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# A Case Report On Chronic Kidney Disease-Mineral Bone Disorder In A 38 Yr Old Female Patient With Secondary Hyperparathyroidism

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**Abstract**

**Aim:** To describe the unusual case scenario of CKD –MBD in a 38-year-old female with secondary hyperparathyroidism who ended up with bilateral fracture neck of femur both sides.

Background: Chronic Kidney Disease-Mineral Bone Disorder (CKD-MBD) is a systemic disorder of the mineral and bone metabolism seen in patients with chronic kidney disease (CKD). Long-term untreated secondary hyperparathyroidism in CKD patients results in severe renal osteodystrophy with skeletal deformities.

**Case vignette:** We present the case of a 38-year-old female with chronic kidney failure on dialysis and secondary hyperparathyroidism. She now presented with history of a slip and severe pain right hip.

Past history: Patient was diagnosed 5 years back with HELLP syndrome during 2nd pregnancy. She went into kidney failure, for which she was undergoing regular dialysis for past seven years. Later she had joint pain and associated problems for which she consulted the clinician and was found to have elevated levels of PTH, increased creatinine and calcium levels of 10mg/dl. Patient was diagnosed as secondary hyperparathyroidism owing to CKD and have been advised removal of parathyroid glands.

**Diagnosis & treatment:** Xray showed fracture neck of femur and was corrected with right hemiarthroplasty. Intraoperatively, bones were found to be fragile. Later while weight bearing during recovery period, she developed fracture left femur.

**Histopathology :** Right hemiarthroplasty specimen sent for histopathology showed irregular bony trabeculae and fragments and spicules of inadequately mineralized osteoid and immature bone rimmed by osteoblasts. There is osteoblastic and osteoclastic proliferation with lacunar resorption of bone along with focal tunnel resorption. On correlating with clinical, radiological and histological features, diagnosis of Chronic Kidney Disease-Mineral Bone Disorder(CKD-MBD) was made.

**Conclusion:** Identification of secondary hyperparathyroidism is of clinical significance in patient with chronic kidney failure allowing the identification of a subgroup of patients with failure of the medical treatment of secondary hyperparathyroidism and who benefit from surgical treatment to stop the progression of the skeletal changes. The bone biopsy remains the gold standard for diagnosing the abnormalities of the bone turnover

Clinical significance: The early identification and treatment of secondary hyperparathyroidism in patient with CKD may allow for better outcomes with reduced morbidity.

**Keywords**: CKD-MBD, Renal osteodystrophy, Secondary hyperparathyroidism

## Introduction

Chronic Kidney Disease-Mineral Bone Disorder (CKD- MBD) is a systemic disorder of the mineral and bone metabolism seen in patients with chronic kidney disease (CKD). It is manifested by either one or a combination of the following:

(a) Abnormalities of calcium, phosphorus, PTH, or vitamin D metabolism.
(b) Abnormalities in bone turnover, mineralization, volume, linear growth, or strength.
(c) Vascular or other soft- tissue calcification.1

Renal osteodystrophy: Collectively describes all the skeletal changes of chronic renal disease, including those associated with dialysis.

## Pathogenesis of CKD-MBD

Classically, prior to the discovery of fibroblast growth factor 23 (FGF23), phosphate retention due to a decline in renal function had been considered as the main trigger of secondary hyperparathyroidism. The retained phosphate leads to a triad of hyperphosphatemia, low 1,25(OH)2D3 and hypocalcemia which are well- known stimuli for PTH secretion that in turn enhances phosphate excretion and development of secondary hyperparathyroidism in advanced CKD.2

Role of FGF23 in the Pathogenesis of Secondary Hyperparathyroidism

Fibroblast growth factor 23 (FGF23) is derived from osteocytes and plays a vital role in vitamin D and phosphate metabolism. It requires klotho (a transmembrane protein) to enable it to bind to the FGF receptor (FGFR) in classic target organs such as kidneys and parathyroid glands3Plasma FGF23 enhances phosphate excretion in the proximal renal tubule.

## Bone biopsy

Histologic bone changes in individuals with end stage renal failure can be divided into 3 major types of disorders:

1. High turnover osteodystrophy characterized by increased bone resorption and bone formation, with the former predominating
2. Low turnover or aplastic disease manifested by adynamic bone (little osteoclastic and osteoblastic activity) and less commonly, osteomalacia.
3. Mixed pattern of disease4

Case description: We present the case of a 38-year-old female with chronic kidney failure on dialysis and secondary hyperparathyroidism. She now presented with history of a slip and severe pain right hip.

