Section: Pediatrics

Original Research Article

SEVERE MINERAL BONE DISEASE IN A CHILD WITH A SOLITARY KIDNEY AND NEGLECTED END-STAGE RENAL DISEASE ON MAINTENANCE HAEMODIALYSIS

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ABSTRACT

Chronic kidney disease–mineral and bone disorder (CKD-MBD) represents a systemic complication of advanced renal dysfunction characterized by abnormalities in calcium, phosphate, vitamin D metabolism, and secondary hyperparathyroidism leading to skeletal and cardiovascular complications. We report a neglected pediatric case of end-stage renal disease (ESRD) with a solitary kidney that developed severe mineral bone disease due to prolonged absence of follow-up and lack of supplementation. A male child in late childhood with a history of vesicoureteral reflux–related renal damage and prior nephrectomy presented with progressive severe bone pain, immobility, and reduced urine output. The child had been advised dialysis several years earlier, but was lost to follow-up and had not received mineral or vitamin D supplementation. Clinical evaluation suggested advanced CKD with severe skeletal involvement consistent with CKD-MBD. The patient was initiated on maintenance Hemodialysis with supportive metabolic management. This case highlights the devastating skeletal consequences of untreated CKD-MBD in children and emphasizes the importance of early detection of reflux nephropathy, long-term nephrology follow-up, and strict metabolic monitoring in pediatric CKD patients.

Keywords: CKD–mineral bone disorder, End stage renal disease, Solitary kidney, Hemodialysis Pediatric renal disease.

INTRODUCTION

Chronic kidney disease (CKD) is associated with progressive disturbances in mineral metabolism, collectively termed CKD-mineral and bone disorder (CKD-MBD). This syndrome encompasses abnormalities in calcium, phosphate, parathyroid hormone (PTH), and vitamin D metabolism, leading to bone pathology and vascular calcification.[1-3]

In pediatric patients, CKD-MBD is particularly significant because childhood and adolescence represent critical periods for skeletal growth and bone mineralization. Impaired kidney function completely disrupts phosphate excretion and vitamin D activation, resulting in hypocalcaemia and secondary hyperparathyroidism, which accelerate bone turnover and skeletal deformities.[1]

End-stage renal disease (ESRD)pediatric patients undergoing maintenance Hemodialysis frequently develop mineral bone disease due to persistent disturbances in mineral metabolism, with elevated PTH and phosphate levels contributing to renal osteodystrophy and increased morbidity.[4]

Congenital or acquired solitary kidney conditions also carry an added and significant risk for progressive renal impairment. Vesicoureteral reflux (VUR) is found asone of the most common pediatric urological abnormalities leading to renal scarring and eventual CKD if untreated.[5,6]

Delayed diagnosis, inadequate follow-up, or poor adherence to metabolic therapy in pediatric CKD can therefore culminate in severe skeletal complications and growth impairment.[1,7]

This report describes a Pediatric case of ESRD with a solitary kidney presenting with severe CKD-MBD due to prolonged absence of nephrology follow-up and supplementation.

Case

Ethics statement: This study was exempted from review by the Institutional ethics Board Written informed consent was obtained from the Parent of thepatientto participate in the study.

A male child firstborn to non consanguineouslymarried couple ,with insignificant family history,uneventful birth history, immunised upto 5 yrs of age ,hailing from lower socioeconomic status, in late childhood was brought by his mother with complaints of severe generalized bone pain and inability to walk, which had progressively worsened since early childhood. The patient also reported reduced urine output for approximately five days prior to presentation.

There was no history of breathlessness, chest pain, recurrent respiratory infections, cola-colored urine, fever, skin rash, jaundice, headache, visual disturbances, abdominal pain, vomiting, diarrhea, or altered sensorium.

The child hadsignificant past history as follows : The patient's renal disease was first detected in infancy.

At approximately 40 days of life, the infant presented with poor weight gain and abdominal distension. He remained hemodynamically stable and was initially managed conservatively.

