Cureus
Part of SPRINGER NATURE

Open Access Case Report

Bell-Shaped Thorax in Severe Renal Osteodystrophy: A Case Report

Lucca C. de Oliveira [1], Ludmila L. de Moraes [2], Vitor B. de Souza [1]

1. Internal Medicine, Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo, São Paulo, BRA 2. Medicine, Centro Universitário FAMINAS (UNIFAMINAS), Muriaé, BRA

Corresponding author: Lucca C. de Oliveira, luccamarinho@gmail.com

Review began 11/05/2026
Review ended 11/16/2026
Published 11/26/2026

© **Copyright** 2026
de Oliveira et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.120298

Abstract

Thoracic cage deformity is a rarely reported skeletal complication of chronic kidney disease-mineral and bone disorder (CKD-MBD). We present the case of a 26-year-old woman on maintenance hemodialysis, with a previous 90-cm small bowel resection and end ileostomy, who had severe hyperparathyroidism refractory to conservative measures, with parathyroid hormone (PTH) levels above 4,000 pg/mL. She presented with disabling bone pain, a reported height loss of approximately 10 cm, markedly reduced bone mineral density, and an atraumatic femoral neck fracture. Preoperative imaging had already demonstrated sequelae of rib fractures and accentuated spinal curvatures. She underwent total parathyroidectomy with forearm autotransplantation, which was complicated by hungry bone syndrome. Histopathology revealed four-gland hyperplasia. Seven weeks later, she sustained an atraumatic contralateral femoral fracture and became wheelchair-dependent. Seven months after surgery, despite biochemical control, radiography and computed tomography demonstrated a bell-shaped thorax with verticalized, crowded ribs, and spirometry showed reduced FVC with a preserved FEV1/FVC ratio, suggestive of restriction. Hyperparathyroidism was the most likely dominant driver, with a possible contribution from intestinal malabsorption. This case illustrates that thoracic deformity may develop in long-standing, inadequately controlled hyperparathyroidism as a late marker of advanced disease and may persist despite parathyroidectomy and a marked reduction in PTH levels. Thus, the opportunity for intervention lies earlier, in timely biochemical control and referral for parathyroidectomy when hyperparathyroidism is refractory to medical therapy.

Categories: Radiology, Nephrology, Endocrinology/Diabetes/Metabolism
Keywords: bell-shaped thorax, chest wall deformity, ckd-mbd (chronic kidney disease-mineral and bone disorder), hyperparathyroidism, renal osteodystrophy

Introduction

Chronic kidney disease-mineral and bone disorder (CKD-MBD) is a systemic disorder in which declining kidney function disrupts the regulation of calcium, phosphate, vitamin D, and parathyroid hormone (PTH), leading to abnormalities in bone and mineral metabolism and vascular calcification [1]. Its skeletal component, renal osteodystrophy, comprises alterations in bone morphology caused by these disturbances [1]. When hyperparathyroidism remains uncontrolled for years, high-turnover bone disease, in which accelerated and disorganized remodeling produces fragile bone, can cause bone pain, fractures, height loss, and progressive skeletal deformity. Its most extreme expression is Sagliker syndrome, a rare and disfiguring condition characterized chiefly by craniofacial deformity and described mostly in young patients with prolonged, inadequately treated disease [2,3].

Deformity of the thoracic cage has received far less attention. In a five-year radiographic follow-up study of patients on regular hemodialysis, about one quarter developed thoracic cage deformity, which was associated with PTH and alkaline phosphatase (ALP) levels and reduced vital capacity. The changes were mostly mild, and some were detectable only by comparing serial radiographs [4]. Since then, only isolated reports have described the bell-shaped thorax, in which the chest is narrow superiorly and widens toward its base, in severe renal osteodystrophy, occasionally accompanied by restrictive ventilatory impairment [5-7]. We report an extreme thoracic manifestation of this common disorder, a marker of advanced, long-standing disease, in a young woman on hemodialysis with severe hyperparathyroidism in whom thoracic deformity documented before parathyroidectomy persisted after biochemical control, with spirometric findings suggestive of restriction. This report follows the CARE guidelines [8].

