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A case of rare association of Sporadic Primary Hypoparathyroidism with Rupioid Psoriasis

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I. INTRODUCTION:

The causes of hypoparathyroidism are hereditary, sporadic and post- surgical.[1] This patient presented with hypocalcaemia features. The neuromuscular effects of calcium are due to positive bathmotropic effects. When the ionized calcium level is <4.4mg/dl, neuromuscular effects are noted namely Trousseau's sign, Chvostek's sign, and seizures.[2] The association of hypoparathyroidism with generalised pustular psoriasis have been reported previously, but association with rupioid psoriasis yet to be documented.[3,4]

The term "rupioid" (from the Greek rhupos, meaning filth) is used to describe oyster or limpet shell-shaped thick keratotic lesions. In contrast to rupioid forms, regular plaque-type psoriasis has a white, nonadherent and thin, scaly surface. Severe psoriatic flares and atypical forms of the disease (including rupioid plaques) have been reported in HIV positive patients. Rupioid psoriatic flares have also been associated with drug treatments, such as oral or intravenous corticosteroids, nonsteroidal anti-inflammatory drugs. One report attributed a flare to oral steroid reduction.[5] We present a young female with hypoparathyroidism and rupioid psoriasis.

II. CASE REPORT:

A 34-year-old female presented to Emergency department with maculopapular skin lesions all over the body for one month. She was a known case of seizure disorder and recurrent tetany from 10 yearsof age. (Figure1). She had generalized tonic clonic seizure episodes during childhood and was on antiepileptic drugs since then. She is also on antianxiety drugs for the past 20 years. During childhood there is a history of failure of secondary dentition. She is a known case of psoriasis on treatment for 20 years of age.

Cataract surgery was done in the both eyes at 13 years of age, dental implantation was done at 18 years of age. She had normal menstrual periods since puberty which was attained at 15 years of age. Ovarian cystectomy was done at 20 years of age for serous cystadenoma of ovary which was followed by amenorrhea for 6 months. She resumed menstruation following treatment with hormone pills. There was no history of neck surgery. No other family members had similar history.

On General examination, patient was comfortable at rest. She had bilateral pedal edema. Vitals were stable. On Dermatological examination, multiple erythematous papules and some vesicles with surrounding rim of erythema and central necrosis were present over the entire trunk and bilateral upper limbs and lower limbs. (Figure 2). On examining central nervous system carpopedal spasm, Chvostek's sign and Trousseau sign were present. Examination of cardiovascular, respiratory, gastrointestinal systems were normal.

Investigations were as follows: Total white cell count -30600 cells/cumm, DC Polymorphs 92%, Lymphocytes -2%, Eosinophils2%, Renal and liver function tests were normal. Serum calcium was 6.7mg/dl Corrected serum calcium 8.3mg/dl,S.Magnesium-2.0mg/dl, Serum Proteins- 5.2gm/dl, S.Albumin3.2gm/dl S.Phosphorus 10.3mg/dl, S.Parathyroid hormone <0.02pg/ml(1055pg/ml).

Calcium phosphate product was 69.01, Absolute Eosinophilic Count was 0.59 cells/cumm, Serum IgE-878 IU/ml. Patient had a normal thyroid profile. HBsAg, Anti HCV, HIV tests were negative. Anti-nuclear antibody, Rheumatoid factor,

Rapid Plasmin Reagin flocculation tests were negative. Ultrasound neck, abdomen and pelvis showed normal study. Skin biopsy showed features suggestive of psoriasis. Peripheral smear showed normocytic normochromic picture. CT Brain showed multiple discrete calcifications noted involving bilateral subcortical white matter, bilateral basal ganglia, bilateral thalamus, bilateral centrum semiovale and bilateral cerebellum. Skull wall thickening with clover leaf pattern was noted

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suggestive of hypoparathyroidism (Figure 3). CT chest showed bilateral minimal pleural effusion with sub segmental atelectasis.

III. DISCUSSION:

The Hypoparathyroidism was diagnosed in our patient based on low ionized and total calcium, high phosphate and very low parathyroid levels in the presence of normal magnesium levels. In the background of early onset clinical features, no history of thyroid surgery and the absence of other endocrine involvement suggesting Autoimmune polyendocrinopathy, a diagnosis of sporadic hypoparathyroidism was made. The seizure was due to hypocalcemia. Causes of pathological cerebral calcifications in these settings include hypoparathyroidism, Fahr's disease, tumours, congenital infections and neurocutaneous syndrome.

