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Fragility fractures in renal disease: Case report

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Abstract
Osteoporosis and Chronic renal disease usually co-exist. Fragility fractures are usually under diagnosed and under evaluated. Proper evaluation can help prevent the occurrence of such fractures and thereby improve the quality of life. We are reporting a case of fragility fracture in a chronic renal disease patient. With the advent of new antiresorptive medication prevention of such fractures is possible if properly evaluated.
Case: A 69-year-old female presented to our OPD (Outpatient department) with complaints of pain over right forearm with no history of trauma on subsequent evaluation was found to have fragility fracture of multiple bones.
Management: She was evaluated and managed conservatively. Her metabolic condition was addressed and fracture healed well.
Conclusion: Management of osteoporosis in CKD also poses a challenge since most of the drugs which are used are renal dependent. Because of the complexity of bone fragility in CKD, a multidisciplinary management approach which includes nephrologists and bone experts in CKD-MBD is required before the initiation of anti-resorption or bone anabolic treatments.

Keywords: Fragility fracture, chronic renal disease, osteoprosis

Introduction
Osteoporosis is defined by the World Health Organization as "a skeletal disorder characterized by compromised bone strength predisposing to an increased risk of fracture" [1]. Chronic renal disease (CKD) is a known cause for osteoporosis. Chronic kidney disease-related bone and mineral disease (CKD-MBD) is defined by the Kidney Disease Improving Global Outcomes (KDIGO) as "a systemic disorder of mineral and bone metabolism due to CKD, which is manifested by abnormalities in bone and mineral metabolism and/or extra-skeletal calcification" [2]. Often the cause of osteoporosis is under looked in CKD and its evaluation are not complete. Here we report a case of fragility fracture of multiples bone in a lady with chronic kidney disease.

Case presentation
A 69-year-old female presented to our OPD (Outpatient department) with complaints of pain over right forearm. She didn't give any history of trauma. She was a known case of Diabetes mellitus and Chronic Kidney disease (CKD) on treatment. On clinical evaluation she had tenderness over right ulna with limitation of pronation and supination movement. Xray evaluation showed Fracture of Ulna with old resorbed fracture of radial neck. (figure 1). She was managed conservatively by a long arm slab. 1 month later she presented to Emergency department (ED) with complaints of pain left arm and pain right leg with inability to walk after a trivial trauma. On evaluation was found to have fracture both bone of right leg, and hairline fracture of humerus. (Figure 2, figure 3)

Management: She was admitted and evaluated Blood investigations showed a raised creatine value (3.82) with low serum calcium (5.4 mg/dl),normal serum phosphorous (3.3 mg/dl)low vitamin d (24.24 nmol/l) elevated Parathormone level –(917 pg/ml).A raised alkaline phosphatase level – 704.2 U/L. Usg (ultrasonogram)of the neck showed no evidence of Parathyroid lesion.
Dexa (dual energy Xray Absorptiometry) evaluation – showed osteoporosis (Figure 4).
Fractures were managed conservatively with above elbow slab for humerus fracture and above knee slab for tibia fracture. Metabolic condition was managed symptomatically and once her calcium levels improved to normal (8.5 mg/dl) she was started on Denosumab.
She became symptomatically better her fractures showed healing (figure 6)

Discussion
Although fragility fractures are common in CKD, often are under evaluated. Calcium supplements are usually given empirically. As in this case hypocalcemia caused a secondary hyper parthyroidism which resulted in further worsening of bone quality which resulted in fractures of multiple bones. Both CKD and osteoporosis often coexist as they both are strongly age associated. However, the management of fragility fractures in CKD poses many dilemmas. These include diagnosing the aetiology of fractures and choosing appropriate treatment.

Management of osteoporosis in CKD also poses a challenge since most of the drugs which are used are renal dependent. Unlike other antiresorptives, denosumab is not dependent on renal clearance, and, therefore, renal impairment is not a contraindication for its administration. There are now no clear recommendations for the management of fractures in CKD patients. As fractures occurred regardless of the circulating levels of bone biomarkers, therapeutic decisions first require the corrections of mineral disturbances. In patients with high PTH, decreasing PTH will improve the bone status. Studies [3, 6] recommend lowering PTH as a first action to reduce cortical bone loss and to limit the risk of peripheral fractures.