Case Presentation

Background

A 26-year-old woman was referred to our tertiary center for evaluation of severe hyperparathyroidism. At 15 years of age, she had undergone emergency laparotomy for a perforated acute abdomen, with resection of 90 cm of small bowel and creation of an end ileostomy, which remains in place. Crohn's disease was presumed but never confirmed, and colonoscopies found no abnormalities. Renal asymmetry noted during that operation prompted referral to nephrology, where she was diagnosed with advanced CKD of

undetermined cause. She had a history of recurrent urinary tract infections in childhood and no nephrolithiasis, and kidney biopsy was not performed. She was managed conservatively for approximately eight years before starting maintenance hemodialysis three times weekly. She also had hypothyroidism treated with levothyroxine.

According to the patient, she was referred for parathyroidectomy shortly after starting dialysis because of hyperparathyroidism refractory to clinical management, but she was evaluated at our tertiary center only approximately three years later. Records from her dialysis unit approximately two years before referral showed PTH around 2,000 pg/mL, ALP 840 U/L, and serum phosphorus of 7.0-9.5 mg/dL. Treatment had included sevelamer, calcium carbonate, cholecalciferol 25,000 IU twice weekly, and calcitriol; cinacalcet 30 mg/day was discontinued because of intolerance.

Clinical presentation and investigation

At referral, she reported severe bone pain, predominantly in the hips (10/10 on a visual analog scale), requiring methadone and morphine, and progressive difficulty walking. She reported a height loss of approximately 10 cm (from 161 to 150 cm) and a previous shoulder fracture after a fall. According to the patient, she had no skeletal deformity until the year before referral, when she noticed progressive changes, particularly in her hands. Examination showed broadening and shortening of the distal phalanges, most evident in the thumbs, with bulbous fingertips and short, wide nails, a pattern suggestive of pseudoclubbing due to acro-osteolysis; hand radiographs were not obtained. Laboratory tests showed PTH above 4,000 pg/mL (reference range, 28-129 pg/mL), total calcium 9.8 mg/dL (9.0-10.4 mg/dL), and phosphorus 7.3 mg/dL (2.5-4.5 mg/dL). Bone densitometry (dual-energy X-ray absorptiometry) of the right hip showed a femoral neck bone mineral density of 0.393 g/cm² and a total hip bone mineral density of 0.444 g/cm² (Z-score −4.1 at both sites). One month later, she sustained an atraumatic right femoral neck fracture while climbing stairs, confirmed by magnetic resonance imaging. Because of poor bone quality, she was not considered a surgical candidate, and the fracture was managed conservatively.

Technetium-99m sestamibi scintigraphy showed four foci of parathyroid uptake, one of them in the superior mediastinum, together with skeletal uptake in the calvarium, mandible, and long bones. Neck CT confirmed four enlarged parathyroid glands and showed diffuse mixed lytic and sclerotic bone changes, deformities and sequelae of rib fractures, accentuated cervical lordosis and thoracic kyphosis, and an expanded mandible with cortical disruption and condylar deformity. These craniofacial changes were apparent only on imaging, without visible facial deformity. Intravenous paricalcitol was added at each dialysis session. In the two months before surgery, PTH remained above 3,000 pg/mL, ALP rose to 1,868 U/L (44-107 U/L), total calcium was 9.6 mg/dL, ionized calcium was 4.58 mg/dL (4.49-5.29 mg/dL), phosphorus was 7.7 mg/dL, and 25-hydroxyvitamin D was 36.9 ng/mL (laboratory reference for patients with CKD, 30-60 ng/mL).

Surgical treatment

Approximately six months after referral, she underwent total parathyroidectomy with autotransplantation of parathyroid tissue into the forearm. Intraoperative PTH fell from a pre-excision peak of 2,940 pg/mL to 273 pg/mL at 20 minutes, a decline of more than 90%. Within 24 hours, total calcium fell to 6.7 mg/dL, with PTH of 11 pg/mL. She developed hungry bone syndrome, with phosphorus as low as 1.3 mg/dL and ALP rising above 4,500 U/L, and required continuous intravenous calcium together with high-dose oral calcium carbonate and calcitriol. Histopathology showed four parathyroid glands measuring 1.5 to 3.0 cm, with nodular proliferation of oxyphil and chief cells favoring hyperplasia and no features of malignancy; the inferior glands were removed en bloc with thymic tissue.

