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Severe bone loss in a premenopausal woman

ABSTRACT

A 47-year-old female presented after osteoporotic fracture of the left femoral neck 6 months earlier. Hip joint alloplasty was performed. In the medical history there was epilepsy, chronic kidney disease and tophaceous gout. At the visit the patient complained about weight loss, lack of appetite, muscle weakness. On examination there was no swollen joints, 38 tender joints, no tophi. She had telescopic fingers, her hand and feet x-rays revealed massive geodes and bone erosions. In laboratory tests parathormone was 1876 pg/mL (normal is 10

do 60 pg/mL), vitamin D 8 ng/mL, Ca 4.5 mmol/L. The aim of the article is to discuss abnormalities in radiological images and laboratory tests of this patient. Medical history of the patient was analysed, 7 references from PubMed database and one book were reviewed in September 2023.

Secondary hyperparathyroidism in a patient with chronic kidney disease was diagnosed. The patient was consulted by an endocrinologist and qualified for parathyroidectomy.

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KEY WORDS: bone loss; fracture; secondary hyperparathyroidism

CASE PRESENTATION

A 47-year-old female presented after osteoporotic fracture of the left femoral neck 6 months earlier. Hip joint alloplasty was performed. In the medical history there was epilepsy, chronic kidney disease and tophaceous gout. At the visit the patient complained about weight loss (10 kg/last year), lack of appetite, muscle weakness. On examination there was no swollen joints, 38 tender joints, no tophi. She had telescopic fingers (V of her right hand, II and III of her left hand). Her hand and feet x-rays revealed massive geodes and bone erosions (Fig. 1). Laboratory examination showed: negative rheumatoid factor (RF), anti-cyclic citrullinated antibodies < 5 U/mL, uric acid 6 mg/dL, creatinine 2.18 mg/dL, hemoglobin 7.9 g/dL, iron (Fe) 11 ug/dL, parathormone (PTH) 1876 pg/mL (normal is 10–60 pg/mL), vitamin D 8 ng/mL, calcium (Ca) 4.5 mmol/L. In abdominal ultrasound nephrocalcinosis was visualized. Anemia, nephrocalcinosis and bone loss are features of secondary hyperparathyroidism in a patient with chronic

kidney disease [1]. The patient was consulted by an endocrinologist and qualified for parathyroidectomy. She uses alfacalcidol 1 µg/day and febusostat 80 mg/day.

The aim of the article is to discuss abnormalities in radiological images and laboratory tests of this patient.

Medical history of the patient was analyzed and 7 references from PubMed database as well as one book were reviewed in September 2023.

DISCUSSION

Progressive reduction in glomerular filtration rate in chronic kidney disease increases plasma phosphate levels, leading to a decrease in calcium levels. To compensate these disturbances, parathyroid cells secrete PTH, which inhibits tubular phosphate reabsorption. The new equilibrium is achieved by increasing the concentration of PTH. Kidney damage leads to a reduction in ability to hydroxylate vitamin D and a reduction in the formation of its active form 1,25 (OH). In chronic kidney

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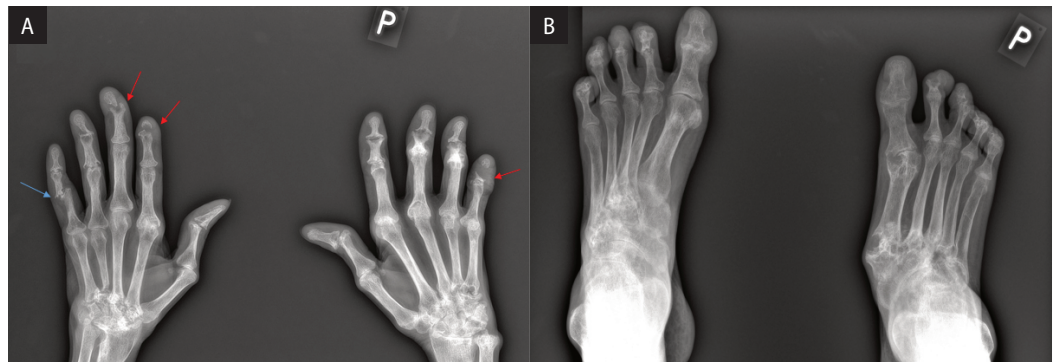


Figure 1. X-rays of the hands (A) and feet (B) of the patient with severe secondary hyperparathyroidism. A pseudo-joint in the fifth finger of the left hand (A). Partial erasure of the bone structure of the distal phalanx of the fifth finger of the right hand and of the second and third fingers of the left hand (A)

disease, the number of receptors that mediate the effects of vitamin D on tissues decreases. Lowering the concentration of 1,25 (OH) vitamin D leads to a reduction in its inhibitory effect on PTH production, a decrease in intestinal calcium absorption, hypocalcemia, and thus an increase in PTH concentration [2]. Parathyroidectomy is a treatment of choice in case of severe secondary hyperparathyroidism [3]. Physicians should classify 25(OH)D concentration as adequate if > 20 ng/mL without elevated PTH. The concentrations of 25(OH)D < 15 ng/mL should be treated irrespective of PTH level. Patients with 25(OH)D 15–20 ng/mL may not require treatment if there is no evidence of counter-regulatory hormone activity [4].

