

Available online at [www.sciencedirect.com](http://www.sciencedirect.com)

ScienceDirect

journal homepage: [www.elsevier.com/locate/radcr](http://www.elsevier.com/locate/radcr)

## Case Report

# Spontaneous bilateral femoral neck fracture unveiling end-stage chronic kidney disease in a pediatric patient: A case report <sup>☆</sup>

Mohammed Bouchoual<sup>a,\*</sup>, Kawtar Dadi<sup>a</sup>, Soufiane Bigi<sup>b</sup>, Marouane Jabrane<sup>a</sup>, Arrayhani Mohamed<sup>a</sup>

<sup>a</sup> Department of Nephrology, Agadir University Hospital Centre, Morocco, Faculty of Medicine and Pharmacy, University ibn-zohr, Agadir

<sup>b</sup> Department of Radiology, Agadir University Hospital Centre, Morocco, Faculty of Medicine and Pharmacy, University ibn-zohr, Agadir

## ARTICLE INFO

## Article history:

Received 12 March 2024

Revised 16 April 2024

Accepted 19 April 2024

## Keywords:

Mineral and bone disorders (MBD)

Spontaneous bilateral fracture in children

End-stage chronic kidney disease

## ABSTRACT

Perturbations in bone and mineral metabolism associated with chronic kidney disease (CKD) present a nuanced challenge, particularly in the context of their implications for fracture susceptibility in the pediatric demographic. Despite the well-established escalation of fracture risk in adults afflicted with end-stage renal disease, the extant scientific literature addressing this phenomenon in pediatric cohorts remains notably limited.

Within this framework, we present the case of a 16-year-old adolescent devoid of significant medical antecedents, admitted to our facility due to terminal chronic kidney disease of indeterminate etiology. The diagnosis was conclusively established following pronounced manifestations of mineral and bone disorders, exemplified by a bilateral fracture involving both femoral necks.

© 2024 The Authors. Published by Elsevier Inc. on behalf of University of Washington.

This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>)

## Introduction

Chronic kidney disease (CKD) in children represents a noteworthy clinical challenge, exerting a substantial impact on health and bone development. The mineral and bone disorders (MBD) that arise in the context of CKD are incapacitating

systemic complications that critically influence the long-term quality of life and prognosis for these young patients [1].

In spite of substantial strides in elucidating the underlying mechanisms of mineral and bone disorders (MBD) in the context of chronic kidney disease (CKD), lingering uncertainties surround the optimal management of these skeletal alterations in pediatric CKD cases. The profound clinical

<sup>☆</sup> Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

\* Corresponding author.

E-mail address: [Medbouchoual93@gmail.com](mailto:Medbouchoual93@gmail.com) (M. Bouchoual).

<https://doi.org/10.1016/j.radcr.2024.04.051>

1930-0433/© 2024 The Authors. Published by Elsevier Inc. on behalf of University of Washington. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>)

implications of MBD underscore the imperative for a nuanced and targeted approach aimed at augmenting the quality of life and prognostic outcomes for these vulnerable populations [2].

We document the case of a 16-year-old female patient without noteworthy medical history, who was admitted to our facility due to severe renal insufficiency. She exhibits a gradual onset of functional impairment affecting both lower limbs.

## Observation

This concerns a 16-year-old girl with a medical history devoid of notable incidents. Throughout the last 9 months, she has exhibited an ongoing and developing issue with her walking, unrelated to any traumatic events. Initially marked by a swaying walk, her condition worsened as she experienced a restricted walking range, coupled with partial functional impairment. Eventually, it escalated to complete functional impairment, accompanied by muscle pain in both lower limbs. Despite efforts with initial pain relievers, there was no response, and relief in specific positions was elusive.

Upon admission, the clinical examination identified a conscious patient presenting with hypertension at 160/85 mmHg. Respiratory status remained stable, diuresis was preserved, and there was evidence of delayed growth and weight. The urinary sediment exhibited active glomerular proteinuria and microscopic hematuria.

The traumatological assessment disclosed challenges in mobilizing both lower limbs due to bone pain, coupled with pronounced tenderness upon bilateral trochanteric palpation.

The Front X-rays of the pelvis and both legs above and below the joints, performed in the emergency department revealed a bi-cervical femoral fracture on demineralized bone with rarefaction of the trabeculae in the suspension zones, bi-cervical cortical and the attitude of the fractured lower limb in external rotation (Figs. 1 and 2). The pelvic computed tomography (CT) scan demonstrated a bilateral femoral neck fracture classified as GARDEN III on the right and IV on the left (Figs. 3 and 4), accompanied by bilateral hemarthrosis and discreet liquefied hematoma infiltration in localized regions of the adjacent muscles (Fig. 5).

