Case Report

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Multiple Pathological Fractures Secondary to Renal Osteodystrophy A Rare Case Report

R B Uppin[1], Amit Motwani[2], S K Saidapur[3], Gangadhar Bhuti[4], Satish Bacchu[5,] Vishal Kulkarni[6]

Author Affiliation: [1]Professor, [2,6]Postgraduate, [3]Associate Professor, [4]Senior Resident, [5]Resident, Department of Orthopedics, KAHER Jawaharlal Nehru Medical College, Belgaum 590010 Karnataka , India.

Corresponding Author: Amit Motwani, Postgraduate, Department of Orthopedics, KAHER Jawaharlal Nehru Medical College, Belgaum 590010 Karnataka, India.

E-mail: motwaniamit6@gmail.com

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Abstract

Renal osteodystrophy is reflected as soon as the kidney function starts to worsen. The plethora of skeletal changes seen in renal osteodystrophyis classified by the status of bone turnover. It is a key cause of fractures in patients with chronic kidney disease. Upto 10% populace is affected by chronic kidney disease. The incidence of fracture is directly related to stage of kidney disease. This is consequently associated with decreased quality of life, economic burden and increased mortality. Rarely the case reports and studies have focussed on fractures associated with renal osteodystropy. So we present a rare case of spontaneous femur shaft and bilateral ulna pathological fractures secondary to chronic kidney disease leading to disability in a young male. The goal of this case report is to deal with the surgical and medical management of the condition, early ambulation and good functional outcome of the patient.

Keywords: Young male; Chronic kidney disease; Renal osteodystrophy; Pathological fractures.

Introduction

Osteodystrophy is fair comman with chronic kidney disease, which leads to altered bone morphology, defective mineralization and so the bone turnover. Research on animals have found skeletal changes in the very early stages of kidney disease which on progression can present with bone pain or even spontaneous fractures.

It increases the risk of fractures, which ultimately result in decreased quality of life, economic burden and mortality. Rarely the case reports and studies have focussed on fractures associated with renal osteodystrophy.

The strength of bone depends on composition, mineralization and thereby its density. Defect in the mineralization or the changes in bone turnover increase the risk of bone fracture.[1] In this case we will see how the young patient with chronic kidney disease with multiple pathological fracture has been addressed.

Case History

A 17 years old boy came with a chief complaint of pain in the left thigh region since 4 years. Patient was apparently alright 4 years back then he started experiencing pain in his left thigh which was insidious in onset, progressive, intermittent on and off type which aggravates on walking, squatting, climbing stairs and relieves partially on rest and pain medications. The pain eventually becomes severe that patient left his school in 10th standard,

stopped walking and standing. Now since 10 days pain has aggravated so much.

Past History: Patient is a known case of right solitary kidney. In 2012, patient came with chief complaint of severe abdominal pain. Patient was referred to urology department and there he was diagnosed with Acute renal failure secondary to renal stones in right solitary kidney. Patient treated with right nephrolithotomy with D-J stenting. His serum Creatinine was 2.5 mg/dl and blood urea 62

Developmental History: Growth retardation in the form of decreased height and weight.

General Examination: Conscious and Oriented, Thin built, Vitals stable.

Head to Toe: Coarse hair, Chest with rachitic rosary, wrist and knee widened, quadriceps atrophy.

Systemic Examination: No Significant finding.

Local Examination:

Right lower limb: Muscle atrophy of thigh noted, knee joint swelling noted, tenderness over middle one third of thigh noted, range of motion at hip and knee joint: painful and restricted, no distal neurovascular deficit.

Investigations

Date	Urea (mg/dl)	Sr.creatinine (mg/dl)	PTH (pg/dl)	Calcium (mg/dl)	Phosphorus (mg/dl)	Vitamin D (ng/dl)	ALP	ESR mm/1 hr
18/02/2020	84	3.4	239.1	7.3	9.2	39.5	355	50
25/02/2020	80	3.0	-	-	-	-	-	-
07/03/2020	71	2.4	152	9.2	4.5	-	180	-

18/02/2020 :

HB:12.6 mg /dl, PCV:40.3%, MCHC : 31.3 g/dl, RDW: 44.5%, RBC: 4.44 million/µL, WBC : 13.18 (103/µL) Sodium : 138 mEq/lt, Potassium:3.9 mEq/lt.

