Case Report

Multiple Osteosclerotic Lesions: A Rare Presentation of Hyperparathyroidism Secondary to Hypovitaminosis D

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ABSTRACT

Hypovitaminosis D is an under-recognised and important factor responsible for secondary hyperparathyroidism. Hyperparathyroidism mostly presents with osteolytic lesions, especially in secondary hyperparathyroidism with renal failure. There are some rare presentations of hyperparathyroidism as focal or generalized osteosclerotic lesions. The exact mechanism for the osteosclerosis is still unknown, but there are some theories suggesting an exaggerated response of osteoblasts in response to prolonged osteolytic activity in order to restore bone loss. Here, we report a case of secondary hyperparathyroidism secondary to severe Vitamin D deficiency presented as multiple osteosclerotic lesions.


INTRODUCTION

Hypovitaminosis D is an under-diagnosed and a significant cause of secondary hyperparathyroidism. The prolonged duration of vitamin D deficiency leads to bone pains, fractures, and osteomalacia in adults or rickets in children.1 Unlike West, in Asian countries, a substantial number of people are significantly deficient in Vitamin D, leading to skeletal manifestations.2 Parathyroid hormone (PTH) synthesis and secretion is increased, secondarily to decreased levels of Vitamin D; hence, referred to as secondary hyperparathyroidism. The later, increases the osteoclastic activity resulting in osteopenia, brown tumors, bone erosions and fractures.3,4 Osteosclerotic lesions, either focal or diffuse, have rarely been described in literature, especially in patients with co-existing hypovitaminosis D and hyperparathyroidism.5

Here, we report a case presenting as multiple osteosclerotic lesions due to secondary hyper-parathyroidism resulting from vitamin D deficiency.

CASE REPORT

A 40-year lady presented to us with chief complaints of generalized body aches and pains for the last 10 years and progressive proximal muscle weakness for the last 3 years. She was bedridden for the last 2 years due to weakness and painful joints which restricted her indoors. Investigations revealed severe 1, 25-dihydroxyvitamin D deficiency (10.2 ng/ml), low ionized calcium (3.9 mg/dl), serum phosphate levels (2.0 mg/dl) and increased PTH hormone (461.6 pg/ml). Renal function tests, acid phosphatase, procalcitonin, thyroid functions tests and ultrasound neck, were normal. Skeletal survey showed multiple lytic and sclerotic lesions in pelvic bones, skull and axial skeleton with multiple dorso-lumbar vertebral collapses as shown in Figure 1. There was no scintigraphic evidence of parathyroid adenoma as depicted in Figure 2a and 2b. Bone scan was suggestive of metabolic bone disease as illustrated in Figure 3. Malignancy, as a possible cause of osteosclerotic lesions was ruled out on the basis of the parathyroid scintigraphy and bone scan. She was diagnosed as vitamin D deficient, resulting into secondary hyperparathyroidism. She was started on vitamin D and calcium replacements therapy and improved significantly on follow-up visit.

DISCUSSION

Vitamin D plays a vital role in mineralization of bone and regulation of calcium levels in the blood.6 PTH has been recongnised as a catabolic hormone for the skeletal system causing calcium mobilization by bone resorption, leading to osteolytic lesions.7 The pathophysiology of range and effect of PTH on bones has been largely elusive; ironically, some patients develop osteosclerotic lesions (focal, generalized, or both), especially in secondary hyperparathyroidism with renal failure.8 Bone resorption and later on its replacement leads to decrease in the bone density, formation of cysts, bone tumors, and even pathological fractures.2

The western countries have improved the vitamin D status of the population and it significantly decreased the diseases of bone related to vitamin D deficiency. Unfortunately, the citizens of developing countries still suffer from low vitamin D levels resulting in increase in frequency of bone diseases. The condition of patients is
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more intense when it is associated with co-existing endocrine disorders especially of thyroid and parathyroid disorders.2

Fujino and his colleagues published a report describing multiple osteosclerotic skull lesions in a 26-year patient suffering from primary hyperparathyroidism.5 Boechat et al. reported two children with primary hyperparathyroidism, present with tibial osteosclerosis.9 Chopra and his colleagues reported two female patients with osteosclerotic skull lesions; one with primary hyperparathyroidism, and the other with vitamin D deficiency leading to secondary hyperparathyroidism.2 Our patient shares the same features as the latter case of this report.

The exact mechanism and pathogenesis of osteosclerotic lesions is still mysterious.10 Prolonged osteoclastic activity renders the catabolic effect of the PTH towards the anabolic effect, resulting in exaggerated response of the osteoblasts and this phenomenon is more pronounced in younger patients having high bone turnover rate.2,5

To conclude, osteosclerotic lesions in secondary hyperparathyroidism are a rare presentation. Additionally, more research work is warranted in order to identify the exact pathogenesis of osteosclerotic lesions.

REFERENCES
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