Past history: Patient was diagnosed 5 years back with HELLP syndrome during 2nd pregnancy. She went into kidney failure, for which she is undergoing regular dialysis for past seven years. Later she had joint pain and associated problems for which she consulted the clinician and was found to have elevated levels of PTH, increased creatinine and calcium levels of 10mg/dl. Patient was diagnosed as secondary hyperparathyroidism owing to CKD and have been advised removal of parathyroid glands.

Clinical examination: Tenderness over right hip was present; ROM of right hip was restricted and patient was unable to bear weight. Right hip was in external rotation.



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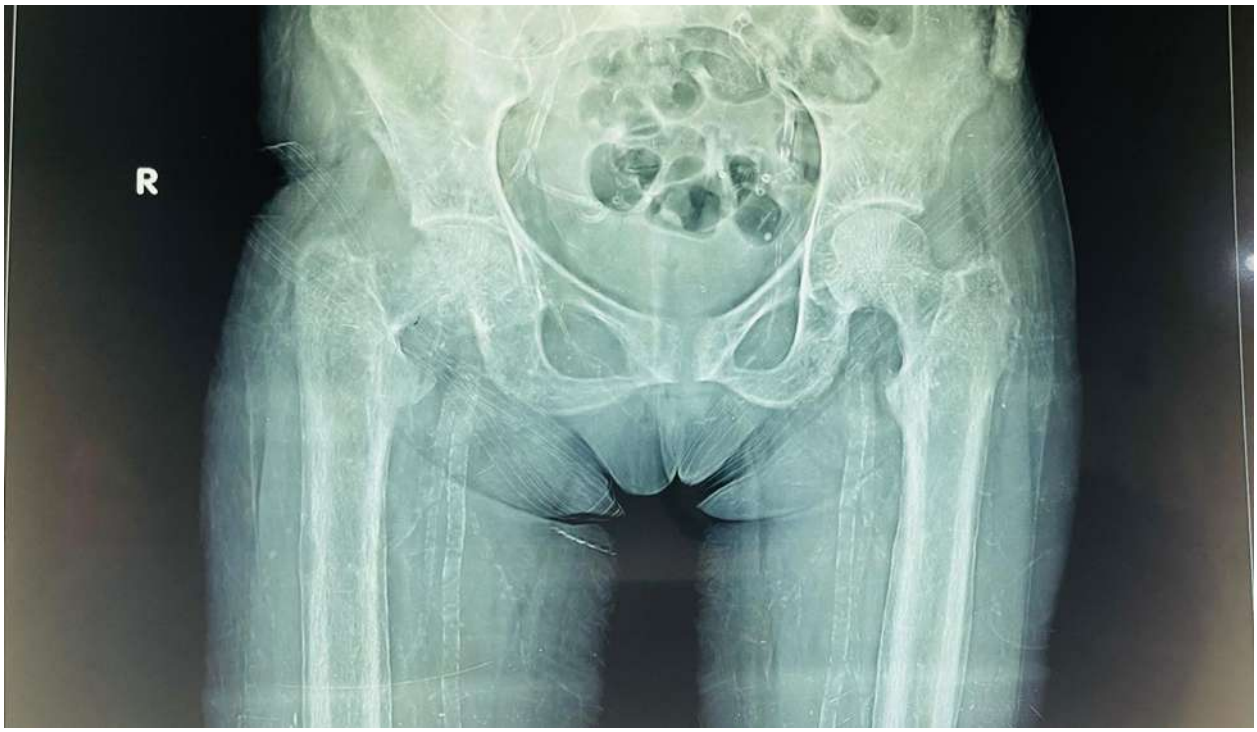
**Investigation**

**Fig 1 :- X-Ray pelvis showing fracture neck of femur(right).**




1. Alkaline Phosphatase- 604 IU/L
2. PTH-4000 microg/dl (15 to65)
3. Phosphorus-10.1mg%
4. RFT- Urea-215mg%, Creatinine-13.53mg%

**Diagnosis:** Pathological fracture neck of femur, due to secondary hyperparathyroidism

**Treatment:** Displacement was corrected with right hemiarthroplasty. Intraoperatively, bones were found to be fragile. Later while weight bearing during recovery period, she developed fracture left femur.



