

## CASE REPORT

**Hypocalcemic Tetany in a 10 Year Old Girl****Mustafa Kamil AbdUlmajeed Alqaysi****BACKGROUND:**

Autoimmune hypoparathyroidism is rare in children. We present a 10 year old girl who presented with hypocalcaemic tetany due to autoimmune hypoparathyroidism.

The term hypoparathyroidism refers to a group of disorders in which the relative or absolute deficiency of PTH leads to hypocalcemia and hyperphosphatemia. common cause of hypoparathyroidism are surgical removal of parathyroid glands, radiation, drugs such as alcohol, malignancy, autoimmune disorders and genetic mutations.<sup>(1)</sup> Autoimmune hypoparathyroidism may occur alone or in association with additional features , including mucocutaneous candidiasis and adrenal insufficiency, as a component of the autoimmune polyglandular syndrome type 1 (APS).<sup>(2)</sup> We report a 10 year's old girl with isolated autoimmune hypoparathyroidism.

**CASE REPORT:**

A 10 year old Indian girl presented with recurrent carpopedal spasm and tetanic postures a both hands and feet since 2 months .She was born of third degree consanguineous marriage and was 2nd of 3 children with other siblings being normal. Her vegetarian diet contained no milk or milk products or vegetables. On examination she had no signs of rickets or short stature. Her blood pressure was normal. Her trousseau's sign was positive .She had no skin pigmentation or oral thrush. Thus she was suspected to have hypocalcemic tetany due to either hypoparathyroidism or chronic renal failure. Investigations showed hypocalcemia (serum calcium=7 mg %) with hyperphosphatemia (serum phosphors =10.0 mg %) and elevated serum alkaline phosphatase(320IU/L) with serum ionic calcium of 0.5mmol/L. Her renal functions, blood gas analysis, hemogram and urine calcium/creatinine was normal. Her serum parathyroid levels were normal 21.4 pg/ml (normal=12-72pg/ml) inspite of a low serum

calcium suggestive of primary hypoparathyroidism. Her thyroid function tests were normal. Antimicrosomal antibody was elevated suggesting isolated autoimmune hypoparathyroidism. She was treated with IV, calcium, oral calcitriol and calcium carbonate supplements.

**DISCUSSION:**

Hypoparathyroidism can occur due to aplasia or hypoplasia of parathyroid glands, as in (DiGeorge syndrome, shprintzen syndrome), parathyroid hormone gene mutations, autoimmune parathyroiditis( in isolation or with Addison's disease and mucocutaneous candidiasis , hemosiderosis, Wilson's disease, Familial congenital variant with dysmorphic features or surgical removal or Damage to the parathyroid glands during thyroidectomy<sup>(1)</sup>

Autoimmune hypoparathyroidism is suggested by the finding of parathyroid antibodies and by frequent association with other autoimmune disorders. It is often seen as apart of polyglandular autoimmune disease type 1(at least 2 of the following are required, autoimmune hypoparathyroidism, Addison's disease, and chronic mucocutaneous candidiasis). In this polyglandular endocrinopathy candidiasis precedes the other disorders followed by hyperparathyroidism and then Addison's disease.<sup>(2)</sup>

Patients present with muscle pain and cramps, stiffness, tingling of hands and feet with laryngeal or carpopedal spasm. Convulsion and cataract may occur in long standing untreated cases. Investigations reveal hypocalcemia with hyperphosphatemia. Serum alkaline phosphatase is normal or low but high levels may be seen in patients with severe hypocalcemia. Serum parathyroid levels are low<sup>(3)</sup>. Laboratory finding in hypoparathyroidism are hypocalcaemia,

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hyperphosphatemia and low or inappropriately normal parathyroid level that because reserve secretory capacity of parathyroid gland is present, ultimately the level of parathyroid hormone is low when reserve secretory capacity is lost. Treatment consists of intravenous calcium gluconate (10% solution 5 to 10 ml rate of 0.5-1 mL/min) for emergency treatment of tetany. supplementation with 1, 25 dihydroxycholecalciferol in adose of 0.01 to 0.1 ug/kg/day to maximum of 1-2 ug/day is required. Once normocalcemia is achieved, one can continue therapy with vitamin D2 (50,000-1, 00,000 IU) daily to make therapy economically feasible. Vitamin D3 has the advantage of rapid onset of action and rapid reversal of hypocalcaemia after discontinuation in the event of over dosage. Calcium supplement should be ensured. High phosphorus containing food such as milks, eggs and cheese should be avoided <sup>(4,5,6,7)</sup> monitoring of patient and frequent estimation of serum calcium levels is required to determine the requirement of vitamin D.

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