Open Access Library Journal
2025, Volume 12, e14418
ISSN Online: 2333-9721
ISSN Print: 2333-9705

When Crystals Break Bones: A Case Report of Femoral Fracture Revealing Silent Bone Oxalosis

Hasnae Ismaili[1], Fadwa Harmouch[1], Abdelmajid Elmrini[2], Layla Tahiri Elousrouti[1], Nawal Hammas[1], Laila Chbani[1]

[1]Pathology Department, Hassan II University Hospital, Fez, Morocco
[2]Traumatology and Orthopedic Surgery Department, Hassan II University Hospital, Fez, Morocco
Email: ismailihasnae@gmail.com

How to cite this paper: Ismaili, H., Harmouch, F., Elmrini, A., Elousrouti, L.T., Hammas, N. and Chbani, L. (2025) When Crystals Break Bones: A Case Report of Femoral Fracture Revealing Silent Bone Oxalosis. *Open Access Library Journal*, **12**: e14418.
https://doi.org/10.4236/oalib.1114418

Received: October 9, 2025
Accepted: December 9, 2025
Published: December 12, 2025

Abstract

Secondary oxalosis results from systemic oxalate accumulation most frequently associated with advanced renal failure and long-term hemodialysis. Although deposition most commonly affects the kidney, bone involvement may lead to skeletal fragility, pain, and pathological fractures. Bone oxalosis represents a rare but clinically significant complication of systemic oxalosis. We report the case of a 64-year-old woman undergoing long-term hemodialysis who presented with a pathological femoral neck fracture. Radiological assessment and histopathological analysis were consistent with bone oxalosis secondary to chronic kidney failure. This case underscores the diagnostic challenge posed by bone oxalosis, which often presents with nonspecific skeletal manifestations. Although infrequently reported, bone oxalosis should be considered in the differential diagnosis of unexplained skeletal pathology in dialysis patients. An early recognition and a multidisciplinary approach integrating clinical, radiological, and histopathological assessment is critical for accurate diagnosis and effective management.

Subject Areas

Pathology, Nephrology, Traumatology

Keywords

Oxalosis, Bone Oxalosis, Chronic Kidney Failure, Fracture

1. Introduction

Bone oxalosis is a systemic complication of oxalosis, a rare metabolic disorder

characterized by the pathological deposition of calcium oxalate crystals within the skeletal system [1]. This condition often occurs in patients with chronic renal failure, particularly those undergoing long-term hemodialysis [2]. Its progression is often insidious, and may later be revealed by the occurrence of a fracture.

In this article, we report the case of a 64-year-old female patient being on hemodialysis for more than six years presenting femoral fracture caused by bone oxalosis.

This case highlights the need for heightened clinical awareness of secondary oxalosis as a potentially serious and underrecognized complication in dialysis patients.

2. Case Report

A 64-year-old woman with a history of chronic kidney failure, being on hemodialysis for the past six years, presented to the emergency department with right lower limb pain.

Radiographic imaging revealed a femoral neck fracture. Laboratory tests showed elevated parathyroid hormone (PTH) at 106 pg/mL, serum calcium at 92 mg/L, phosphate at 24 mg/L, and alkaline phosphatase at 276 IU/L; Results reflecting disordered calcium-phosphate regulation.

The patient underwent hip prosthesis implantation, and the resected bone specimen was sent for histopathological evaluation.

On gross examination, whitish deposits were identified within the medullary intertrabecular spaces of the femoral bone (**Figure 1**). Microscopic examination revealed bone trabeculae surrounding marrow cavities containing partially calcified rosette-like structures that were birefringent under polarized light (**Figure 2**), findings consistent with calcium oxalate crystal deposition. A Von Kossa stain was additionally performed (**Figure 3**). These results led to the attribution of the femoral fracture to bone oxalosis. Therefore, the dialysis regimen was adjusted to enhance oxalate clearance.

Figure 1. Whitish deposits within the medullary intertrabecular spaces of the epiphyseal portion of the femur (black arrow).

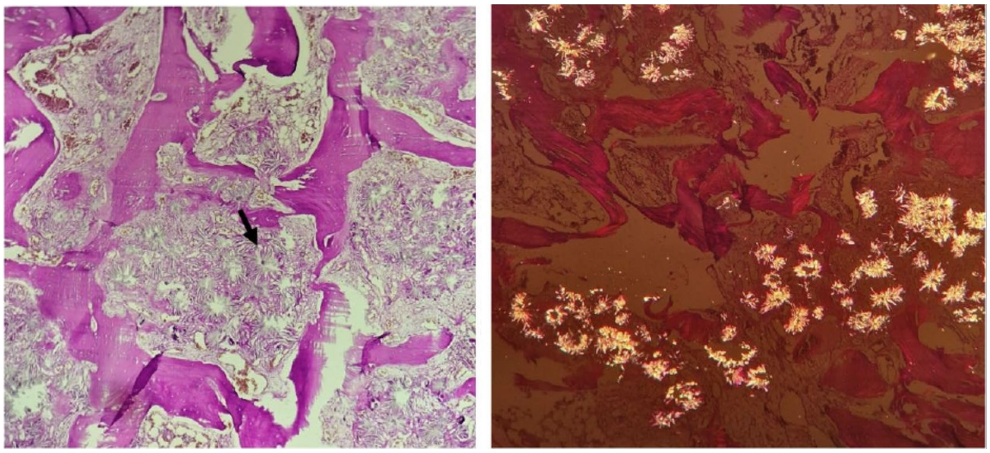

Figure 2. Histological appearance showing calcium oxalate crystals within the bone trabeculae (black arrow) (left), under polarized light (right) (HE ×100).

