Childhood Hypocalcemia: The Aetiological Pattern

By Nasir A. M. Al Jurayyan, MD
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Abstract - Background: Hypocalcemia is not that rare condition and could be a potentially life threatening. Identifying the etiology is important for successful management.

Results: A total of 60 patients were seen in the period under review, December 1989 and June 2016, with childhood hypocalcemia. Twenty-seven (45.0%) patients were parathyroid hormone deficient, while rickets diagnosed in 25 (41.7%) patients.

Design and setting: A retrospective, hospital based study was conducted at King Khalid University Hospital (KKUH), Riyadh, Saudi Arabia during the period December 1989 and June 2016.

Materials and Methods: Medical records of children beyond the neonatal period with hypocalcemia were reviewed for aetiological diagnosis. Detailed history, clinical manifestation, and results of all the laboratory, and radiological investigations were obtained.

Conclusion: This study showed that parathyroid hormone (PTH) deficiency (45%) and rickets (41.7%) were the most common causes of childhood hypocalcemia.

Keywords: aetiology, childhood, hypocalcemia.

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I. INTRODUCTION

Hypocalcemia is a potentially life-threatening metabolic disturbance. It can result in severe symptoms that require rapid management. Hypocalcemia occurs most commonly as a result of deficiency of parathyroid hormone (PTH). Though, there are many other potential etiologies of hypocalcemia, one usually does not consider them seriously unless the most common cause is ruled out or unless the initial evaluation suggest another cause is likely.

In primary hypoparathyroidism, an assay that measures intact circulating PTH will be low, while in virtually all other causes associated with hypocalcemia. PTH levels are elevated.1-4

This article focuses upon the etiology of hypocalcemia beyond the neonatal period, seen in a major teaching hospital, King Khalid University Hospital (KKUH), Riyadh, Saudi Arabia over three decades, December 1989 to July 2016.

Then KKUH is the main teaching hospital of the King Saud University (KSU) and considered as one of the major referral hospitals in the region, and provides primary, secondary, and tertiary health care services for the local population and also receives patients referred from all over the country.

II. MATERIALS & METHODS

During the period under review, December 1989 to June 2016, all patients who were diagnosed, beyond the neonatal period to have hypocalcemia were retrospectively reviewed. Detailed history, clinical manifestations and results of all the laboratary, radiological and ancillary investigations were obtained. The aetiological diagnosis was based on specific investigations as recommended.

III. RESULTS

During the period under review, December 1989 and June 2016, a total of 60 patients beyond neonatal period were seen by the author in the pediatric endocrine service, King Khalid University Hospital, Riyadh, Saudi Arabia. Table, showed the aetiological diagnosis of the group. In 27 (45.0%) patients, parathyroid hormone (PTH) deficiency was found while rickets was the diagnosis in 25 (41.7%) patients. Celiac disease was diagnosed in 6 (10.0%) patients.

IV. DISCUSSION

Hypocalcemia beyond neonatal period is not that rare. It varies from an asymptomatic biochemical abnormality to a life threatening conditions, depending on the duration, severity and rapidity of development. Hypocalcemia is caused by loss of calcium into circulation. In a community with high prevalence of consanguinity mating and increased incidence of autosomal disorders,5-6 various forms of hypoparathyroidism exist and constitute the major cause. Simple hypoparathyroidism usually occurs sporadically, though an autosomal dominant pattern of inheritance has been reported. In most cases the pathogenesis is unknown, but agenesis, partial or complete atrophy, and inflammatory damage of the parathyroid glands are possible mechanisms. However the diagnosis of isolated hypoparathyroidism cannot be made with certainty in childhood, since children who first appears to have this disorder often develop additional endocrine or immunological abnormalities later on.7-9

Damage to the parathyroid glands is a well-established risk of neck surgery, especially during total or subtotal thyroidectomy. Permanent parathyroid
deficiency occurs in about five to ten percent of subtotal thyroidectomies and is significantly more common after total thyroidectomy for malignant thyroid disease. Hypoparathyroidism is usually caused by an interference with the blood supply of the glands and is rarely due to complete ablation of the parathyroid tissue. Non-surgical damage to the parathyroid glands can occur as a result of massive doses of external irradiation. However, the parathyroids are relatively radiation resistant so definite hypoparathyroidism following treatment for thyroid disease is exceptionally rare.

A well-established relationship exists between magnesium and calcium homeostasis. Magnesium deficiency may lead to hypocalcemia by either PTH synthesis or release and end-organ, bone, refractoriness to the effects of PTH. Vitamin D deficiency was common, however, derangement in Vitamin D metabolites or action is not that rare.

Celiac disease should be considered in patients with hypocalcemia of unknown etiology, especially because gastrointestinal symptoms may be absent or mild. Six (10%) patients in our series were diagnosed to have celiac disease.

In conclusion, this study showed that parathyroid hormone (PTH) deficiency (45%) and rickets (41.7%) were the most common causes of childhood hypocalcemia.

V. ACKNOWLEDGEMENT

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Conflict of Interest

The author have no conflict of interest to declare.

REFERENCES REFERENCES REFERENCIAS

**Table 1:** Aetiology of childhood hypocalcemia in 60 patients

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number</th>
<th>(%)</th>
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</thead>
<tbody>
<tr>
<td><strong>Parathyroid hormone (PTH) deficiency (45%)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Isolated hypoparathyroidism</td>
<td>10</td>
<td>16.67</td>
</tr>
<tr>
<td>• Hypoparathyroidism associated with Sanjad-Sakatisyndrome</td>
<td>6</td>
<td>10.00</td>
</tr>
<tr>
<td>• Hypoparathyroidism associated with autoimmune polyendocrine syndrome</td>
<td>7</td>
<td>11.67</td>
</tr>
<tr>
<td>• Post-thyroidectomy</td>
<td>2</td>
<td>3.33</td>
</tr>
<tr>
<td>• Hypomagnesium</td>
<td>2</td>
<td>3.33</td>
</tr>
<tr>
<td><strong>Rickets (41.7%)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Nutritional rickets</td>
<td>13</td>
<td>21.67</td>
</tr>
<tr>
<td>• Anti-convulsant induced rickets</td>
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<td>6.67</td>
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<tr>
<td>• Vitamin D dependent rickets Type 2</td>
<td>5</td>
<td>8.33</td>
</tr>
<tr>
<td>• Hypophosphotemic Rickets</td>
<td>2</td>
<td>3.33</td>
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<tr>
<td>• Pseudo hypo-hypo-parathyroidism type 1b</td>
<td>1</td>
<td>1.67</td>
</tr>
<tr>
<td><strong>Miscellaneous (13.3%)</strong></td>
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<td></td>
</tr>
<tr>
<td>• Celiac disease</td>
<td>6</td>
<td>10.0</td>
</tr>
<tr>
<td>• Chronic renal failure</td>
<td>2</td>
<td>3.33</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>60</td>
<td>100</td>
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