Follow-up and outcome

Seven weeks after surgery, with PTH below 6 pg/mL, she sustained an atraumatic left femoral fracture, which was also managed conservatively; since then, she has used a wheelchair. During calcium and calcitriol repletion, she had transient episodes of hypercalcemia (peak total calcium 11.2 mg/dL; peak ionized calcium 6.11 mg/dL). PTH remained between <6 and 23 pg/mL during the first four postoperative months.

Six months after surgery, she was hospitalized for an unrelated ischemic stroke, from which she recovered without neurological sequelae. During that admission, her bone pain was controlled without regular opioids, which she used only before assisted ambulation during physiotherapy. Chest radiography showed verticalized, crowded ribs, a bell-shaped configuration of the thorax, and diffuse osteopenia (Figure *1*). Chest CT showed the ribs stacked along the lateral chest walls on coronal images and clustered at a single level on axial images, as well as lytic lesions of the humeral heads (Figure *2*).

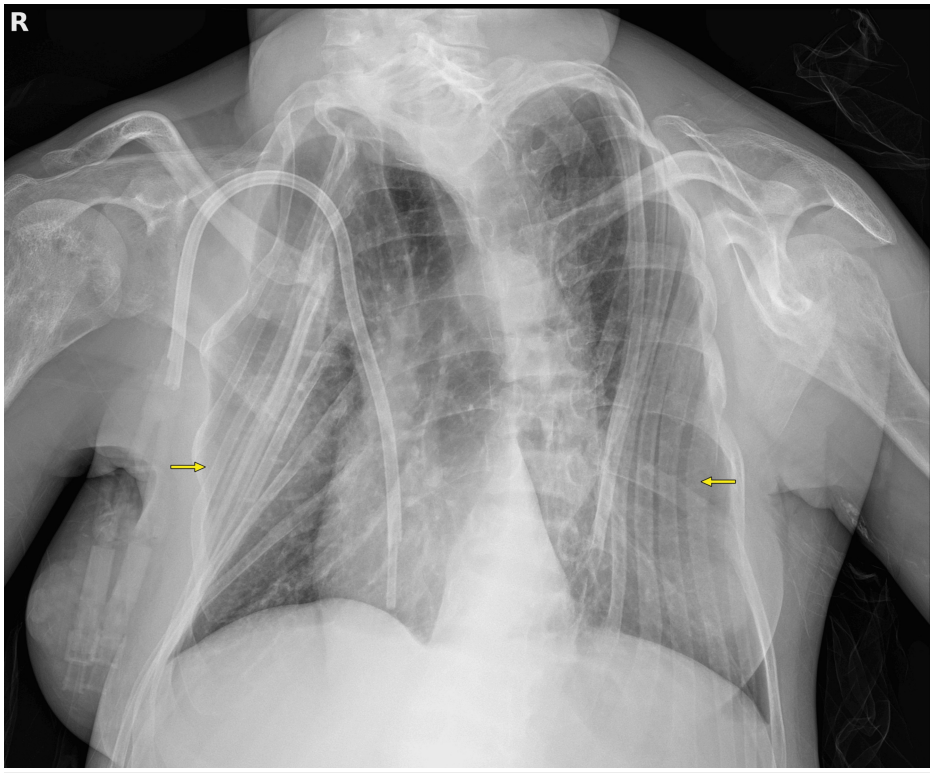


FIGURE 1: Chest radiograph showing a bell-shaped thorax

Anteroposterior chest radiograph approximately seven months after parathyroidectomy, showing verticalized, crowded ribs along both lateral chest walls (arrows), with a bell-shaped configuration of the thorax and diffuse osteopenia. A tunneled hemodialysis catheter is seen in the right internal jugular vein