Radiography Report

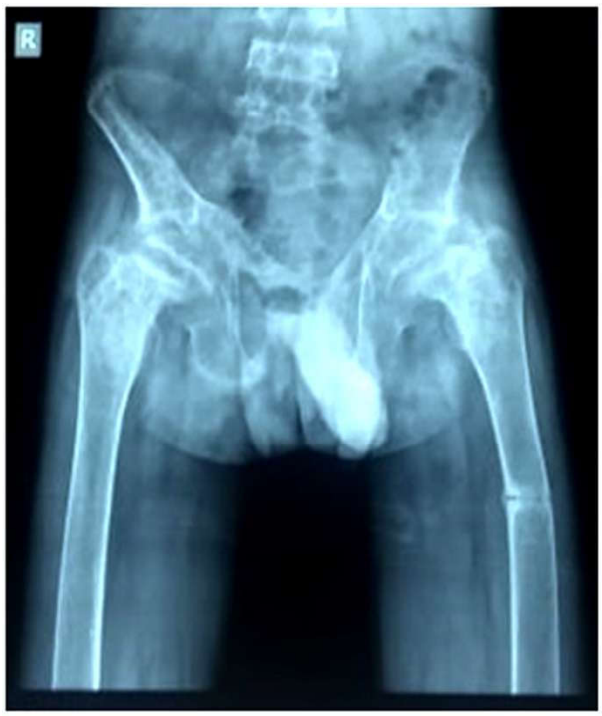


Fig. 1: X-Ray radiograph of Pelvis with bilateral hip joint.

- Transverse fracture of femur shaft, bilateral ulna suggestive of pathogical origin.

Radiography Report

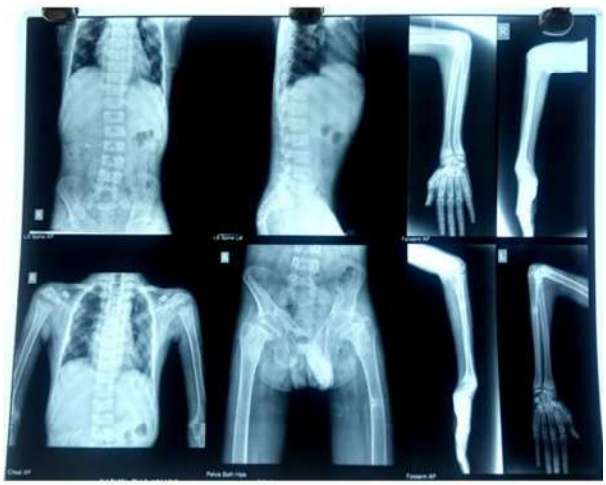
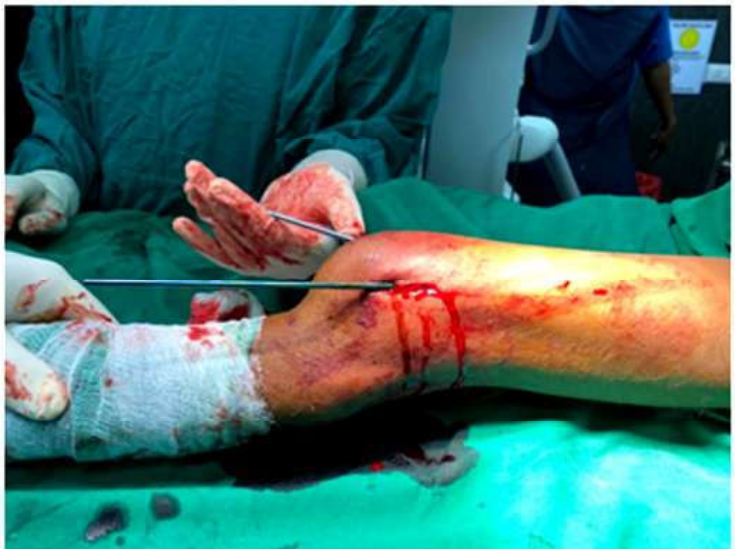


Fig. 2: X-ray radiograph of lumbosacral spine antero posterior and lateral view, forearm with elbow antero posterior and lateral view, chest antero-posterior view, Pelvis with bilateral hip joints.

- Bilateral ulna, proximal one third, un displaced, transverse, shaft fracture managed conservatively.
- Left femur, middle one third, un displaced, transverse, shaft fracture fixed surgically with Closed reduction and internal fixation.

Intra operative Images



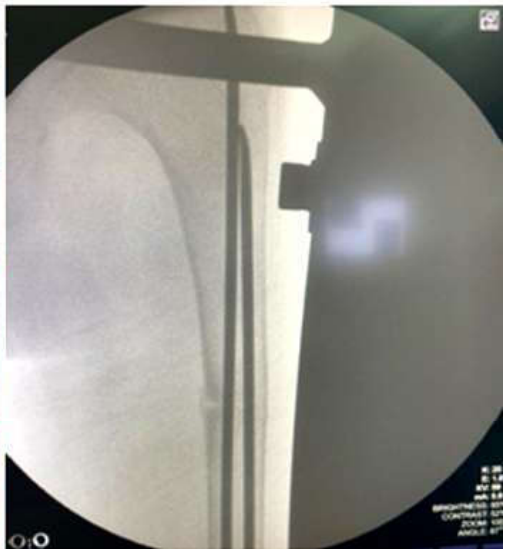


Fig. 3: Retrograde Titanium Elastic Nail System. **Fig. 4:** Two nails in intramedullary canal.