At around four months of age, the child developed discolored urine, raising suspicion for urinary tract infection. Ultrasonography of the abdomen and pelvis revealed bilateral grade III hydroureteronephrosis, with possible causes including vesicoureteral reflux (VUR), cystitis, or posterior urethral valves.

At five months of age, the child underwent bilateral ureterostomy and fulguration. Although reflux was reduced, the right kidney demonstrated poor output.

At approximately one year of age, the left ureterostomy was reversed with ureteral re-anastomosis.

By two years of age, imaging demonstrated marked reduction in right renal function with a contracted kidney and irregular parenchymal thickness, leading to right nephrectomy.

The child was subsequently diagnosed with chronic kidney disease and advised dialysis at around five years of age, but was lost to follow-up and did not receive dialysis or metabolic supplementation until the current presentation.

On examination, the child was conscious, oriented, and afebrile. Pallor was present. There was no icterus, cyanosis, clubbing, generalized lymphadenopathy, or pedal edema.Anthropometric measurements demonstrated severe growth retardation.

The child exhibited several skeletal abnormalities suggestive of chronic metabolic bone disease, including Frontal bossing, Craniotabes, Dental hypoplasia, Rachitic rosary, Pectuscarinatum, Harrison sulcus, Swelling of the wrist and ankle joints, and Windswept deformity of the lower limbs. These findings were consistent with advanced mineral bone disorder secondary to chronic kidney disease. [Figure 1]

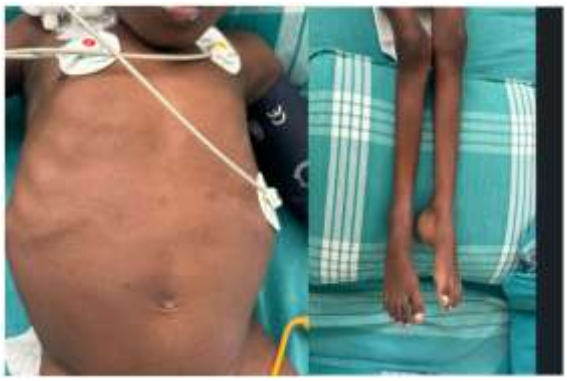

Figure 1: General Examination- Skeletal abnormalities

Table 1: Anthropometry and Vital Signs

Parameter	Findings	Interpretation
Weight	14 kg	<3rd centile indicating severe growth retardation
Height	94.5 cm	<3rd centile indicating chronic stunting
BMI	15	Within 25–50th centile
Respiratory rate	18–30/min	Within normal range
Oxygen saturation	≥95%	Normal oxygenation
Pulse rate	70–120/min	Normal for age
Blood pressure	Age appropriate	Within 50–90th centile
Temperature	97–99°F	Afebrile

Investigation

Laboratory evaluation revealed severe anemia, hyperphosphatemia, hypocalcemia, vitamin D deficiency, and markedly elevated parathyroid hormone levels consistent with advanced CKD-mineral bone disorder. [Tables 3]

Table 3: Biochemical parameters

Investigation	Result	Interpretation
Haemoglobin	5.3 g/dL	Severe anemia consistent with anemia of chronic kidney disease
Total Leukocyte Count (TLC)	15,100 cells/mm³	Leukocytosis suggesting possible infection or inflammatory response
Platelet Count (PLT)	3.6 lakh/mm³	Within normal limits
Sodium	130 mEq/L	Mild hyponatremia is commonly seen in chronic kidney disease

Chloride	95 mEq/L	Slightly decreased chloride may occur with metabolic disturbances in CKD
Serum calcium	6 mg/dL	Often low in CKD-MBD
Serum phosphate	5.6 mg/dL	Often elevated due to impaired excretion
Parathyroid hormone	520 pg/mL	Elevated in secondary hyperparathyroidism
Vitamin D	17 ng/mL	Frequently deficient in CKD

Elevated PTH, phosphate retention, and vitamin D deficiency are recognized biochemical hallmarks of CKD-MBD.3,7

