CLINICAL IMAGE

Tumoral calcinosis in the periarticular soft tissue of the left shoulder joint secondary to end-stage renal disease

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End-stage renal disease (ESRD) is associated with mineral and bone metabolism abnormalities. The hyperphosphatemic state ultimately leads to secondary and tertiary hyperparathyroidism (HPT), especially in dialysis patients. This can result in tumoral calcinosis (TC)—a condition in which calcified masses grow in the soft tissue, particularly around joints.[1,2]

We present a case of a 60-year-old woman who was on dialysis due to ESRD. She was referred to the department of endocrinology for an assessment of HPT. She has been on dialysis since 2017; ESRD developed due to polycystic kidney disease. She also had choroidal melanoma, hypertension, and atherosclerosis, and had previously undergone curative surgical treatment for colon cancer. Initial laboratory workup showed significantly increased concentrations of parathormone (PTH; 2693 pg/ml; reference range [RR], 15–65 pg/ml), total calcium (12.07 mg/dl; RR, 8.80–10.20 mg/dl), and phosphate (7.55 mg/dl; RR, 2.70–4.50 mg/dl), with a 25-hydroxyvitamin D level of 75 ng/ml (RR, 30–80 ng/ml). An ultrasound examination of the neck revealed a multinodular goiter. Two hypoechogenic lesions were visualized posterior to the inferior poles of each thyroid lobe. Scintigraphy performed after an intravenous administration of methoxyisobutylisonitrile labeled with ^{99m}Tc (^{99m}Tc-MIBI) confirmed an increased tracer uptake in these regions (FIGURE 1A and 1B). A diagnosis of tertiary HPT was made.

On admission, the patient complained of a swollen left shoulder joint. There was no history of injury. The swelling has been slowly and constantly progressing for about 6 months. Clinical examination revealed a painless, firm lesion surrounding the left shoulder joint. The pathological mass was significantly impairing the motion range of the joint. An X-ray of the affected area revealed lobular, cloudlike calcifications within the periarticular soft tissue (FIGURE 1C and 1D). The patient was consulted in the orthopedic department, where computed tomography (CT) and magnetic resonance imaging (MRI) were performed. Considering the patient's comorbidities (choroidal melanoma, colon cancer), a surgical biopsy of the pathological mass was performed. The histopathological examination revealed granulomatous tissue with giant cell granulomas and deposits of crystalline masses without evidence of malignancy. The biopsy excluded metastases of the previously diagnosed neoplasm or a new primary neoplasm (eg, chondrosarcoma). Thus, the lesion was eventually diagnosed as TC. As the patient had been previously treated with cinacalcet without improvement, she was referred for parathyroidectomy and transferred to the surgery department for further treatment.

TC is a consequence of calcium phosphate deposition. It occurs most commonly within the periarticular soft tissue; however, it may rarely be found in other localizations. The disease may be of a primary (genetic) or secondary origin. Most commonly, it is associated with ESRD and dialysis therapy; however, it may also develop due to HPT, malignancy, or hypervitaminosis D. TC is rare, affecting 0.5% to 3% of patients with ESRD. TC progresses slowly and can be asymptomatic for a long time; however, bone pain, impaired joint mobility, or neurovascular compression syndromes might eventually occur.[1-3]

An X-ray with calcium and phosphate homeostasis evaluation can often be sufficient for diagnosing secondary TC in an ESRD patient. Neck ultrasound and ^{99m}Tc-MIBI scintigraphy should be performed in HPT. Complex cases may require further diagnostics