Post Operative Radiograph

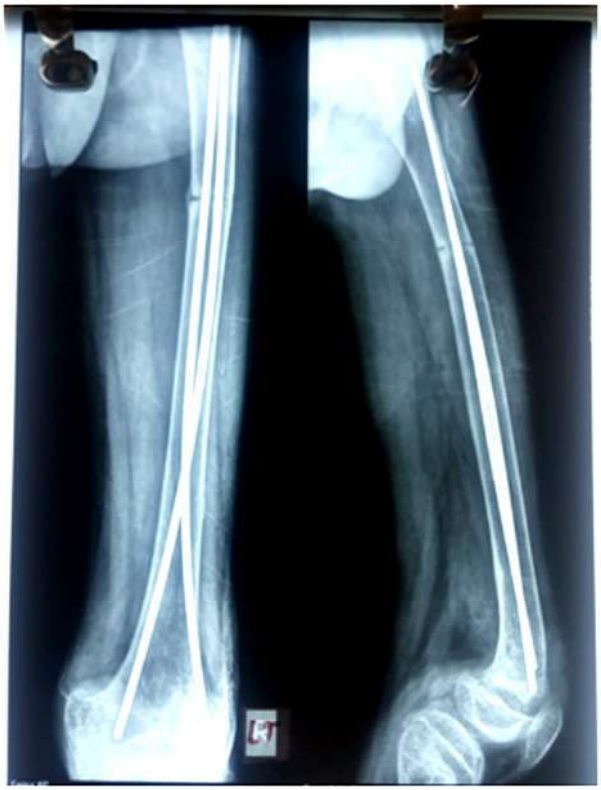


Fig. 5: Left Hip Joint Antero-posterior and lateral view with TENS in intramedullary canal.

- Syrup cholecalciferol 5ml (600 IU) OD for 1 month
- Tablet Shelcal HD 500 mg 1-0-1 for 1 month.

Follow Up after four weeks

Fig. 6: Partial weight bearing ambulation with walker.

Date	Urea (mg/dl)	Sr. Creatinine (mg/dl)	PTH (pg/dl)	Calc-ium (mg/dl)	Phosphorus (mg/dl)	ALP
22/03/2020	50	2.0	98.2	10.2	4.5	147

Discussion

Most comman site for pathological fracture in femur is neck followed by subtrochantricPathological fractures of the femur are most common in the femoral neck, followed by the sub trochanteric regions and the diaphysis. Diaphyseal pathological fractures are commonly treated with intramedullary nails or plate osteosynthesis.[2]

Surgical treatment of the above provides excellent pain relief and early rehabilitation. It improves the quality of life and soon the patient can continue his activities of daily living.[3]

The preferred treatment for femur shaft fractures more than 12 years of age is intramedullary nailing. Other options include flexible nails, external fixation, sub muscular plating.[4]

Flexible TENS (Titanium Elastic Nail System) preserves the fracture hematoma and soft tissue leading to faster fracture unionwithout causing damage to the physis.

Our method of management is reliable, quite simple with short learning curve. It has lot of advantages over other techniques of fixation. The results of flexible TENS are comparable with the recent and previous studies for managing the paediatric diaphyseal femur fractures.[5]

Renal osteodystrophy is characterized by abnormalities in bone turnover, mineralization and bone volume. These parameters should be assessed while treating the condition. The major treatment modalities for renal osteodystrophy include phosphate binders which helps in the excretion of phosphate, activated vitamin D supplements which a malfunctioning kidney cannot activate and the and calcimimetics.[6]

Secondary hyperparathyroidism is also the complication of malfunctioning kidney causing skeletal and cardiovascular complications in such patients. Activated vitamin D i.e Calcitriol therapy effectively manages PTH levels. Market has come with newer and costly preparations in an effort to manage these complications of hyperparathyroidism. Less evidence is there which can show comparative results with calcitriol therapy. Further future research is still required to clearly discover newer agents which can compete with the historically gold standard treatment i.e calcitriol therapy.[7] Though Calcitriol and calcium salts can be used to suppress PTH and improve osteomalacia but there is growing concern that these agents predispose to the development of vascular calcification, cardiovascular morbidity, low turn over bone disease and fracture. Newer preparations contains less calcium vitamin D analogues, bisphosphonates for hyperparathyroidism and sevelamer also for hyperphosphetemia. Hormone-replacement therapy (HRT) and the calcitriol maintains bone mineral density (BMD) even in end stage renal disease (ESRD) ion few patients. Renal osteodystrophy is generally improved after renal transplantation but BMD doesn't improve. Bisphosphonate therapy may be better option for some selected patients who are at risk of fracture. When kidney bone disease i.e renal osteodystrophy is assessed with the help of biochemical markers, histology and bone densitometry, early intervention and the careful use of an increasing number of effective therapies can reduce the morbidity associated with this common problem.[8]

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