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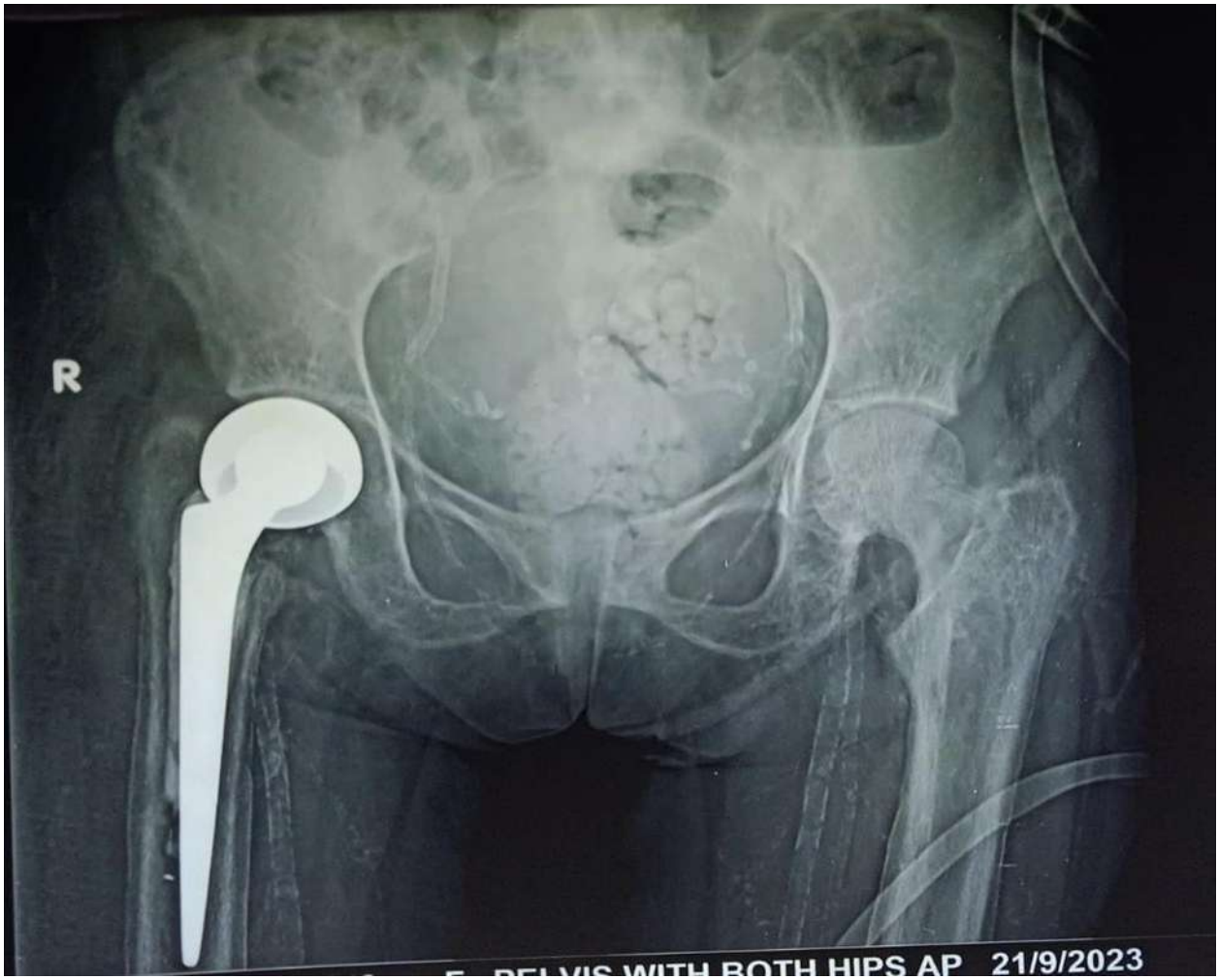
**Fig 2 : - X- Ray Pelvis after right hemiarthroplasty**




**Histopathology:** Right hemiarthroplasty specimen sent for histopathology showed irregular bony trabeculae and fragments and spicules of inadequately mineralized osteoid and immature bone rimmed by osteoblasts. There is osteoblastic and osteoclastic proliferation with lacunar resorption of bone along with focal tunnel resorption. On correlating with clinical, radiological and histological features, diagnosis of Chronic KidneyDisease-Mineral Bone Disorder(CKD-MBD) was made



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**FIG:3 – Irregular bony trabeculae and inadequately mineralized osteoid and immature bone**