Biologically, we noted an imbalance in the phosphocalcic profile, marked by severe hypocalcemia at 34 mg/L [85-100 mg/L], hyperphosphatemia at 76 mg/L [25-45 mg/L], secondary hyperparathyroidism at 935 pg/mL [13-65 pg/mL], and a deficiency in 1,25-hydroxy-vitamin D at 3 ng/mL [ $> 30$  ng/mL].

In light of profound hypocalcemia and secondary hyperparathyroidism, a meticulous etiological investigation revealed the presence of end-stage chronic kidney disease, marked by a serum urea concentration of 3.4 g/L [0,15-0,45 g/L], serum creatinine level of 150 mg/L [6-12 mg/L], and a glomerular filtration rate of 4 mL/min/m<sup>2</sup> as per the Schwartz formula.

The remainder of the laboratory work-up for chronic renal failure showed an arregenerative normocytic normochromic anemia at 6.5 g/dL hemoglobin [12-16g/L], with normal white blood cell and platelet counts.

Additionally, a comprehensive etiological investigation into end-stage chronic kidney disease was undertaken, en-

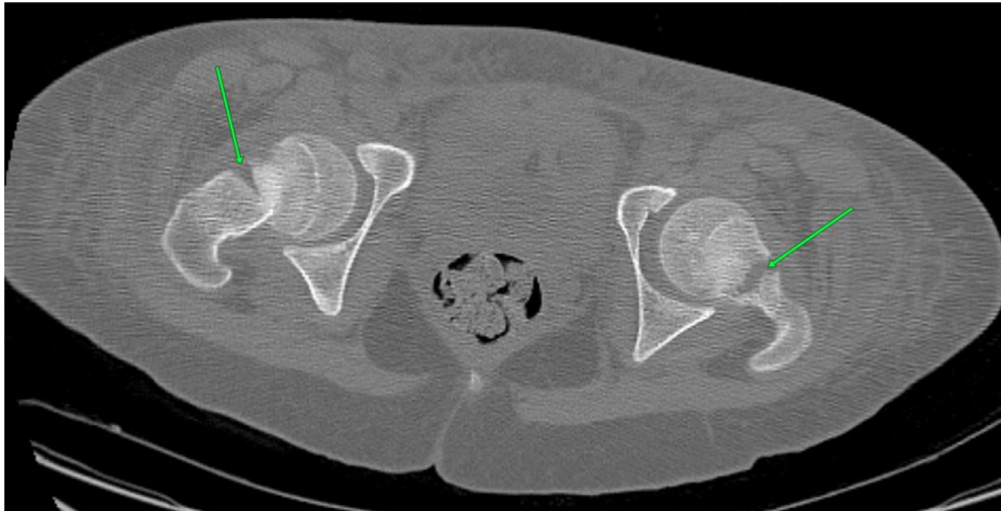


**Fig. 1 – Frontal X-rays of the pelvis showing a bi-cervical femoral fracture (white arrows) on demineralized bone with rarefaction of the trabeculae in the suspension zones with bi-cervical cortical notches (red arrows).**