Secondary hyperparathyroidism may present in various forms with osteoporosis being the most common one. In this patient the main radiographic feature typical for secondary hyperparathyroidism is subperiosteal resorptions at the middle phalanges of the 2nd and 3rd fingers. High turnover bone disease (osteitis fibrosa) may occur in the course of secondary hyperparathyroidism in chronic kidney disease (CKD), when increased serum PTH levels are able to overcome skeletal PTH resistance. The diagnosis of secondary uremic hyperparathyroidism and osteitis fibrosa relies mainly on serum biochemistry. X-ray and other imaging methods of the skeleton provide diagnostically relevant information only in severe forms [5].

Nephrocalcinosis may be associated with secondary hyperparathyroidism, but is un-

usual. Nephrocalcinosis demonstrates characteristic deposition of calcium in the kidney parenchyma and tubules. It can be incidentally detected in a radiograph from a patient with normal kidney function or present with an acute or CKD [6].

The erosive joint diseases may be enhanced by the hyperparathyroidism. In part, some of the joint abnormalities seem to be due to tophaceous gout, e.g., one greater toe. The majority of the joint findings in this patient would most appropriately fit to psoriasis. Psoriatic arthritis may be diagnosed even if no skin abnormalities are observed [7]. The patient was an only daughter without siblings. Her parents passed many years ago and there was no documented family history of psoriasis. Hyperuricemia is often associated with psoriasis, thus supporting this suspected diagnosis [8]. Additional information might be gained from images of the spine, dual-energy computed tomography (CT) of the hands and feet but the patient did not give her consent for these diagnostic procedures.

CONCLUSION

The described abnormalities are most probably a mixture of secondary hyperparathyroidism, gout and possibly psoriatic arthritis.

CONFLICT OF INTEREST

Author declare no conflict of interest.

FUNDING

None declared.

1. Levy AR, Xing S, Brunelli SM, et al. Symptoms of Secondary Hyperparathyroidism in Patients Receiving Maintenance Hemodialysis: A Prospective Cohort Study. *Am J Kidney Dis.* 2020; 75(3): 373–383, doi: [10.1053/j.ajkd.2019.07.013](https://doi.org/10.1053/j.ajkd.2019.07.013), indexed in Pubmed: [31629575](https://pubmed.ncbi.nlm.nih.gov/31629575/).
2. Wiland P. Zmiany narządowe w chorobach reumatycznych [in Polish]. Wydawnictwo Medyczne Górnicki, Wrocław 2016.
3. Mathur A, Ahn JB, Sutton W, et al. Increasing rates of parathyroidectomy to treat secondary hyperparathyroidism in dialysis patients with Medicare coverage. *Surgery.* 2022; 172(1): 118–126, doi: [10.1016/j.surg.2022.02.005](https://doi.org/10.1016/j.surg.2022.02.005), indexed in Pubmed: [35314072](https://pubmed.ncbi.nlm.nih.gov/35314072/).
4. Melamed ML, Chonchol M, Gutiérrez OM, et al. The Role of Vitamin D in CKD Stages 3 to 4: Report of a Scientific Workshop Sponsored by the National Kidney Foundation. *Am J Kidney Dis.* 2018; 72(6): 834–845, doi: [10.1053/j.ajkd.2018.06.031](https://doi.org/10.1053/j.ajkd.2018.06.031), indexed in Pubmed: [30297082](https://pubmed.ncbi.nlm.nih.gov/30297082/).
5. Drüeke TB. Hyperparathyroidism in Chronic Kidney Disease. 2021 Oct 18. In: Feingold KR, Anawalt B, Boyce A, et al. ed. *Endotext* [Internet]. MDText, South Dartmouth (MA) 2000.
6. Vaidya SR, Yarrarapu SNS, Aeddula NR. Nephrocalcinosis. 2021 Aug 13. In: Aeddula NR. ed. *StatPearls* [Internet]. StatPearls Publishing, Treasure Island (FL) 2022.
7. Ocampo D V, Gladman D. Psoriatic arthritis. *F1000Res.* 2019; 8, doi: [10.12688/f1000research.19144.1](https://doi.org/10.12688/f1000research.19144.1), indexed in Pubmed: [31583079](https://pubmed.ncbi.nlm.nih.gov/31583079/).
8. Tripolino C, Ciaffi J, Ruscitti P, et al. Hyperuricemia in Psoriatic Arthritis: Epidemiology, Pathophysiology, and Clinical Implications. *Front Med (Lausanne).* 2021; 8: 737573, doi: [10.3389/fmed.2021.737573](https://doi.org/10.3389/fmed.2021.737573), indexed in Pubmed: [34631755](https://pubmed.ncbi.nlm.nih.gov/34631755/).