Case Reports

Left Ventricular Dysfunction Due to Hypocalcemia in a Neonate

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SUMMARY

Hypocalcemia is a relatively uncommon but reversible cause of left ventricular dysfunction in infants and children. A 30-day-old boy with idiopathic hypocalcemia presented with congestive heart failure and convulsive seizures. He had no evidence of underlying cardiac disease. The cardiac failure responded to calcium therapy. It is suggested that hypocalcemia should be considered as a possible cause of left ventricular dysfunction in infants. (Jpn Heart J 34: 355-359, 1993)

Key Words: Hypocalcemia  Left ventricular dysfunction

It is well known that calcium, phosphorus and magnesium are intimately involved in the metabolism and function of cardiac muscle. Although calcium plays a key role in excitation-contraction coupling, the occurrence of left ventricular dysfunction secondary to hypocalcemia is quite rare, with only a handful of reports in the literature.1)-5)

We present one infant with hypocalcemia-induced left ventricular dysfunction due to idiopathic hypocalcemia who was treated with calcium alone. The serum calcium level rose to normal within 10 days following administration of calcium gluconate (first 7 days) and afterwards calcium lactate, associated with complete clearing of the signs and symptoms of heart failure. Improvement in cardiac function was documented by an increase from 21 to 33 percent in the left ventricular fractional shortening.

CASE REPORT

The patient was a Turkish boy, who was the product of full-term, normal
Fig. 1. Chest radiography revealed cardiomegaly on admission (A), sixteen days later cardiac size was reduced to normal limits (B).

Table I. M-mode Echocardiographic Findings

<table>
<thead>
<tr>
<th></th>
<th>On admission</th>
<th>16 days later</th>
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</thead>
<tbody>
<tr>
<td>IVSTD</td>
<td>5 mm</td>
<td>6 mm</td>
</tr>
<tr>
<td>LVIDD</td>
<td>31 mm</td>
<td>23 mm</td>
</tr>
<tr>
<td>LVPWD</td>
<td>4 mm</td>
<td>5 mm</td>
</tr>
<tr>
<td>IVSTS</td>
<td>7 mm</td>
<td>8 mm</td>
</tr>
<tr>
<td>LVIDS</td>
<td>24 mm</td>
<td>17 mm</td>
</tr>
<tr>
<td>LVPWS</td>
<td>6 mm</td>
<td>6 mm</td>
</tr>
<tr>
<td>EF</td>
<td>0.44</td>
<td>0.64</td>
</tr>
<tr>
<td>FS</td>
<td>0.21</td>
<td>0.33</td>
</tr>
<tr>
<td>Mitral-septal distance</td>
<td>21 mm</td>
<td>4 mm</td>
</tr>
</tbody>
</table>

IVSTD=interventricular septal thickness (diastolic); LVIDD=left ventricular end diastolic dimension; LVPWD=left ventricular posterior wall thickness (diastolic); IVSTS=interventricular septal thickness (systolic); LVIDS=left ventricular end systolic dimension; LVPWS=left ventricular posterior wall thickness (systolic).

delivery, with a birthweight of 3200 g. At 30 days of age he was admitted to Doctor Sami Ulus Children Hospital with generalized convulsive seizures and a 5-day history of nonproductive cough. Seizures of one minute duration occurred 15–20 times a day. Physical examination on admission revealed a pulse rate 152/min, blood pressure 80/60 mmHg, temperature 37°C, respiratory rate 52/min, height 54.5 cm and weight 4600 g. Nutritional status was good. The thyroid was not enlarged. Bibasilar pulmonary moist rales were heard. Heart sounds were normal and no murmurs were heard. The liver edge was palpable 3 cm below the right costal margin.

Admission laboratory data included normal serum sodium, potassium, chloride, albumin, bicarbonate, glucose, blood urea nitrogen, creatinine, serum glutamic oxalacetic transaminase, complete blood count and urinalysis. The total serum calcium level was 4.2 mg/dl, phosphorus 8.5 mg/dl, alkaline phosphatase 6 U, magnesium 2.3 mg/dl (normal 1.6–2.2), 1–25 dihydroxy Vit D 24.1 pg/ml
Fig. 2. M-mode and two-dimensional echocardiograms on admission (A) and sixteen days later in the long axis view (B).