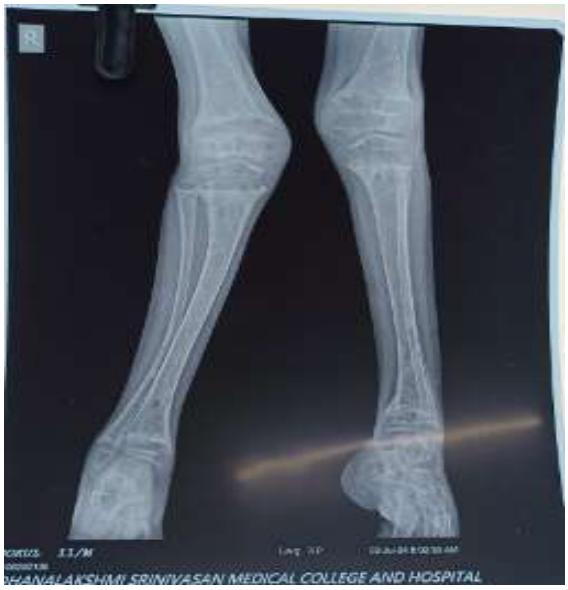

Figure 2: Skeletal radiograph

Figure 2: Plain radiograph of the lower limbs demonstrating features suggestive of renal osteodystrophy including generalized osteopenia and cortical thinning consistent with severe CKD-mineral bone disease.

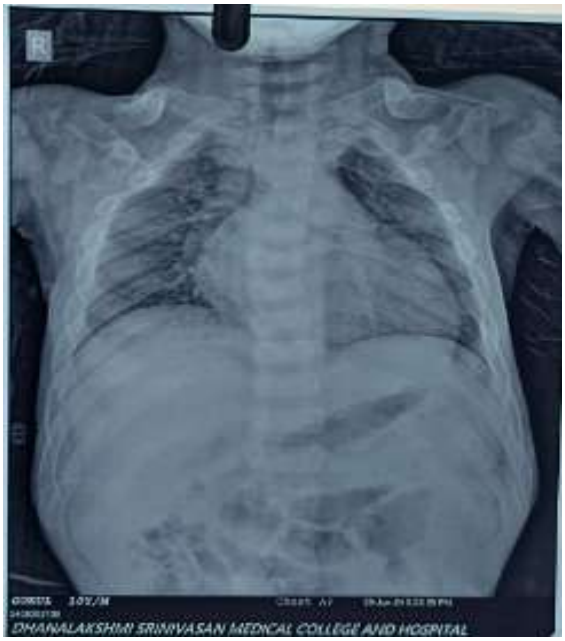

Figure 3: Chest-PA view radiograph

Figure 3: Radiograph of the chest-PA view demonstrating reduced bone density and structural skeletal changes associated with secondary hyperparathyroidism in advanced chronic kidney disease.

Table 4: Clinical timeline

Age/Stage	Clinical Event
Neonatal period	Poor weight gain and abdominal distension
Early infancy	Bilateral hydroureteronephrosis detected
Infancy	Bilateral ureterostomy and fulguration
Early childhood	Progressive renal dysfunction
Around early childhood	Right nephrectomy due to a non-functioning kidney
Later childhood	Advised dialysis, but lost to follow-up
Current presentation	Severe bone pain, immobility, and oliguria
Hospital visit	Initiation of maintenance Hemodialysis

Table 4: Clinical timeline illustrating progression from congenital urinary tract abnormality and vesicoureteral reflux to solitary kidney, chronic kidney disease, loss to follow-up, and eventual development of severe CKD-mineral bone disorder requiring maintenance haemodialysis.

