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Oncogenic hypophosphatemic osteomalacia: From the first signal of disease to the first signal of healthy



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ABSTRACT

INTRODUCTION: The physical incapacitation of the oncogenic hypophosphatemic osteomalacia (OHO) can be catastrophic and can lead to deformities, metabolic and organic instability and death. The only positive outcome is through early diagnosis by the clinical suspicion. At this moment, medical center infrastructure is also a keypoint.

PRESENTATION OF CASE: This case report is about a 60-year old woman with multiple fractures, gradual loss of strength and muscle mass and limiting deformities in two years of evolution until the diagnostic.

DISCUSSION: The lack of knowledge of this disease causes a delay in diagnosis that can bring deformities to the patient, as well as death. Is crucial that is hypothesized to carry out the necessary tests, since they are expensive and not always available.

CONCLUSION: This case reinforces the importance to understand the OHO and tumoral search, once this lesion is, in most cases, imperceptible to physical examination or several imaging studies.

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1. Introduction

OHO is an uncommon paraneoplastic syndrome, mostly induced by small benign mesenchymal tumors that can be found in any location [1]. This syndrome causes structural deformations by demineralization of the bone owing to low levels of phosphatemia and 1,25-dihydroxy-vitamin D, hyperphosphatury (with normal serum parathyroid hormone, calcium and 25-hydroxy-vitamin-D levels) [2,3].

Low tubular phosphorus absorption and some alterations on vitamin D metabolism are unleashed by excessive Fibroblast Growth Factor 23 (FGF 23) secreted by the tumor, resulting in structural changes. At the end of this cycle, the patient develops osteomalacia [3,4]. The first signals are pain to develop some activities, loses body mass, evolve to body moves limitations, multiple fractures, renal and heart dysfunctions that can lead to death [4].

A tumor resection is the only way to stop this event loop and, as sooner the diagnosis is made (and consequently the surgery), smaller will be the sequels [5].

2. Presentation of case

A 60-year old woman showed malnutrition, repeated cramps, general pain and multiples fractures after minimal traumas in two years of evolution. She used non-steroidal anti-inflammatory drugs and opioids to control the pain, but without great results. A year ago, she broke the left humerus after a minimal energy trauma, requiring the use of plaster cast for eight months. After ten months, she broke the right humerus, presented gradual loss of strength and muscle mass, especially in lower limbs, which led to the use of crutches and, posteriorly, confined to bed.

She had a long term diabetes-2, hard control hypertension and subclinical hypothyroidism, without familiar diseases compatible with her issues. Several fractures were found, when she was admitted in hospital, in ribs, right subtrochanteric area, bilateral coxarthrosis and right tibia. The laboratory analysis revealed normal levels of calcium and parathyroid hormone, high levels of alkaline phosphatase, hypokalemia and hypophosphatemia (Table 1). The hypothesis of osteomalacia induced by mesenchymal tumor phosphatonin producer was raised. The research of FGF-23 was not performed, since this test is quite expensive in Brazil, only being quantified in research protocols a few centers

The patient did a Photon Emission Computed Tomography/Computed Tomography (SPECT/CT) to locate the tumor (Fig. 1). The SPECT/CT reconstruction presented multiples fracture areas

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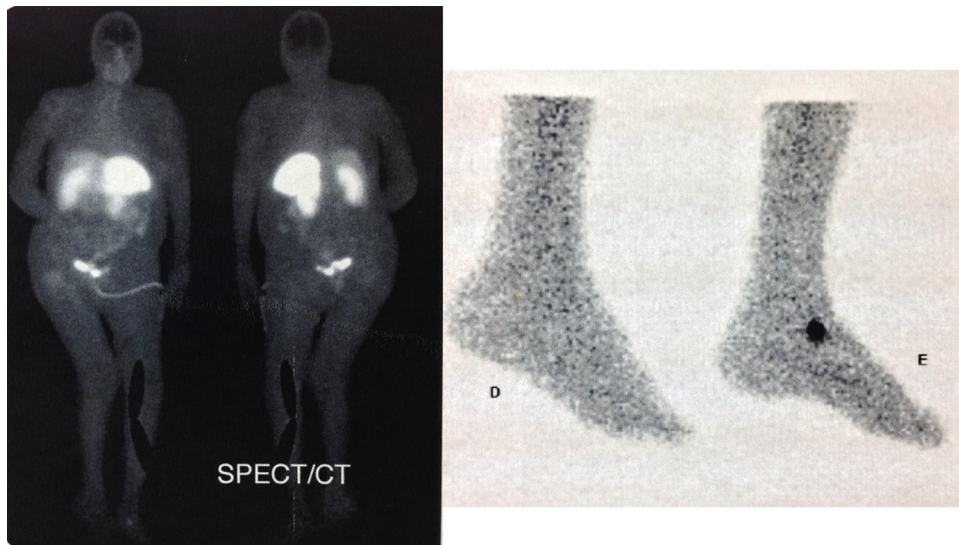


Fig. 1. SPECT/CT.



Fig. 2. SPECT/CT and Reconstruction.

and a hypercaptation in left tibial articular plateau, measuring about 0.8 cm (Fig. 2).

The tumor was surgically removed with free margins (Fig. 3), revealing a glandular aspect in macroscopy (Fig. 4) and mes-

enchymal aspect in microscopy pathology analysis, leading to the diagnosis of oncogenic hypophosphatemic osteomalacia. At any moment was measured calcitonin since have been no clinical decompensation of the patient.

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