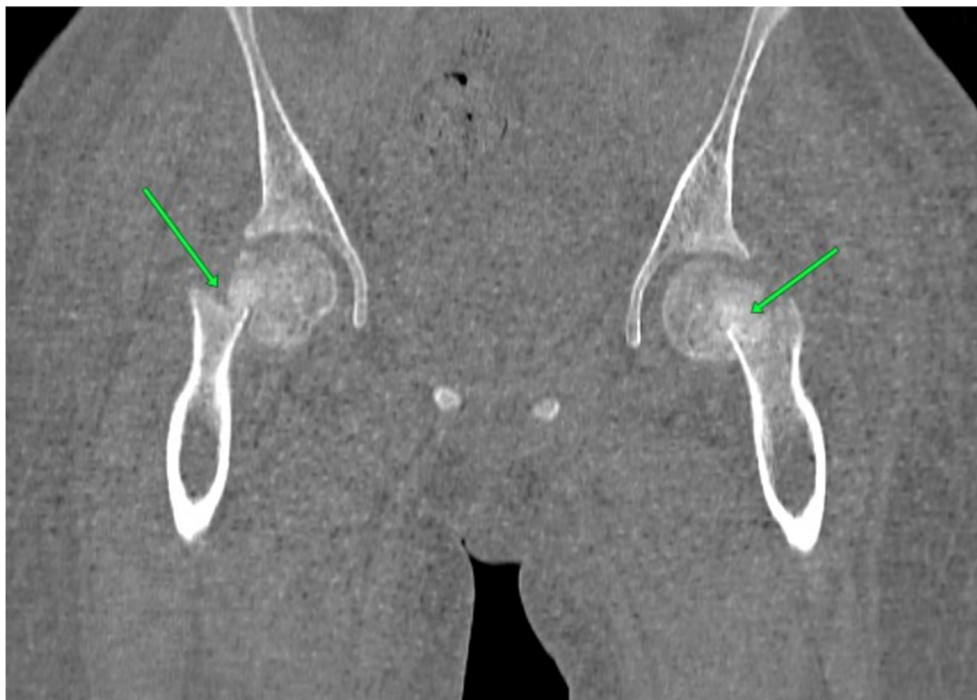


**Fig. 2 – Frontal X-rays of both legs taking above and below joints, showing attitude of the fractured lower limbs in external rotation (yellow arrows).**

compassing immunological assessments such as antinuclear antibodies and anti-DNA antibodies, complement C3 and C4 levels, serological screenings for HIV, hepatitis B and C, and serum protein electrophoresis. All findings yielded negative results.



**Fig. 3 – Axial computed tomography (CT) scan in bone window at the 2 upper ends of the femur revealing 2 cervical fractures on underlying bone insufficiency.**



**Fig. 4 – Coronal bone window computed tomography (CT) scan capturing both upper extremities of the femur highlight 2 cervical fractures occurring on a foundation of underlying bone insufficiency.**

Renal biopsy was abstained from as the kidneys exhibited diminished size and inadequate corticomedullary differentiation on renal ultrasound, indicative of chronic renal failure.

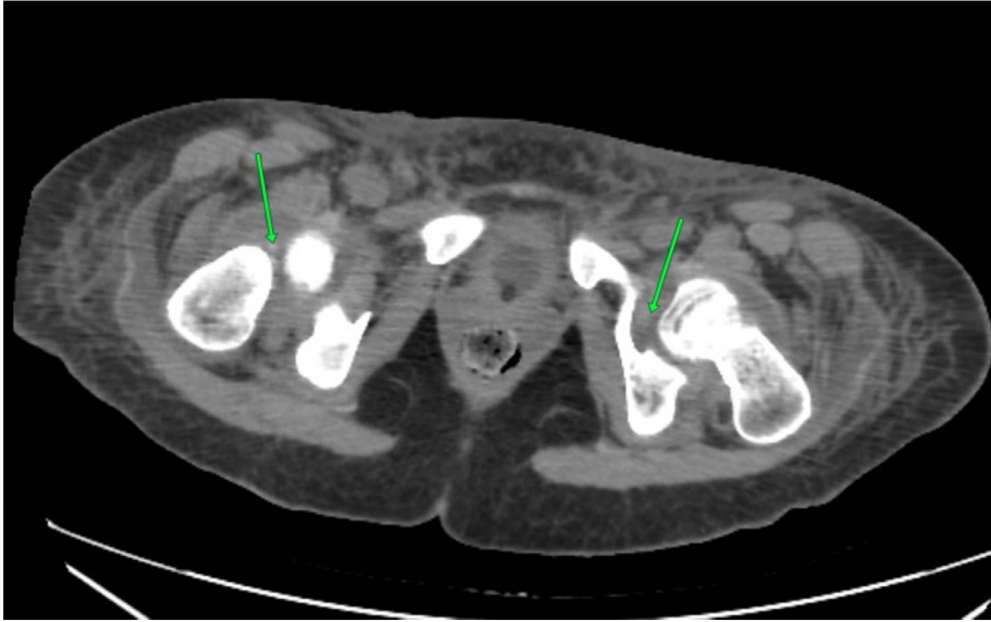
The therapeutic approach entailed the initiation of hemodialysis utilizing a tunneled right catheter at a frequency of 3 sessions per week, concomitant with pharmacological correction of phosphocalcic imbalances through the oral administration of calcium carbonate, vitamin D, and alfacalcidol.

The surgical plan included the implantation of a bilateral total hip prosthesis, contingent upon the enhancement of the phosphocalcic equilibrium.

---

## Discussion

The bone and mineral metabolism experience substantial disruptions during chronic kidney disease, potentially exerting



**Fig. 5 – Axial cross-sectional computed tomography (CT) scan traversing both hip joints, indicating minimal bilateral hemarthrosis.**

significant effects on the growth and final height of children. Moreover, these disturbances affect both their bone health and cardiovascular well-being, contributing to an elevated risk of fractures, renal osteodystrophy, and vascular calcifications.

The incidental discovery of bilateral femoral neck fractures in a 16-year-old adolescent suffering from end-stage chronic kidney disease (ESCKD) prompts essential considerations regarding the management of this uniquely vulnerable patient population. Our case report, though rare, underscores the specific challenges associated with early detection and intervention of fractures in the context of chronic kidney disease among adolescents.