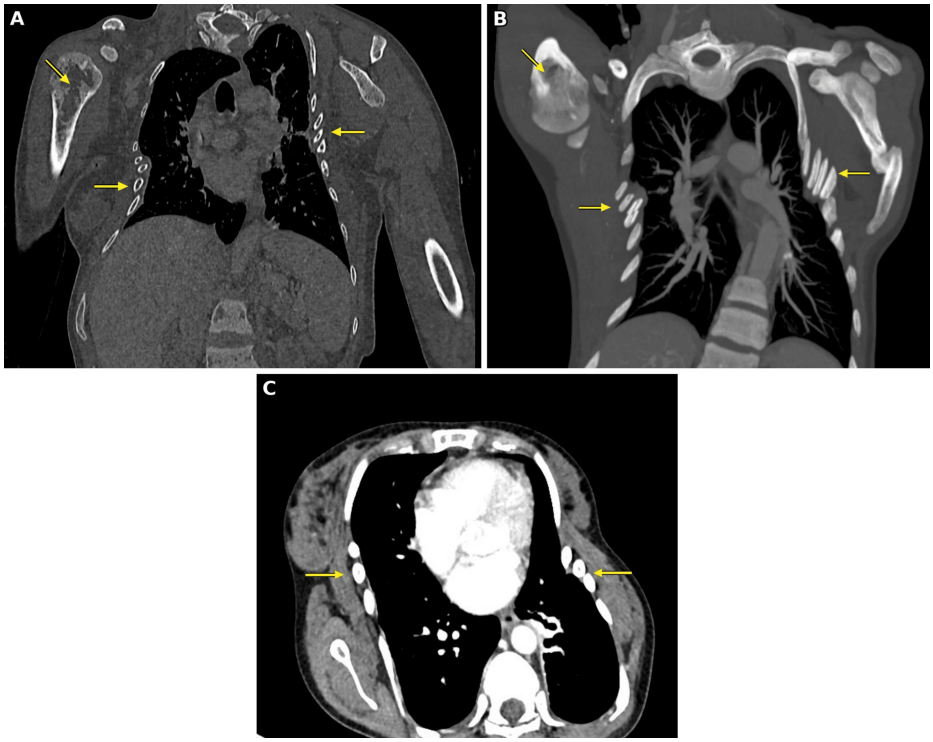


FIGURE 2: Chest CT showing verticalized, crowded ribs

Images obtained approximately six to seven months after parathyroidectomy. (A) Coronal image, bone window, and (B) coronal maximum-intensity projection: the ribs appear in cross-section, stacked along both lateral chest walls (horizontal arrows), indicating their vertical orientation; lytic lesion of the right humeral head (oblique arrows). (C) Axial image: several ribs are clustered along each lateral chest wall at a single level (arrows)

CT: computed tomography

Three-dimensional reconstructions demonstrated the bell-shaped thoracic cage with verticalized ribs (Figure *3*). Cranial CT showed a heterogeneous, mottled calvarium with irregular thickening of the diploë, and reconstructions of the pelvis showed multiple lytic lesions of the iliac bones and deformity of both proximal femora (Figure *4*). Spirometry of acceptable quality (grade A) showed a forced vital capacity (FVC) of 1.77 L (55% of predicted; lower limit of normal, 2.60 L), a forced expiratory volume in one second (FEV1) of 1.49 L (53% of predicted), and a preserved FEV1/FVC ratio of 0.84. Post-bronchodilator testing and lung volume measurements were not performed, and the pattern was considered suggestive of restriction. Predicted values were calculated using her current height of 150 cm; because height was reduced by the deformity, the percentages of predicted values may underestimate the impairment. She reported no dyspnea, although her exertion was limited by wheelchair dependence.