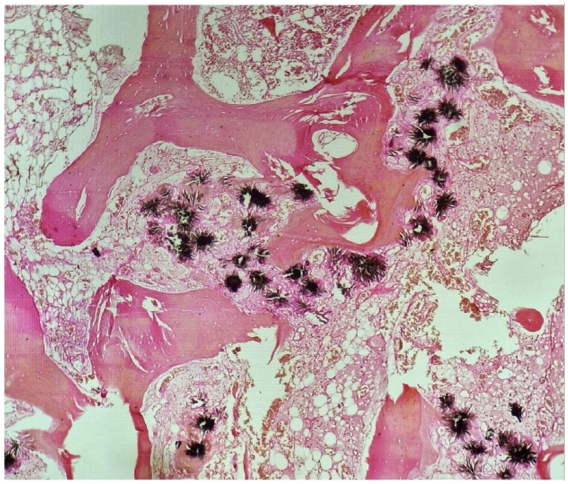

Figure 3. Positive Von Kossa staining in calcium crystals.

3. Discussion

Deposition of calcium oxalate in tissues can occur either as a result of primary or secondary hyperoxalemia [3]. Since the kidneys are the primary route of oxalate elimination, renal impairment leads to systemic accumulation of oxalate, resulting in tissue deposition, notably in bones [4]. Secondary oxalosis complicating chronic renal failure have been reported infrequently and estimating the frequency of this occurrence on the basis of published data is difficult.

Prolonged dialysis duration, insufficient oxalate clearance, and poor residual renal function appear to be the principal predisposing factors. The bone is one of the major targets of systemic oxalosis due to its high calcium content and constant remodeling activity, providing a favorable microenvironment for crystal precipitation. Over time, these deposits interfere with bone metabolism, leading to microarchitectural disorganization and structural fragility.

Bone oxalosis is rare and frequently underdiagnosed especially since its clinical presentation often presents with nonspecific symptoms such as bone pain, patho-

logical fractures, and skeletal deformities resulting from structural weakening of the bone matrix.

On the radiographic examination, bone oxalosis has been described as "diffuse bone sclerosis (with a homogeneous pattern on axial skeleton and a patchy appearance on the peripherical skeleton), bone translucency, loss of normal trabecular pattern, metaphyseal widening, pagetoid pattern, cystic change, pathologic fracture, and cortical irregularity" [5]. These imaging findings, although evocative, are not pathognomonic and can easily be confused with other metabolic or infiltrative bone diseases such as osteopetrosis, fluorosis, or metastatic osteosclerosis.

The histopathologic examination remains the gold standard for diagnosis. Characteristically, calcium oxalate crystals appear as translucent or refractile deposits that exhibit strong birefringence under polarized light. They are often arranged in radial or rosette-like patterns within the bone matrix or marrow spaces. The surrounding tissue usually shows a granulomatous inflammatory response composed of macrophages and multinucleated giant cells, sometimes associated with fibrosis and bone remodeling [6]. The Von Kossa stain serves as a valuable histochemical tool for confirming the presence of calcium-containing mineral deposits within the tissue.

This case illustrates the necessity of considering secondary oxalosis in the differential diagnosis of unexplained skeletal pathology in dialysis patients. Its recognition is crucial, not only to avoid diagnostic delays but also to adapt therapeutic approaches by optimizing dialysis protocols to improve oxalate clearance.

4. Conclusions

In summary, bone oxalosis should always be considered in patients with chronic renal insufficiency presenting with unexplained skeletal pain, fractures, or radiologic abnormalities. Although rare, its recognition carries significant clinical implications, as it reflects systemic oxalate accumulation secondary to impaired renal clearance. Early identification is essential to prevent diagnostic delays and to avoid misclassification as other metabolic or neoplastic bone diseases.

An integrated multidisciplinary approach—combining detailed clinical evaluation, targeted biochemical studies, radiological imaging, and confirmatory histopathological examination—is fundamental for establishing an accurate diagnosis. Furthermore, correlating histological findings with dialysis history and serum oxalate levels can provide valuable insights into disease chronicity and burden. Optimizing dialysis protocols to enhance oxalate clearance and closely monitoring bone health are crucial components of management.

Ultimately, increasing clinician awareness of this underrecognized entity can improve diagnostic accuracy, guide tailored therapeutic strategies, and contribute to better long-term outcomes in patients with advanced renal disease.

Conflicts of Interest

The authors declare no conflicts of interest.

References

[1] Choi, E.J., Chee, C.G., Kim, W., Song, J.S. and Chung, H.W. (2020) Bone Oxaloma—A Localized Manifestation of Bone Oxalosis. *Skeletal Radiology*, **49**, 651-655.

[2] Celasun, B., Safali, M. and Yenicesu, M. (1995) Secondary Oxalosis of Bone in a Dialysis Patient. *Scandinavian Journal of Urology and Nephrology*, **29**, 211-214. https://doi.org/10.3109/00365599509180564

[3] Shah, A., Leslie, S.W. and Ramakrishnan, S. (2025) Hyperoxaluria. StatPearls Publishing.

[4] Lorenzo-Sellares, V., Lorenzo, V., Torres-Ramírez, A., Torres, A. and Salido, E. (2014) Primary Hyperoxaluria. *Nefrologia*, **34**, 273-424.

[5] Benhamou, C.L., Bardin, T., Tourlière, D., Voisin, L., Audran, M., Edouard, C., *et al*. (1991) Bone Involvement in Primary Oxalosis. Study of 20 Cases. *Revue du Rhumatisme et des Maladies Osteo-Articulaires*, **58**, 763-769.

[6] Valdivielso, J.M. (2011) Vascular Calcification: Types and Mechanisms. *Nefrología*, **31**, 142-147.