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ART IN THE WARD

Nephrology picture: bone loss due to absence of adequate therapy for severe secondary hyperparathyroidism

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A 43-year-old black female was referred to our service in 2017 due to severe secondary hyperparathyroidism (SHPT). She had hypertension and had been on hemodialysis since 2010, thrice weekly for 4 h, via an arteriovenous fistula. She presented with diffuse bone pain, limited walking, requiring a wheelchair for mobility, and restricted light physical activity. She was on 0.5 µg of oral calcitriol thrice weekly.

Initially, cinacalcet 30 mg/day was started and surgical treatment was proposed, but the patient refused. Symptoms worsened with a loss of 15 cm in height and severe kyphosis. Two years later, in 2019, a plain lateral radiograph (Fig. 1A, B) and chest tomography revealed a collapsed T11 vertebra (Fig. 1E) and diffuse bone demineralization. In addition, prominent thickening with diffuse alteration of the signal of the cortical bones of the femurs and muscle atrophy was shown by thigh magnetic resonance imaging (Fig. 1F). Cinacalcet dose was increased to 90 mg/day, but laboratory tests pointed to advanced hyperparathyroidism. Due to the lack of clinical response to drug treatment, the patient agreed to undergo parathyroidectomy (PTX) and surgery was scheduled. However, due to the limited number of hospital beds during the coronavirus pandemic, the procedure was postponed. Densitometry was repeated in September 2020, with a significant 18.9% and 9% loss in bone density of the total hip and femoral neck sites, respectively (Sup. Table 1B).

In October 2020 she underwent total PTX with parathyroid autotransplantation on the forearm. Whole-body dual-energy x-ray absorptiometry was repeated in March 2021 and in April 2022. She presented a remarkable bone mineral content increase of 17.6% and 44% at 6 and 18 months, respectively (Sup. Fig. 1).

SHPT is a common morbid condition in patients with chronic kidney disease (CKD). In CKD patients, PTH acts as a uremic toxin due to its deleterious effects on several systems [1]. In the context of advanced SHPT, the compartment of the cortical bone, which is responsible for skeletal resistance, is more severely affected than the trabecular bone [2]. PTX is an effective therapeutic procedure for advanced SHPT that does not respond to clinical treatment. The improvement of symptoms occurs shortly after surgery, and some harmful effects, such as soft-tissue calcification and cardiovascular complications, may be reversible [3, 4]. In this case, the delayed surgical procedure resulted in considerable bone mineral density (BMD) decline in the year prior to PTX, as well as the development of bone deformities, fractures, and worsening of functional capacity. A significant worsening in total BMD (− 7%) was noted, mainly in the hip, which is a cortical bone site. Despite the improvement in functionality after PTX, the patient has irreversible sequelae, including bone deformities and collapsed vertebrae. Although uncommon, these complications still exist, due to the lack of adequate therapy and stress the need for close and early control of SHPT.

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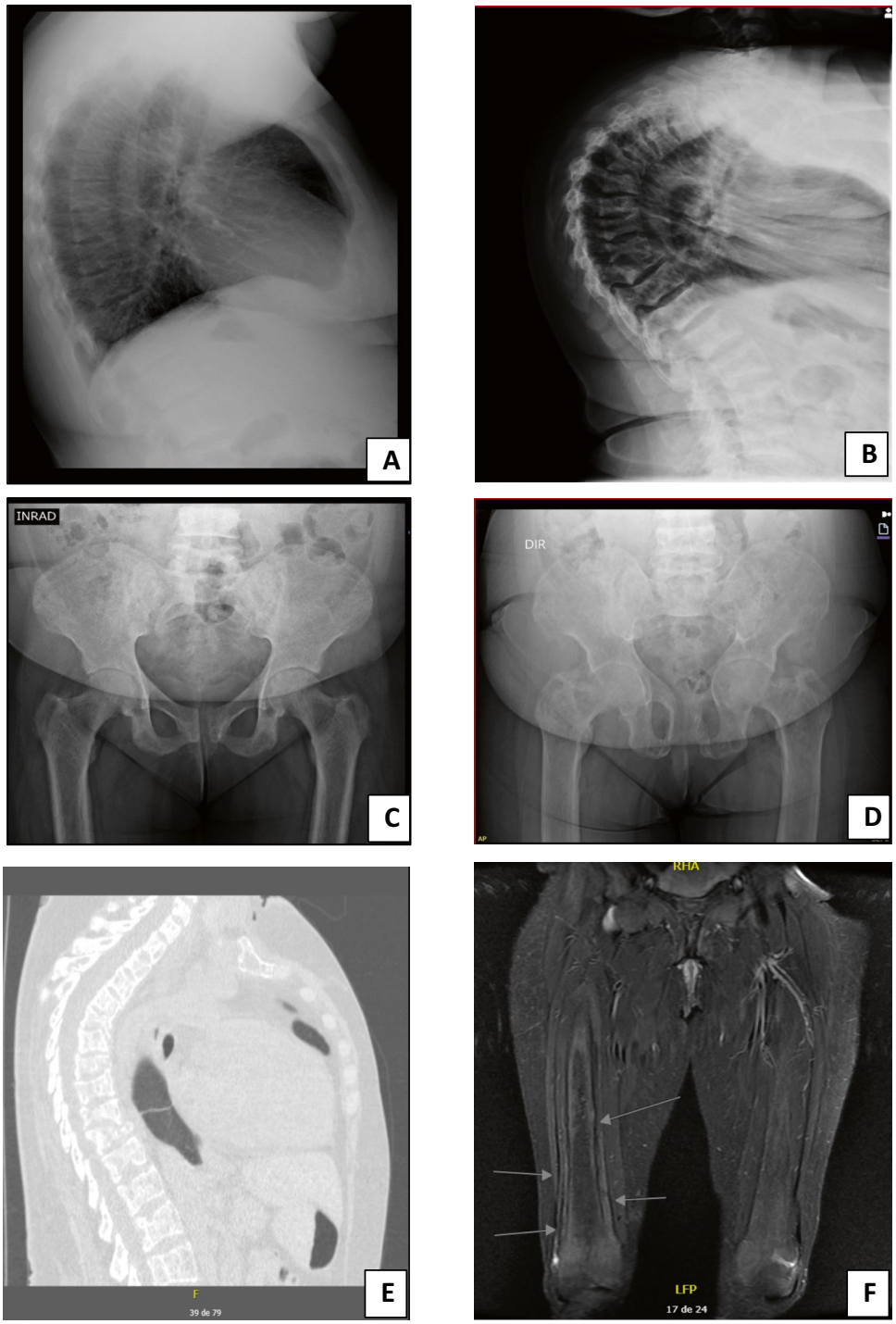

Fig. 1 Imaging studies of the evolution of SHPT without adequate therapy. **A**, **B** Plain chest x-rays revealing marked kyphosis due to collapse of the vertebrae after loss to follow-up. **C**, **D** Hip radiographs show development of brown tumor and pubic symphysis diastasis. **E** Chest tomography. **F** Magnetic resonance imaging

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Declarations

Conflict of interests The authors declare that they have no conflict of interests.

Ethical approval The patient was enrolled in a protocol approved by the Comitê de Ética em Pesquisa do Hospital das Clínicas da Faculdade de Medicina da USP (CAPPesq 87886218.7.0000.0068).

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