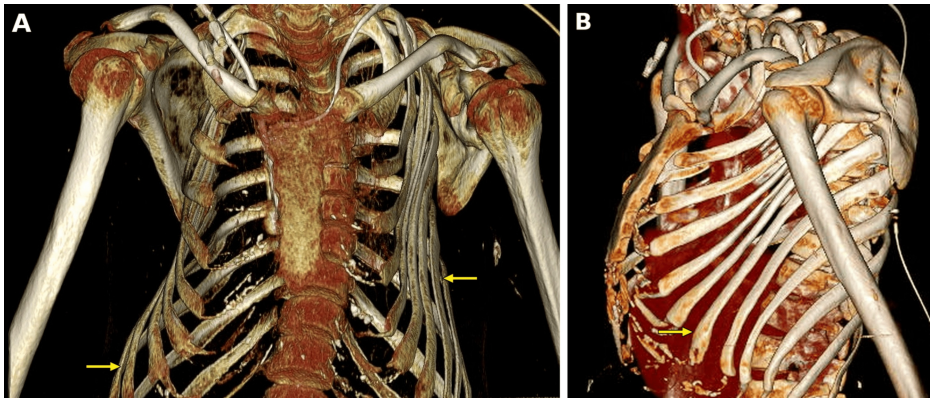


FIGURE 3: Three-dimensional reconstructions of the thoracic cage

Volume-rendered reconstructions obtained approximately six months after parathyroidectomy. (A) Frontal view showing verticalized, crowded ribs along the lateral chest walls, with the lower ribs flaring toward the base, giving the thorax its bell-shaped configuration (arrows). (B) Lateral oblique view showing steeply inclined, crowded ribs; the arrow indicates a rib with an irregular, widened anterior end

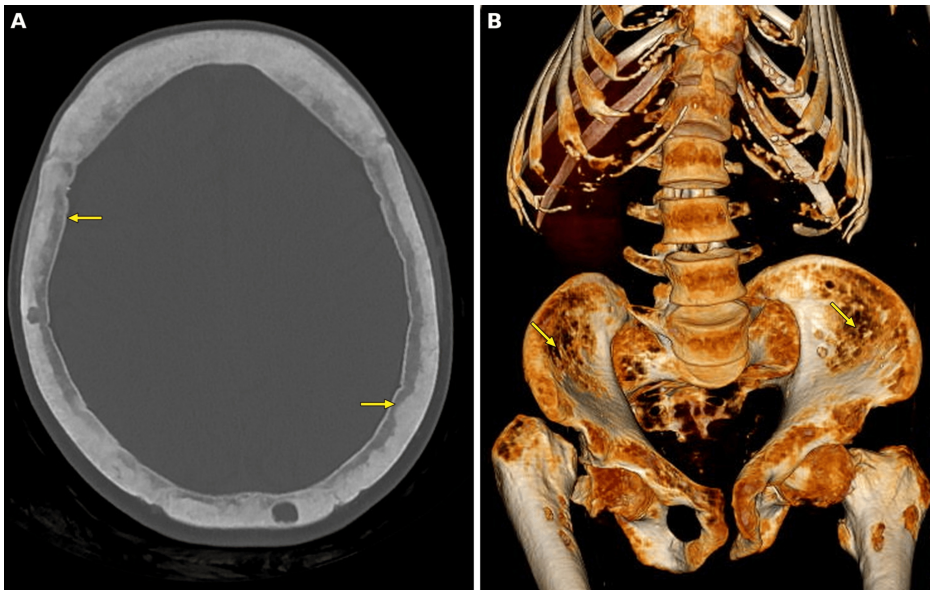


FIGURE 4: Skeletal involvement beyond the thorax

(A) Axial cranial CT (bone window) showing a heterogeneous, mottled calvarium with irregular thickening of the diploë (arrows). (B) Three-dimensional reconstruction of the lumbar spine and pelvis showing multiple lytic lesions of the iliac bones (arrows) and deformity of both proximal femora

CT: computed tomography

Nine months after surgery, PTH was 49-67 pg/mL, ALP was 327-379 U/L, calcium was within the reference range, phosphorus was 2.6 mg/dL, and 25-hydroxyvitamin D was 30.5 ng/mL, consistent with autograft function. The hand deformities persisted (Figure *5*). She remains wheelchair-dependent and is awaiting orthopedic evaluation for total hip arthroplasty.



FIGURE 5: Hand changes suggestive of acro-osteolysis

Dorsal view of both hands approximately nine months after parathyroidectomy, showing broadening and shortening of the distal phalanges, most evident in the thumbs, with bulbous fingertips and short, wide nails, a clinical appearance suggestive of acro-osteolysis (pseudoclubbing). Hand radiographs were not obtained

Discussion

This case documents a bell-shaped thorax in a young woman on hemodialysis with long-standing, severe hyperparathyroidism. Two features deserve emphasis: thoracic deformity and rib fracture sequelae were already present on imaging before parathyroidectomy and persisted after biochemical control, and spirometry showed a pattern suggestive of restriction. Thoracic cage deformity in dialysis patients was described decades ago. Ohnishi reported it in 25.9% of 58 patients on regular hemodialysis followed for five years and found associations with PTH, ALP, kyphosis, height loss, and vital capacity; even in the most affected group, however, mean height loss was 2 cm, considerably less than in our patient [4]. Contemporary reports are scarce and mostly involve patients with prolonged, inadequately treated disease, often in the context of Sagliker syndrome [2,3,5-7]. CT showed mandibular expansion with cortical disruption, but there was no clinically apparent facial deformity, which is the defining feature of Sagliker syndrome [2,3].

