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Recurrent Inflammatory State due to Severe Periarticular Calcifications in a Patient on Hemodialysis: A Case Report

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ABSTRACT

This report highlights a severe manifestation of chronic kidney disease-mineral and bone disorder (CKD-MBD) in a hemodialysis patient: periarticular calcifications causing recurrent inflammation mimicking infection. Diagnostics excluded infections and autoimmune disorders, identifying CKD-MBD as the cause. Low-dose prednisolone effectively mitigated inflammation, reducing hospitalizations.

1 | Introduction

End-stage chronic kidney disease (CKD) and hemodialysis are linked to disruptions in mineral and bone metabolism, resulting in chronic kidney disease-mineral and bone disorder (CKD-MBD) [1]. The condition includes abnormalities in the metabolism of calcium, phosphate, parathyroid hormone (PTH), vitamin D, and fibroblast growth factor-23 (FGF23). CKD-MBD has widespread, multisystemic effects, as it not only alters bone morphology but also can cause extraskeletal calcifications in soft tissues and blood vessels [2]. Patients with CKD-MBD face a higher risk of fractures, cardiovascular issues, and increased mortality [3].

Soft tissue calcification is relatively common in chronic and end-stage kidney disease, but tumoral calcinosis—a condition marked by extensive calcium phosphate deposition in periarticular areas—occurs in only a small subset of patients [4]. In rare cases, CKD-MBD can result in such extensive calcification of periarticular tissues that it triggers a severe inflammatory response. Diagnosing this condition can be particularly challenging, as patients often present with systemic symptoms resembling those of infectious or rheumatic diseases.

The aim of this case report is to highlight a recurring inflammatory condition due to severe periarticular calcification in a patient undergoing hemodialysis.

2 | Case History

We report a case of a white male who began experiencing symptoms described in this report approximately 1 year after starting hemodialysis. The patient was 51 years old at the time. The etiology of the patient's end-stage renal disease was hydronephrosis. The patient also had chronic obstructive pulmonary disease, hepatitis C, hypertension, and hypercholesterolemia.

Approximately 1 year after initiating hemodialysis, the patient experienced multiple hospitalizations due to fever, significantly elevated C-reactive protein (CRP) levels exceeding 200 mg/L, and a declining general condition. The patient mainly

experienced arthralgias, primarily in the hip joints but also in the elbows and shoulders. CRP levels somewhat decreased after initiating antibiotic treatment, raising suspicion of a bacterial infection, although no bacteria were isolated from blood cultures.

2.1 | Differential Diagnosis, Investigations, and Treatment

When the patient first presented with a worsened general condition and elevated CRP levels, chest radiography and abdominal computed tomography (CT) were performed to rule out infection, which was suspected due to the systemic nature of the symptoms. These initial imaging studies revealed no signs of infection. The focus then shifted to the patient's complaint of localized pain and swelling in the right acromioclavicular joint. A radiograph was obtained, demonstrating multiple periarticular calcifications posterior to the joint. To further evaluate this finding, an ultrasound was performed, but the absence of effusion precluded the need for aspiration.

Approximately 1 month later, the patient developed significant pain and swelling in the right elbow joint. Given the concern for a deeper infectious process, an MRI scan was performed, revealing a finding that raised suspicion of osteomyelitis of the olecranon, arthritis of the elbow joint, and olecranon bursitis. Aspiration of the olecranon bursa was performed to confirm or exclude an infectious cause; however, no bacterial growth was identified.

After several months, the patient was re-hospitalized for suspected infection due to fever and new symptoms of tenderness, swelling, and redness in both elbows. Radiographs were obtained, revealing soft tissue calcifications on both sides, located posterior to the proximal ulna (Figure 1A–C). A few weeks later, another hospitalization occurred due to persistent symptoms and a lack of response to vancomycin. At this time, the most significant clinical findings were in the right shoulder, with swelling, pain, and reduced range of motion. A radiograph demonstrated calcific expansion in the acromioclavicular (AC) joint, leading to complete destruction of the lateral clavicle (Figure 1D). MRI and CT scans confirmed extensive periarticular calcifications around the AC and glenohumeral joints without synovial inflammation. The calcified masses exerted pressure on the bone, causing erosions. Blood cultures remained negative, and a whole-body CT scan did not reveal infection

