and he required a blood transfusion. 21 days after admission he was stable enough for discharge home, although his heart remained enlarged with poor functioning (fractional shortening 11%). Thereafter he made excellent progress, with normal biochemistry values at one month and normal cardiac function at twelve months (fractional shortening 30%). Because of persistent mild left ventricular dilatation, his cardiac medications were continued.

COMMENT
Vitamin D deficiency is commonly due to inadequate nutrition and insufficient exposure to ultraviolet. The most prominent biochemical feature is hypocalcaemia, and this can adversely affect ventricular contraction.¹ In treatment

<table>
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<tr>
<th>Table 1</th>
<th>Echocardiographic indices</th>
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<tbody>
<tr>
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<td>Infant 1</td>
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<tr>
<td>Presentation</td>
<td>LVEDD (cm) 4.6</td>
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<td></td>
<td>LVESD (cm) 4.3</td>
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<td></td>
<td>FS (%) 6.5</td>
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<td>Discharge</td>
<td>LVEDD (cm) 3.9</td>
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<td>LVESD (cm) 3.2</td>
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<td></td>
<td>FS (%) 17.4</td>
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<td>Follow-up</td>
<td>LVEDD (cm) 3.1</td>
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<td></td>
<td>LVESD (cm) 2.0</td>
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<td></td>
<td>FS (%) 34</td>
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<td>(15 months)</td>
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LVEDD=left ventricular end-diastolic diameter; LVESD=end-systolic diameter; FS=fractional shortening.
of rickets (often due to vitamin D deficiency), calcium with or without vitamin D supplementation may be more effective than vitamin D alone.2 There are not many reports on dilated cardiomyopathy associated with vitamin D deficiency in infants.3–7 Other nutritional deficiencies to be considered8 are those of carnitine, selenium and taurine—carnitine in particular, because depletion can diminish ventricular contractility9 and oral supplementation of L-Carnitine is effective.10

Dilated cardiomyopathy caused by nutritional deficiencies seems to have a better prognosis than idiopathic dilated cardiomyopathy. Appropriate treatment can lead to complete resolution. Thus, in an infant with cardiac failure, a nutritional cause deserves early consideration.

REFERENCES

1 Uusyal S, Kalayei AG, Baysal K. Cardiac functions in children with vitamin D deficiency rickets. Pediatr Cardiol 1999;20:283–6

The scalp is a common site for benign skin lesions. When excision is contemplated, careful attention must be paid to those in the midline, especially in children.

CASE HISTORIES

Case 1

A boy aged 13 months was admitted to his local hospital for excision of a ‘sebaceous cyst’ from the back of his head. During the procedure the general surgical team noted a cerebrospinal fluid (CSF) leak, closed the wound and sent him across to the Birmingham Children’s Hospital for further treatment. He arrived intubated and ventilated. A CT scan demonstrated a dermoid sinus extending through the occipital bone to the cervico-medullary junction (Figure 1). At operation the original transverse incision was opened to reveal a 0.5–1.0 cm defect in the occipital bone. All abnormal tissue was removed from this area. The defect was closed with a pericranial patch reinforced with Tissel fibrin glue and muscle to achieve a watertight seal. The patient recovered well and the CSF leak did not recur.

Case 2

An 18-month-old girl was taken to her local hospital with increasing drowsiness, vomiting and diarrhoea. There had been an episode of jerking movements associated with eye rolling. She had previously been developing normally, but her family doctor had been consulted about a small discharging scalp lesion on the back of her head. This had been treated with topical antibiotics intermittently for several months. On examination she was drowsy although when woken was very irritable and there was a suggestion of discomfort on moving her neck. She was otherwise neurologically intact. A discharging sinus was noted between the hairline and the external occipital protuberance. Her white cell count was $29.7 \times 10^9/L$. CT scan showed dilatation of the lateral, third and fourth ventricles with meningeal enhancement and she was transferred to our department. An MRI scan then revealed a sinus and

Beware of the midline scalp lump

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239