Among the many humoral factors affecting bone metabolism in disease states, the largest influence is brought by parathyroid hormone (PTH). PTH is secreted by parathyroid gland, which is hyperactivated under CKD conditions.

PTH promotes the activity of bone cells, namely osteoblasts, osteocytes and osteoclasts, at microscopic levels. In macroscopic levels, PTH causes cortical thinning and porosis [4] that could result in increased bone fragility. In fact, elevated serum level of alkaline phosphatase, a likely marker for accelerated bone metabolic activity, is reported to be associated with a higher risk of fracture incidence [5].

Abnormal vitamin D metabolism, hypoproteinaemia, treatments for kidney disease, and parathyroid dysfunction are assumed to be listed as possible causative factors for low bone mass in CKD patients.

Unlike bisphosphonate, the human monoclonal antibody denosumab does not accumulate even in CKD patients. However, its hypocalcaemia action tends to be amplified in CKD patients [7]. It must be in mind that hypocalcaemia in CKD patients directly induces an aggravation of hyperparathyroidism [8]. Hence close monitoring of serum calcium levels is warranted after giving denosumab.

Although teriparatide is, indeed, effective in some CKD patients, its vasodilative action often compels a discontinuation of the treatment because it can induce severe hypotension.

As per studies Denosumab is associated with reduced risk of vertebral, non-vertebral and hip fractures in osteoporotic women [9].

We used denosumab for the treatment of the osteoporosis after correction of serum calcium levels, and it helped in the healing of fractures and improving the bone quality.

Conclusion

The high incidence of fractures and mortality in patients with CKD require the use of the combination of available tools and investigations for evaluating fracture. Because of the complexity of bone fragility in CKD, a multidisciplinary management approach which includes nephrologists and bone experts in CKD-MBD is required before the initiation of anti-resorption or bone anabolic treatments.

Figures

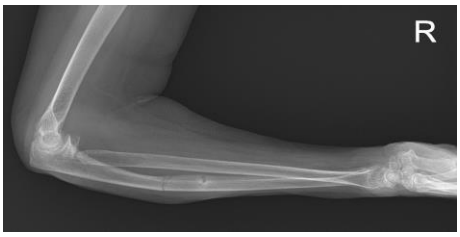

Fig 1: Xray showing Fracture of ulna shaft with old fracture radial neck

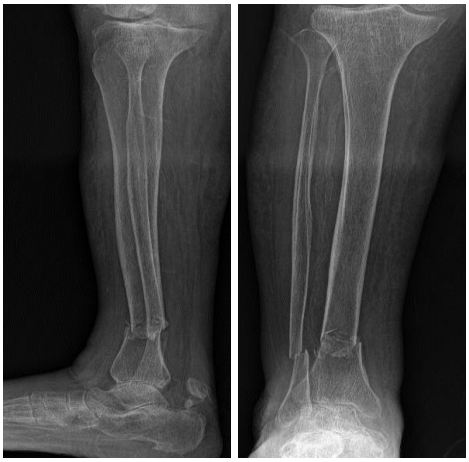

Fig 2: Xray showing fracture of both bone of leg

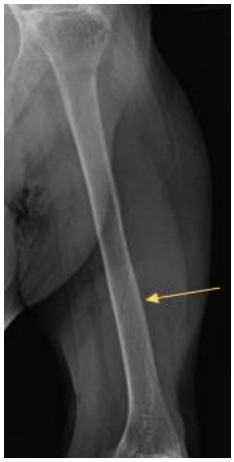

Fig 3: Showing hairline fracture Humerus

Table 1: Dexa Report

Site	Region	Measured Date	BMD	Young -Adult T-score	Age-Matched 1-score	WHO Classification
AP Spine	Ll -LA	26/1012020	0.684 Wcin^2	-4.1	-1.9	Osteoporosis
Left Femur	Neck	26/10/2020	0.428 g/cm^2	-4.4	-2.4	Osteoporosis
Left Femur	Total	26110/2020	0.466 giCTIe	-4.3	-2.5	Osteoporosis

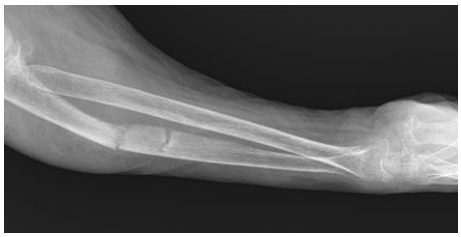

Fig 5: Healing fracture

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