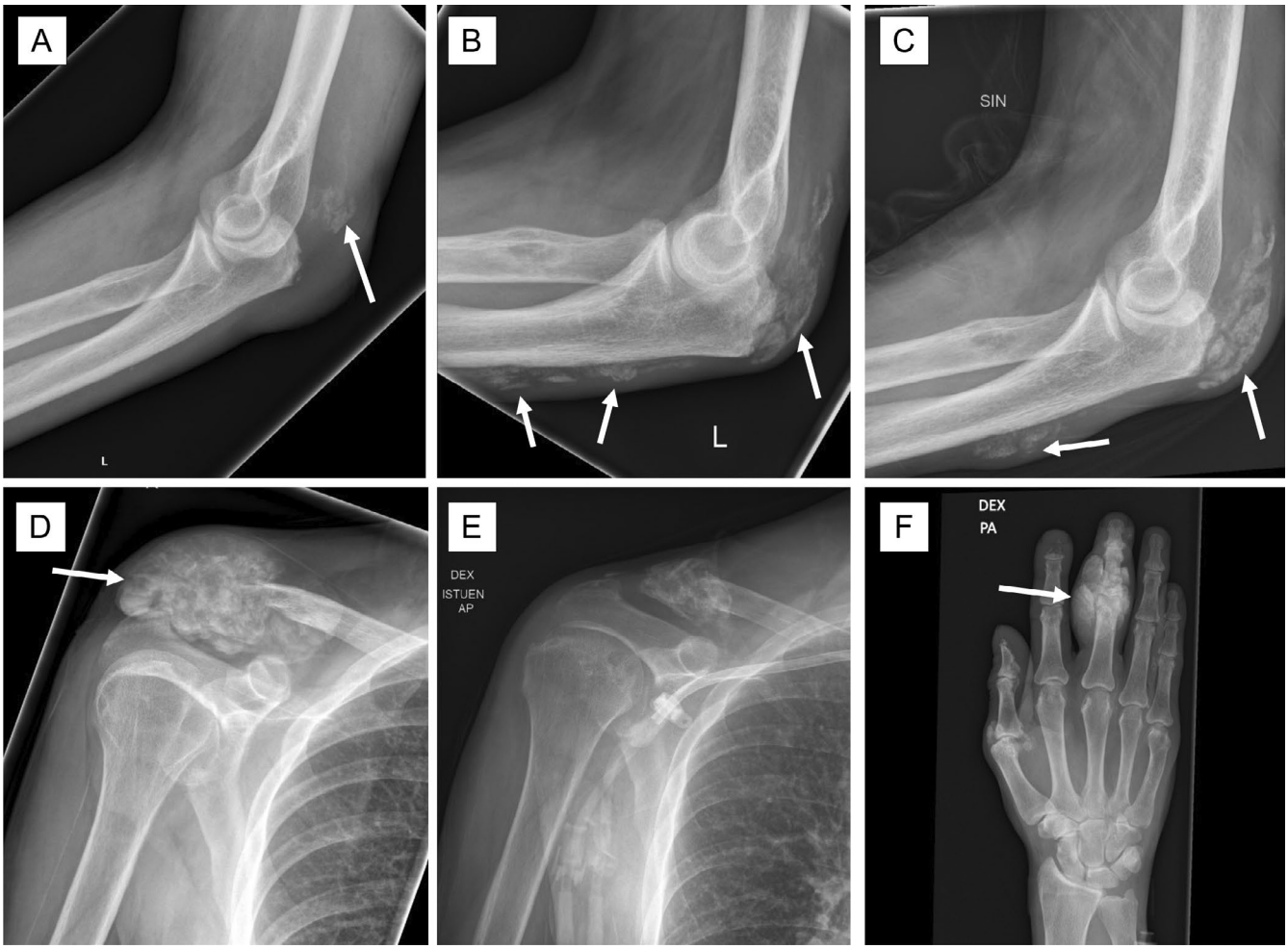

FIGURE 1 | X-ray images showing periarticular calcifications. (A–C) Progressive calcification around the left elbow joint: (A) approximately 1 month after symptom onset, (B) 2 months later, and (C) 3 months later (arrows indicate calcifications). (D) Extensive calcific expansion in the right shoulder region with destruction of the lateral clavicle approximately 4 months after symptom onset (arrow indicates calcification). (E) Significant reduction of calcifications on a follow-up radiograph 10 months later. (F) Large calcified tumorous mass in the area of the right PIP-III joint (arrow).

but did show additional periarticular calcifications in the hip joints (Figure 2C). A suspicious finding in the left hip region, initially thought to be an abscess, was ultimately identified as a hematoma.

A whole-body PET-CT scan, including the neck and upper extremities, was performed to evaluate for malignancy or occult infection. This scan showed no evidence of malignancy or infection but demonstrated increased 18F-fluorodeoxyglucose uptake at multiple periarticular sites, indicative of a widespread inflammatory process linked to severe periarticular calcifications (Figure 2A,B). Finally, a dual-energy CT scan of both upper extremities ruled out the presence of monosodium urate crystals, which were suspected especially in the tumorous mass in the area of the right PIP-III joint (Figures 1F and 2D), thereby excluding gout.

2.2 | Outcome and Follow-Up

The periarticular calcification was attributed to CKD-MBD, as in addition to the imaging findings, the patient exhibited nearly all the typical laboratory markers of the condition: elevated plasma phosphorus levels ranging from 2.03 to 4.05 nmol/L, a low serum 25-hydroxyvitamin D level (23 nmol/L), and increased plasma parathyroid hormone (PTH) levels ranging from 58 to 105 pmol/L. However, hypocalcemia was not observed.

The pattern of repeated hospitalizations and antibacterial treatments was halted following the diagnosis of CKD and hemodialysis-related inflammatory periarticular calcification. The inflammation was initially managed with high-dose glucocorticoids, which were gradually tapered within the first month of treatment. The patient maintained stability on a 5-mg/day maintenance dose of prednisolone, and no further hospitalizations, antibiotic treatments, or surgical interventions were necessary. A substantial resolution of the tumorous calcifications was observed as shown in Figure 1E.