Govindu and Ammar attributed the bell-shaped thorax in severe renal osteodystrophy to osteitis fibrosa combined with vertebral and rib fractures [5]. Our findings are consistent with this explanation: markedly elevated PTH and ALP, diffuse skeletal uptake on scintigraphy, and preoperative CT showing rib fracture sequelae and accentuated kyphosis. Focal lytic lesions, such as those in the humeral heads, may correspond to brown tumors, a recognized skeletal manifestation of hyperparathyroidism [9]. Without bone biopsy, however, neither these lesions nor the underlying histological pattern can be characterized.

A bell-shaped thorax is classically associated with rickets, and the radiological findings of rickets, osteomalacia, and renal osteodystrophy overlap considerably [9]. The same configuration has been described in primary hyperparathyroidism due to parathyroid carcinoma [10]. In our patient, the biochemical profile, four-gland hyperplasia on histopathology, and the clinical course point to hyperparathyroidism as the main driver. We described the disease as severe hyperparathyroidism refractory to conservative measures rather than as secondary or tertiary, because preoperative calcium was normal and records from earlier years were incomplete.

Chest wall deformity can impair ventilation. Yaxley and Scott described respiratory failure attributed to thoracic deformity in CKD-MBD, with FVC reduced to 18% of predicted [6]. Our patient's reduced FVC with a preserved FEV1/FVC ratio may reflect her thoracic deformity, but this interpretation has limits: lung volumes were not measured, and kyphosis, muscle weakness, and deconditioning after prolonged immobility and hospitalization may also have reduced FVC.

Current Kidney Disease: Improving Global Outcomes (KDIGO) guidance suggests parathyroidectomy for patients with CKD G3a-G5D and severe hyperparathyroidism who fail to respond to medical or pharmacological therapy [1]. A recent KDIGO Controversies Conference highlighted data from the PROCEED study showing greater gains in bone mineral density with parathyroidectomy than with calcimimetic therapy, as well as Japanese registry data associating parathyroidectomy with lower mortality than calcimimetics [11]. In our patient, cinacalcet was not tolerated, and approximately three years elapsed

between the initial referral and surgery. The postoperative course, with severe hungry bone syndrome and a new fracture seven weeks after surgery, illustrates the vulnerability of a severely demineralized skeleton in the early postoperative period.

Established skeletal deformities may persist after parathyroidectomy. Govindu and Ammar noted persistence of thoracic deformity after parathyroidectomy [5], and Luchi et al. reported a Brazilian patient whose thoracic deformity and height loss persisted after surgery [7]. Reports of Sagliker syndrome likewise describe skeletal deformities as largely irreversible and point to delayed treatment as a contributing factor [2,3]. In our patient, the deformities documented before surgery remained evident seven months after parathyroidectomy, despite a marked fall in PTH and ALP. The hand changes, although not confirmed radiographically, resemble the pseudoclubbing caused by acro-osteolysis in severe hyperparathyroidism, which has been reported to persist after treatment [5,12].

This report has limitations. Records from the years preceding referral were incomplete, height values were reported rather than measured with a standardized method, bone biopsy was not performed, and pulmonary function was assessed by spirometry alone. Thoracic imaging with the same technique before and after surgery was not available for direct comparison.