IVS=interventricular septum; LV=left ventricle; RV=right ventricle; LA=left atrium; RA=right atrium; IAS=interatrial septum; Ao=aorta; LVPW=left ventricular posterior wall.
(normal 15.1–49.9), 25 OH Vit D 19.8 ng/ml (normal 9.2–38.4), C-terminal parathyroid hormone 0.48 ng/ml (normal<0.92). Chest radiography demonstrated marked cardiac enlargement (Fig. 1A) and increased flow to the upper lobes. Electrocardiography showed low amplitude T waves in leads I, V5 and V6 with a QT interval of 0.28 second. Blood levels of calcium, phosphorus and alkaline phosphatase of the infant’s mother were normal. Two-dimensional echocardiography confirmed enlargement of all cardiac chambers with an ejection fraction of 44 and a shortening fraction of 21 percent. M-mode and two-dimensional echocardiographic findings are shown in Table I and Fig. 2A.

Therapy consisted of calcium gluconate and phenobarbital. On the seventh day therapy was changed to calcium lactate. Although phosphorus level decreased to 5.9 mg/dl on the third day hypocalcemia continued. Sixteen days after admission, when the calcium level was 9 mg/dl, the ejection fraction and shortening fraction had increased to 64 percent and 33 percent, respectively (Fig. 2B, Table I) and chest radiography (Fig. 1B) returned to normal. He was seen forty days later and had remained well with no abnormalities on clinical examination.

**DISCUSSION**

The central role of calcium ions (Ca++) in the sequence of myocardial excitation-contraction coupling and myocardial relaxation in now well appreciated. After it is diffused into the region of the myofibrils, free Ca++, by binding to troponin-C, induces a reversal of the inhibition of action and myosin contraction by the troponin-tropomyosin-complex, an effect that allows the interaction of actin and myosin with resultant sliding of filaments and production of myocardial contraction-relaxation occurring during repolarization via a reduction in myoplasmic Ca++ binding to the sarcoplasmic reticulum. Calcium ions are also apparently involved in the mechanisms of the direct positive inotropic effect of digitalis.6,7

Cardiac dysfunction occurs during acute reductions in serum calcium levels in children and adults as well as in experimental animals,8–10 but in infants and children reports of heart failure due to hypocalcemia have been very rare. Edge8 (1963) described three infants with congestive heart failure associated with hypocalcemia and Najjer et al11 (1967) reported the case of an 18-month-old child with rickets and heart failure. Troughton and Singh12 (1972) described six neonates with congestive heart failure and hypocalcemia, and Bashour et al13 (1980) reported a ten-year-old girl with hypocalcemic cardiomyopathy. We present an infant with seizures and congestive heart failure for which no cause could be found other than a low serum calcium level.
Chronic hypocalcemia has also been associated with myocardial dysfunction in numerous case reports.2–4,11–13 On the contrary, Vered et al14 found that long-standing hypocalcemia of variable severity (serum total calcium 5.3 to 8.5 mg/dl) in the absence of other predisposing factors does not appear to diminish left ventricular performance. Additionally, they found that the presence of electrocardiographic abnormalities during hypocalcemia does not necessarily indicate myocardial dysfunction.

The rarity of heart failure in infants with hypocalcemia has been explained by the theory that convulsive seizures occur early and result in treatment. Thus Levine and Rheams2 (1985) state that hypocalcemia must be pronounced and protracted before it produces cardiac manifestation.

In our patient congestive heart failure was due to hypocalcemia. The role of the high level of phosphorus in cardiac dysfunction was considered, but on the third day, when the phosphorus level was normal, no improvement of congestive heart failure was noted. As the patient had no underlying structural heart disease and left ventricular dysfunction improved with calcium therapy alone, we considered the role of hypocalcemia in its etiology.

Hypocalcemia as a cause of cardiac decompensation is often ignored in standard textbooks of medicine and cardiology. Despite its relatively infrequent occurrence, physicians should be aware that hypocalcemia is a reversible cause of congestive heart failure.

REFERENCES