3 | Discussion

Musculoskeletal problems are prevalent in CKD and hemodialysis patients [5]. The most common are muscle cramps, myalgias, and arthralgias. CKD-MBD is a disorder that is characterized by markedly elevated serum levels of PTH and abnormal regulation of serum calcium and phosphorus levels. It accelerates mineral deposition in blood vessels causing vascular calcification and endothelial dysfunction ultimately leading to an elevated risk of cardiovascular events [6]. CKD-MBD affects more than 90% of patients with stage 5D CKD [7]. The main changes seen in CKD-MBD include increased phosphorus levels, reduced calcium levels, low serum vitamin D, and heightened release of PTH from the parathyroid glands, leading to secondary hyperparathyroidism [8]. CKD-MBD contributes to vascular

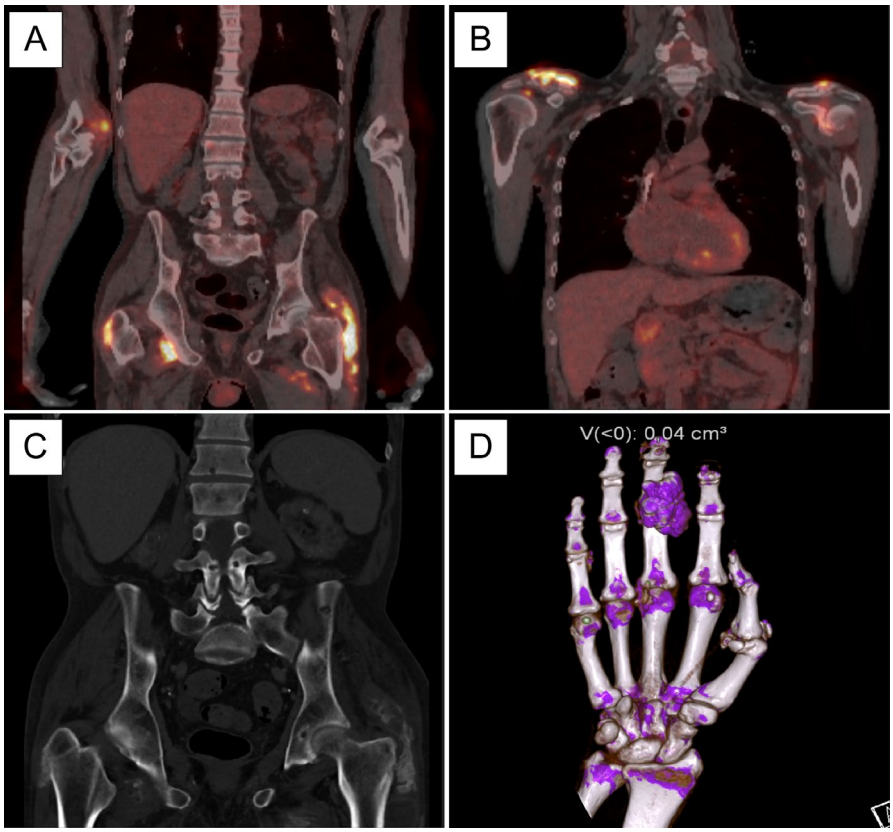

FIGURE 2 | Periarticular calcification on computed tomography images. (A) PET-CT image showing uptake of 18F-fluorodeoxyglucose consistent with inflammation in the calcified periarticular tissue around hip joints and around right elbow joint. (B) PET-CT image showing uptake of 18F-fluorodeoxyglucose in the calcified periarticular tissue in the shoulders. (C) Computed tomography image showing massive periarticular calcification in the area of left hip joint. (D) Dual energy computed tomography image showing no findings consistent with monosodium urate deposits in the right hand.

calcification, heightening cardiovascular risk [9]. Also, the calcification of soft tissue occurs, but uremic tumoral calcinosis, marked by calcium-phosphate deposits in periarticular tissues, is rare (0.5%–1.2%) [10].

4 | Conclusion

Like in our presented case, calcifications may resemble infection or autoimmune rheumatoid conditions. The condition is rare, and the way to correct diagnosis may become unwieldy and expensive. Findings of periarticular calcifications on imaging coupled with exclusion of infections, malignancies, and autoimmune disorders help get to the diagnosis. In our case, low-dose prednisolone effectively mitigated inflammation, eliminating hospitalizations, and diminishing healthcare burden.

Author Contributions

Janis Timsans: conceptualization, methodology, writing – original draft. **Mari Vilpakka:** conceptualization, writing – review and editing. **Niilo Lusila:** conceptualization, writing – review and editing. **Markku Kauppi:** conceptualization, methodology, supervision, writing – review and editing.

Ethics Statement

The authors have nothing to report.

Consent

At the time of preparation of the manuscript, the patient was deceased. Written informed consent was obtained from the next of kin to publish this report in accordance with the journal's patient consent policy.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data used to support this report are included within the article. Further inquiries can be directed to the corresponding author.

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