Conclusions

This case illustrates an unusual thoracic manifestation of severe CKD-MBD in a patient on maintenance hemodialysis with long-standing hyperparathyroidism. A bell-shaped thorax was documented seven months after parathyroidectomy, despite a marked reduction in PTH levels, and spirometry showed a pattern suggestive of ventilatory restriction. The case highlights the extent of skeletal involvement that may be encountered in patients with severe hyperparathyroidism.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Lucca C. de Oliveira, Ludmila L. de Moraes, Vitor B. de Souza

Acquisition, analysis, or interpretation of data: Lucca C. de Oliveira, Ludmila L. de Moraes

Drafting of the manuscript: Lucca C. de Oliveira, Ludmila L. de Moraes

Critical review of the manuscript for important intellectual content: Vitor B. de Souza

Supervision: Vitor B. de Souza

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **AI use statement:** The authors used Claude (Anthropic) as follows: language polishing, translation, structure or formatting, extensive copy editing or writing support, drafting summaries, literature comparison in Entire manuscript text (title, abstract, introduction, case presentation, discussion, conclusions, additional information and figure legends): translation from Portuguese to English, language polishing, extensive copy editing and writing support, and suggestions on structure and formatting (including adaptation to the CARE checklist and to journal reference style). No AI use in data acquisition, data analysis, image generation or image processing. All manuscript content was originally conceived and written by the authors in Portuguese from the patient's medical records. The AI tool was used to translate and edit the text into English. All clinical data, dates, laboratory values and imaging findings were checked by the authors against the original medical records and imaging studies. All references were identified by the authors, and each citation was verified against its source (DOI/PubMed) to confirm that it exists and supports the statement made. All interpretations and conclusions are the authors' own. The final text was reviewed and approved by all authors, who take full responsibility for its content. The authors confirm that they have reviewed and verified all AI-assisted content and take full responsibility for the work. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. KDIGO 2017 clinical practice guideline update for the diagnosis, evaluation, prevention, and treatment of chronic kidney disease-mineral and bone disorder (CKD-MBD). Kidney Int Suppl (2011). 2017, 7:1-59. 10.1016/j.kisu.2017.04.001
2. Morantes Salazar MM, Salazar Florez JC, Jacome-Pérez N, Arenas Borda WD, Lara Rodríguez CA: Severe manifestation of hyperparathyroidism with Sagliker syndrome: a case report. Cureus. 2026, 18:e101753. 10.7759/cureus.101753
3. Erol Ç, Şahin Ö, Şen AE, Aydın Z: Bone and parathyroid scintigraphy findings in Sagliker syndrome . Mol Imaging Radionucl Ther. 2024, 33:40-2. 10.4274/mirt.galenos.2023.24382
4. Ohnishi Y: Thorax cage deformity in patients under regular hemodialysis . J Bone Miner Metab. 1986, 4:40-9. 10.11393/jbmm1983.4.40
5. Govindu R, Ammar H: Thoracic deformity and acro-osteolysis in severe renal osteodystrophy . Mayo Clin Proc. 2019, 94:2358-9. 10.1016/j.mayocp.2019.08.004
6. Yaxley J, Scott T: Respiratory failure: a rare complication of chronic kidney disease-mineral and bone disorder. Ochsner J. 2019, 19:282-5. 10.31486/toj.18.0177
7. Luchi WM, Vianna JGP, Roberto LEV, Villas Boas EA, Costalonga EF: Uremic leontiasis ossea. Endocrine. 2019, 65:707-9. 10.1007/s12020-019-01976-z
8. Riley DS, Barber MS, Kienle GS, et al.: CARE guidelines for case reports: explanation and elaboration document. J Clin Epidemiol. 2017, 89:218-35. 10.1016/j.jclinepi.2017.04.026
9. Chang CY, Rosenthal DI, Mitchell DM, Handa A, Kattapuram SV, Huang AJ: Imaging findings of metabolic bone disease. Radiographics. 2016, 36:1871-87. 10.1148/rg.2016160004
10. Kaszczewska M, Popow M, Chudziński W, et al.: A woman with a 27-year history of hyperparathyroidism and hypercalcemia who was diagnosed with low-grade parathyroid carcinoma. Am J Case Rep. 2021, 22:e930301. 10.12659/AJCR.930301
11. Ketteler M, Evenepoel P, Holden RM, et al.: Chronic kidney disease-mineral and bone disorder: conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference. Kidney Int. 2025, 107:405-23. 10.1016/j.kint.2024.11.013
12. Pal R, Dua A, Bhadada SK: Acro-osteolysis in primary hyperparathyroidism . CMAJ. 2024, 196:E1207. 10.1503/cmaj.240818