Previous studies have emphasized the increased prevalence of fractures in patients with chronic kidney disease, particularly due to metabolic disruptions, bone fragility, and the impact on bone mineral density [2,3]. However, the bilateral presentation of femoral neck fractures in an adolescent adds a rare dimension to these complications.

The uncommon occurrence of this presentation underscores the necessity for heightened vigilance in adolescents with end-stage chronic kidney disease, even in the absence of overt symptoms. Previous studies have emphasized the potential underestimation of fractures in chronic kidney disease patients due to latent or absent symptoms [4,5].

Radiological assessment, encompassing both computed tomography and magnetic resonance imaging, proved indispensable for the early identification of asymptomatic fractures. This approach aligns seamlessly with current guidelines, emphasizing the critical role of early imaging in evaluating bone health among individuals with chronic kidney disease [6,7].

The analysis of biochemical parameters revealed significant perturbations in the phosphocalcic metabolism of our

patient, providing further evidence for the intricate interplay between chronic kidney disease, bone fragility, and the predisposition to fractures [8,9].

These findings highlight the pivotal significance of early metabolic management in averting musculoskeletal complications among adolescents diagnosed with end-stage chronic kidney disease.

## Conclusion

In conclusion, our observation illuminates the intricate clinical challenges linked to the serendipitous identification of bilateral femoral neck fractures in a 16-year-old adolescent afflicted with end-stage chronic kidney disease (ESCKD). The asymptomatic presentation of these fractures, their rarity within this particular demographic, and the detrimental impact on bone quality emphasize the urgent necessity for heightened vigilance and a multidisciplinary approach in addressing musculoskeletal complications among adolescents diagnosed with end-stage chronic kidney disease.

The incorporation of advanced radiological data, specifically computed tomography, has proven pivotal in the timely detection of asymptomatic fractures, enabling swift, and targeted intervention. The observed biochemical abnormalities in phosphocalcic metabolism underscore the imperative of early metabolic management to mitigate the risk of severe musculoskeletal complications associated with chronic kidney disease.

This case further emphasizes the critical significance of continuous research in this domain to enhance our comprehension of the underlying mechanisms of mineral and bone

disorders in children afflicted with end-stage chronic kidney disease. Conducting meticulous investigations is crucial to unveil the intricate interplay among chronic kidney disease, metabolic aberrations, and the heightened susceptibility to fractures in adolescents. These scientific endeavors will lay the groundwork for formulating more effective and precisely targeted management strategies tailored to this specific demographic.

---

### Patient consent

For the purpose of scientific research, I declare that I have received written consent from the patient and her guardians to use these data for the publication of this article.

### REFERENCES

---

- [1] Malluche HH, Mawad HW, Monier-Faugere M. Renal osteodystrophy in the first decade of the new millennium: analysis of 630 bone biopsies in black and white patients. *J Bone Miner Res* 2011;26(6):1368–76.
- [2] Abdalbary M, Sobh M, Nagy E, Elnagar S, Elshabrawy N, Shemies R, et al. Editorial: management of osteoporosis in patients with chronic kidney disease. *Front Med* 2023;9:1032219.
- [3] Kolárová M. Host-tumor relationship XXXIII. Inhibitor of hyaluronidase in blood serum of cancer patients. *Neoplasma* 1975;22(4):435–9.
- [4] Alem AM, Sherrard DJ, Gillen DL, Weiss NS, Beresford SA, Heckbert SR, et al. Increased risk of hip fracture among patients with end-stage renal disease. *Kidney Int* 2000;58(1):396–9.
- [5] Coco M, Rush H. Increased incidence of hip fractures in dialysis patients with low serum parathyroid hormone. *Am J Kidney Dis* 2000;36(6):1115–21.
- [6] Hsu CY, Chen LR, Chen KH. Osteoporosis in patients with chronic kidney diseases: a systemic review. *Int J Mol Sci* 2020;21(18):6846.
- [7] Jamal SA, West SL, Miller PD. Fracture risk assessment in patients with chronic kidney disease. *Osteoporos Int* 2012;23(4):1191–8.
- [8] Jean G, Souberbielle J, Chazot C. Vitamin D in chronic kidney disease and dialysis patients. *Nutrients*. 2017;9(4):328.
- [9] Moe S, Drüeke T, Cunningham J, Goodman W, Martin K, Olgaard K, et al. Definition, evaluation, and classification of renal osteodystrophy: a position statement from Kidney Disease: improving Global Outcomes (KDIGO). *Kidney Int* 2006;69(11):1945–53.