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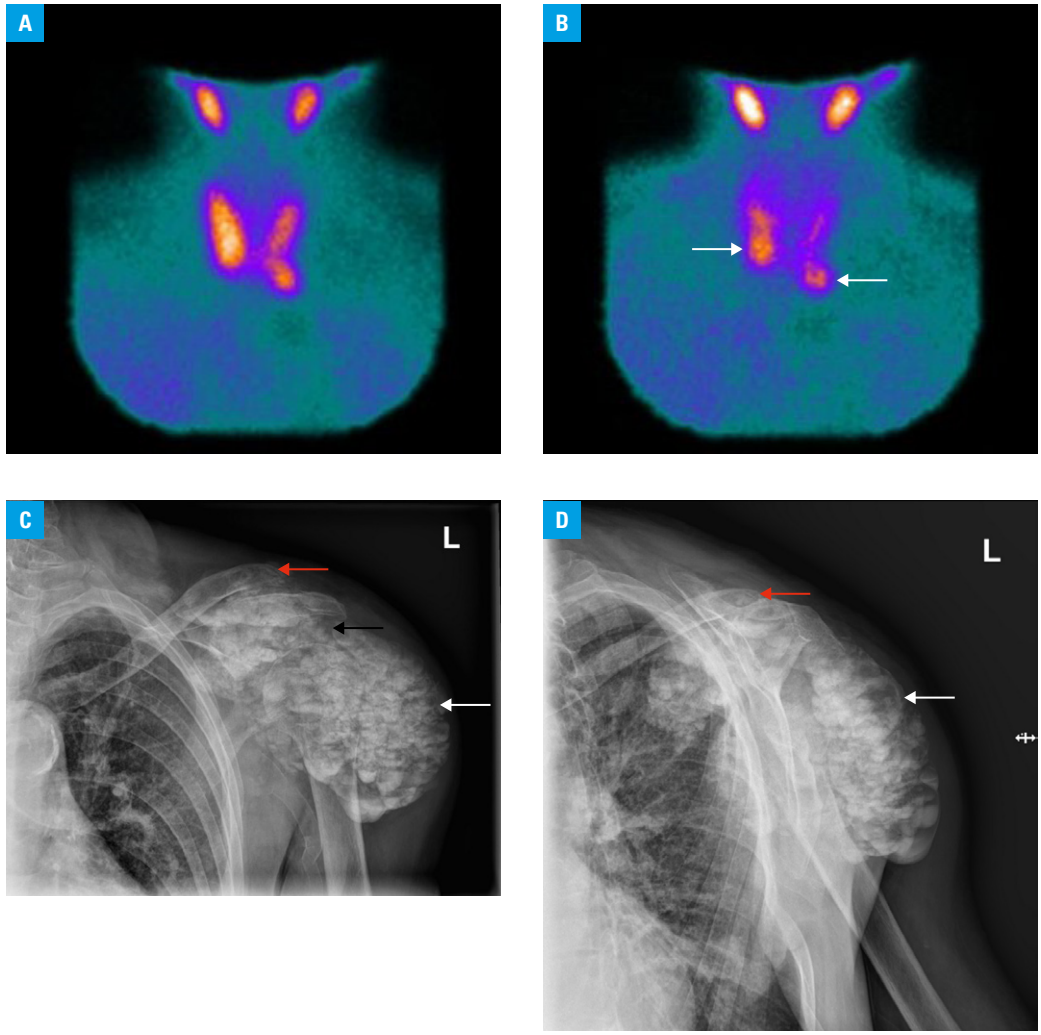

FIGURE 1 **A**, **B** – ^{99m}Tc-MIBI scintigraphy of the neck; **A** – tracer uptake by the thyroid gland in the early phase (after 20 minutes); **B** – 2 areas of increased tracer uptake corresponding to parathyroid adenomas or parathyroid hyperplasia (after 2 hours); 1 lesion (12 × 12 × 10 mm) is localized in lateral and inferior position to the inferior pole of the left thyroid lobe, the other (10 × 15 × 11 mm) can be seen in the area of the inferior pole of the right thyroid lobe (arrows). **C**, **D** – X-ray of the shoulder joint (**C** – anterior-posterior view; **D** – axial view) demonstrating lobular, cloudlike calcifications, calcium sedimentation (with calcium-fluid levels) (white arrow), dislocation of the acromial end of the clavicle (red arrow), and widening of the glenohumeral joint space (black arrow)

(CT, MRI, ^{18}F-fluoromethylcholine positron emission tomography / CT, biopsy) to exclude other pathologies.[2-5]

Treatment options differ between secondary and primary TC. Secondary TC can be managed conservatively with a low calcium and phosphate diet, bisphosphonates, and phosphate binders. However, sometimes a surgical approach (parathyroidectomy) is necessary. A renal transplant can also be curative for TC. In primary TC, an early surgical approach is indicated.[1-3]

ARTICLE INFORMATION

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