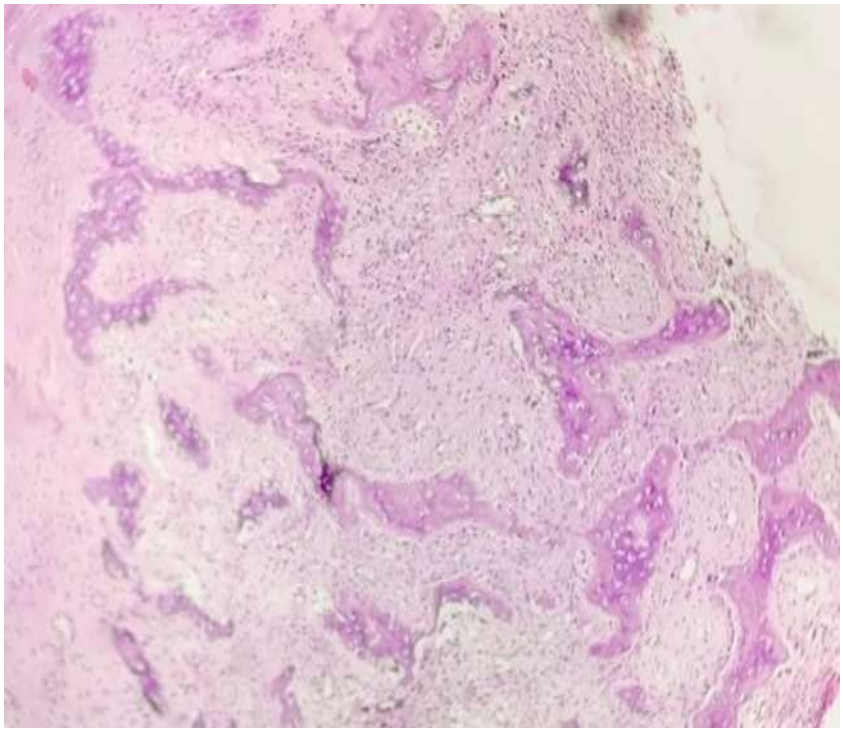

**FIG: - 4 – Lacunar resorption of bone**

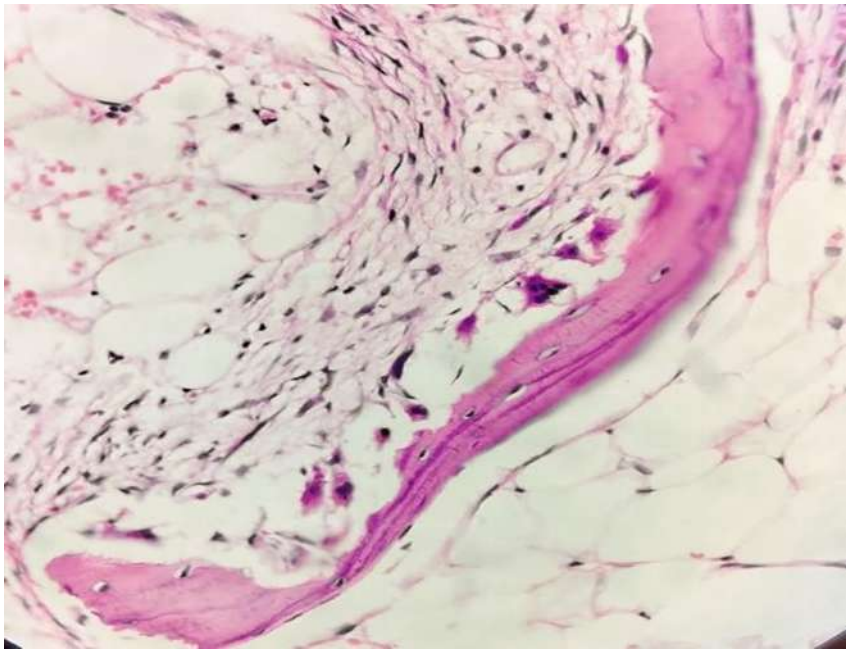



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**FIG:5- Tunnel Resorption by osteoclast**