The child was initiated on maintenance Hemodialysis as recommended earlier in the course of illness. Management of CKD-MBD typically includes:

- Phosphate control through dietary restriction and phosphate binders
- Vitamin D supplementation
- Correction of hypocalcemia
- Management of secondary hyperparathyroidism

These interventions aim to normalize mineral metabolism and prevent progression of skeletal complications.[1,7]

DISCUSSION

Chronic kidney disease–mineral and bone disorder (CKD-MBD) usuallyrepresents a spectrum of systemic abnormalities affecting bone metabolism, mineral homeostasis and vascular integrity in patients with chronic kidney disease. Disturbances in calcium, phosphate, vitamin D and parathyroid hormone regulation form theverybiochemical basis of this disorder and slowly progressively lead to structural bone abnormalities and increased fracture risk. In pediatric patients, these metabolic alterations will have particularly severe consequences because

bone development and linear growth occur rapidly during childhood and adolescence.

The pathophysiology of CKD-MBD begins early in the course of renal dysfunction. As glomerular filtration rate declinesgradually, the kidney loses its ability to excrete phosphate effectively, resulting in phosphate retention. Elevated serum phosphate stimulates fibroblast growth factor-23 (FGF-23) production, which later suppresses renal activation of vitamin D and reduces intestinal calcium absorption. These processes contribute to hypocalcemia, which in turn stimulates parathyroid hormone secretion and leads to secondary hyperparathyroidism. Persistent elevation of parathyroid hormone accelerates bone turnover and results in the various skeletal manifestations collectively known as renal osteodystrophy.

In advanced stages of chronic kidney disease, the imbalance of mineral metabolism becomes more pronounced,where patients frequently develop hyperphosphatemia, hypocalcemia and markedly elevated parathyroid hormone levels. These biochemical abnormalities result in progressive bone remodeling defects characterized by cortical thinning, decreased bone density and increased susceptibility to fractures. In children, the consequences include growth retardation, bone pain, skeletal deformities and impaired mobility.

The present case illustrates the severe consequences of untreated CKD-MBD in a pediatric patient with long-standing renal disease and inadequate follow-up. The child had a history of congenital urinary tract abnormality with bilateral hydroureteronephrosis suspected due to or secondary to vesicoureteral reflux. Vesicoureteral reflux is one of the most common urological abnormalities in childhood and is recognized as a major cause of renal scarring and chronic kidney disease when it is notappropriately managed. Studies have reported that a substantial proportion of children with reflux nephropathy develop progressive renal damage that may ultimately lead to end-stage renal disease.

The patient underwent early surgical interventions including ureterostomy and later nephrectomy due to progressive deterioration of one kidney. Although surgical correction may reduce reflux and prevent further urinary tract damage, the remaining renal parenchyma may still undergo progressive decline in function. In individuals with a solitary kidney, compensatory hyperfiltration initially maintains adequate renal function, but over time this adaptive mechanism also may contribute to progressive nephron loss and chronic kidney disease.

One of the most striking aspects of this case is the prolonged loss to follow-up. The patient had been advised dialysis at an earlier stage but did not continue regular nephrology care. During this period, the child did not receive phosphate binders, vitamin D supplementation or metabolic monitoring. The absence of these interventions likely allowed progressive mineral imbalance and secondary hyperparathyroidism to develop unchecked, ultimately resulting in severe skeletal manifestations.

CKD-MBD is highly prevalent among patients undergoing maintenance haemodialysis. Several studies have reported that abnormalities in mineral metabolism occur in a majority of dialysis patients and are associated with increased morbidity and mortality. Elevated parathyroid hormone levels have been linked to increased risk of fractures, cardiovascular complications and reduced survival in patients with end-stage renal disease.

In pediatric patients, the burden of CKD extends beyond physical complications. Children undergoing long-term dialysis often experience impaired growth, decreased physical activity and significant psychosocial challenges. Reduced mobility and chronic pain may further compromise quality of life and educational participation. Early recognition and aggressive management of metabolic complications are thereforeveryessential in improving long-term outcomes.[8,9]

CONCLUSION

This case emphasizes the devastating consequences of neglected chronic kidney disease in children. Delayed follow-up and lack of metabolic therapy can result in severe skeletal deformities, impaired mobility and reduced quality of life. Improved awareness among caregivers and healthcare providers is essential to ensure early referral and continued monitoring of pediatric CKD patients.

Article information

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