Phosphorus value >4.5 mg/dl is denoted as hyperphosphatemia. Severe hyperphosphatemia is managed with dietary phosphate restrictions, removal of offending drugs, and by treatment with oral phosphate binders. Tablet Calcium acetate is the phosphate binder commonly used at a dose of 667 mg 2 tds. Sevelamer may be used if the above treatment is ineffective.

Patient was treated with Inj Calcium gluconate infusions appropriately and was given maintenance oral calcium up to 2 grams/day. As the corrected calcium was >7.5mg/dl, oral calcium was given and whenever the corrected calcium fell below 7.5mg/dl, 1-2g intravenous calcium was used. Simultaneous correction of vitamin D and magnesium is also warranted.

In severe hypoparathyroidism treatment with Vitamin D is required.PTH deficiency impairs conversion of vitamin D to Calcitriol (1alphahydroxy D3). Therefore, the most efficient treatment is oral tablet Calcitriol 0.5-2mcg OD.Recombinant human PTH(Natpara) can also be used as an adjuvant to calcium and vitamin D. If Calcium Phosphate product >70mg/dl, it leads to calcification. We have to maintain Calcium phosphate product below 55 mg/dl.

Hypoparathyroidism is rare among endocrine disorders in not being treated with the missing hormone. The full-length molecule PTH (1-84) has been shown to be effective and is now FDA-approved for therapy of hypoparathyroidism.

Published reports illustratethat its use substantially reduced the requirements for supplemental calcium and active Vitamin D to maintain serum calcium. Recent recommendations on management of hypoparathyroidism suggests its use, particularly in patients with inadequate control of blood calcium, requirement for excessively high doses of calcium and active vitamin D replacement and or high urine calcium[6].

Figure 1: Tetany

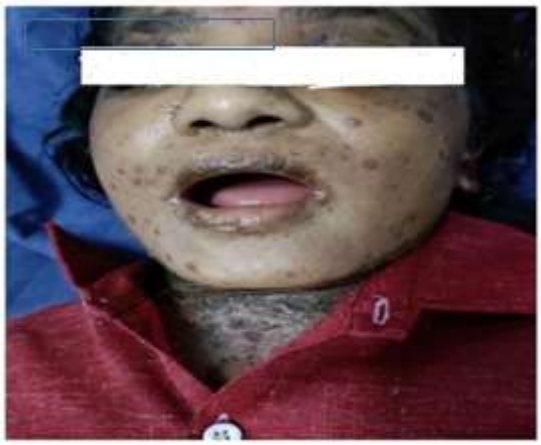

Figure 2 : Psoriasis

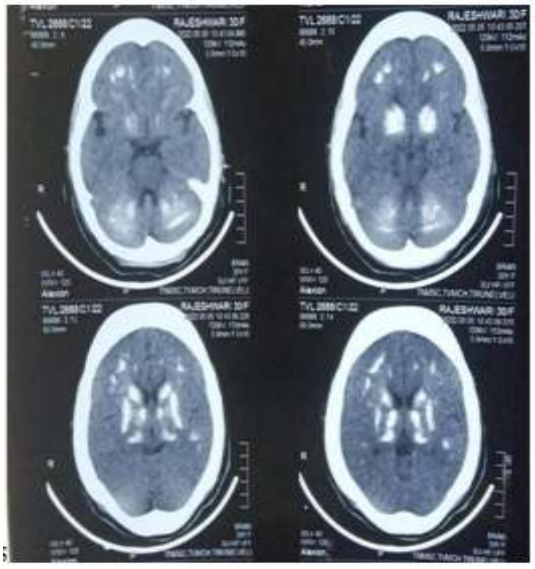

Figure 3: INTRACEREBRAL CALCIFICATIONS

IV. CONCLUSION:

This case is reported for the rare association of rupioid psoriasis with sporadic primary hypoparathyroidism. Further insights into whether there are genetic associations between psoriasis and hypoparathyroidism needs to be evaluated.

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