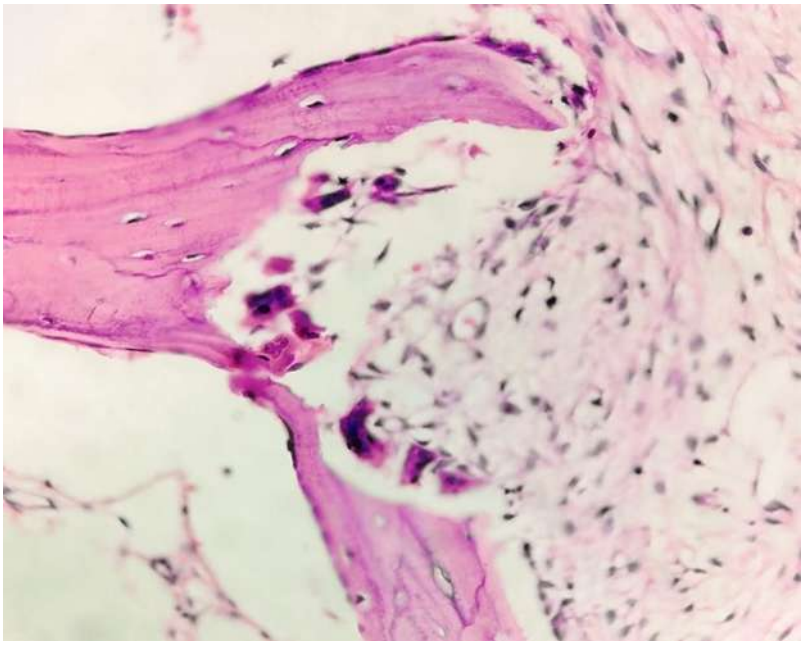
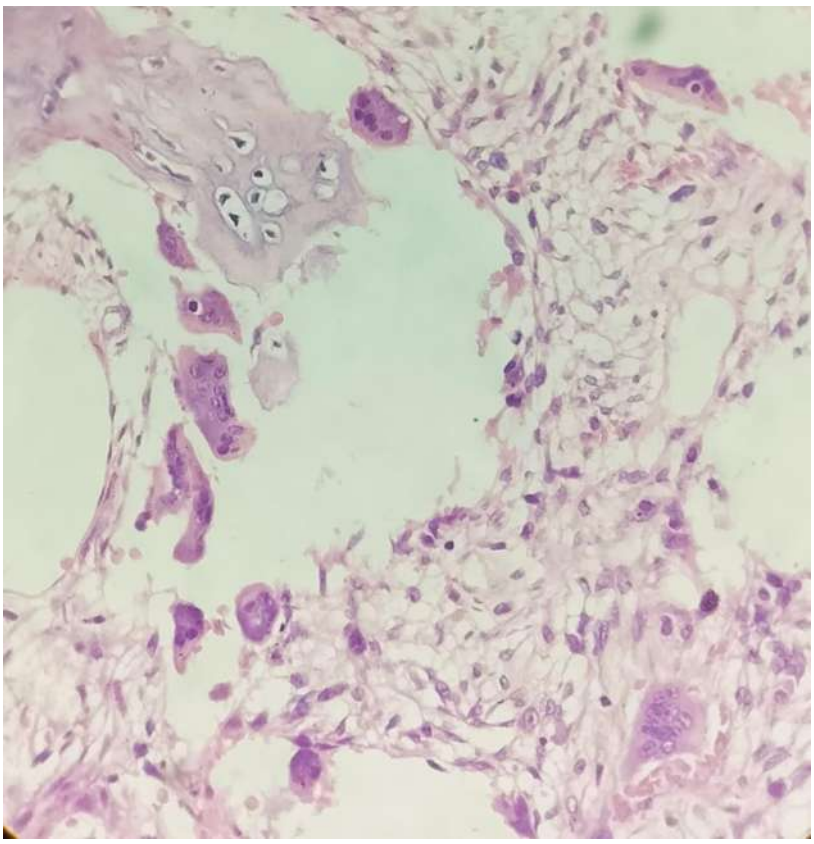


**Fig 6:- Osteoclastic proliferation**





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## Discussion

Renal osteodystrophy is one of the main complications of end- stage renal disease that leads to skeletal and extra skeletal manifestations. Although it may be unavoidable in patients undergoing hemodialysis, appropriate and timely interventions can help alleviate the symptoms experienced by the patients and also reduce the osteodystrophy-related comorbidities. 5

In our case if early detection and intervention of secondary hyperparathyroidism done, we could prevent the skeletal fractures.

## Prognosis

Complete recovery from renal osteodystrophy is possible only with a renal transplant. While evaluating the overall prognosis of this condition, other factors like the bone-vascular axis should be taken into account. Vascular calcifications, arteriosclerosis of blood vessels, and the subsequent cardiovascular events in patients of renal osteodystrophy is vital in determining the outcome for any patient with this condition.1

Conclusion: Identification of CKD -MBD is of clinical significance, allowing the identification of patients with predictable failure of the medical treatment of secondary hyperparathyroidism and who benefit from surgical treatment to stop the progression of the skeletal changes. The bone biopsy remains the gold standard for diagnosing the abnormalities of the bone turnover. Bone biopsies have been helpful in determining the clinical course and response to treatment. It is necessary to have a rational approach to the diagnosis and assessment of CKD-MBD in order to devise a treatment plan that will lead to improved outcome of the patients.

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