

Chronic Renal Failure Presenting Primarily With Renal Osteodystrophy: A Case Report

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Citation

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Abstract

Common clinical presentations of chronic renal failure include anaemia, growth retardation, urinary complaints, hypertension and mental irritability.

Renal osteodystrophy is a common long-term complication of end stage renal disease.

The authors report an instance where a child presented with ankle pain and was subsequently diagnosed to have severe renal disease as the cause.

A previously fit and well 13year old male with no family history presented with a four month history of ankle pain with no associated trauma. Radiographs illustrated subtle widening of the growth plate. Vigilant investigation led to diagnosis of renal osteodystrophy secondary to hypoplastic kidneys.

Hyperphosphataemia and secondary hyperparathyroidism could be prevented by prompt treatment with initial renal dialysis and subsequent renal transplantation. The importance of a thorough investigation of children who present with non-traumatic skeletal pain, is clearly demonstrated.

BACKGROUND

Chronic renal failure is defined as an irreversible and progressive reduction in the glomerular filtration rate (GFR) to below 25% of normal level for at least three months.¹ The incidence of chronic renal failure ranges from one to three children per million in a population less than 16 years of age.² In children age 5 years or less the commonest causes of chronic renal failure include congenital renal diseases, such as renal dysplasia or renal hypoplasia. In older children, hereditary diseases, metabolic diseases and acquired aetiologies occur more frequently.³

Referral to an orthopaedic surgeon is usually made once a firm diagnosis of renal osteodystrophy has been established. We describe a case not previously described of an individual who presents with ankle pain and severe renal disease was subsequently diagnosed.

CASE REPORT

A 13-year-old Caucasian male child was referred to the orthopaedic clinic with a four month history of bilateral ankle pain and intermittent pain in the right knee after

exercise. He also complained of lethargy since four weeks.

He was born by vaginal delivery at full term. He had been fully immunized. He was on the 3rd centile for height and the 25th centile for weight.

On examination he appeared pale. His cardiovascular, respiratory and abdominal examinations were unremarkable. He had a full range of painless movements in all joints.

Haematological investigations performed included a full blood count, urea and electrolytes, C- reactive protein, erythrocyte sedimentation rate and rheumatoid factor.

Radiological investigations consisted of plain anteroposterior and lateral radiographs of ankles, knees and pelvis.

Results of the blood tests showed normochromic anaemia, raised ESR of 64mm/hr, normal CRP, and rheumatoid factor negative. The blood film showed mild anisocytosis and moderate rouleaux formation. Serum creatinine levels were raised at 576umol/l. (Table 1)

Figure 1

Table 1: Haematological and biochemical test results

INVESTIGATION	RESULT	NORMAL VALUE
Haemoglobin	9.3 gm/dl	13-18 gm/dl
White Cell Count	6.1x10 ⁹ /l	4-11 x 10 ⁹ /l
Red Cell Count	3.25 x 10 ¹² /l	4.5-6.5 x 10 ¹² /l
Haematocrit	0.27	0.4-0.54
Mean Cell Volume	81.9 Fl	80-100 Fl
Mean Cell Haemoglobin	28.4 pg	26.5-31.5 pg
Erythrocyte Sedimentation Rate	64 mm/hr	<13 mm/hr
Plasma Sodium	140 mmol/l	135-145 mmol/l
Plasma Potassium	3.9 mmol/l	3.5-5.5 mmol/l
Serum urea	18.2 mmol/l	3.3-7.8 mmol/l
C – Reactive Protein	5	<9
Serum Creatinine	576 umol/l	30-90 umol/l
Alkaline Phosphatase	546 u/l	50-160 u/l
Serum Calcium	2.46 mmol/l	2.2-2.7 mmol/l

Radiographs of both ankles showed abnormally wide growth plates at the distal end of the tibia. There were less marked changes in the knees and proximal femoral epiphyses. (Figure 1,2 & 3)

Figure 2

Figure 1: Anteroposterior and lateral radiographs of left ankle showing excessive widening of the growth plate.

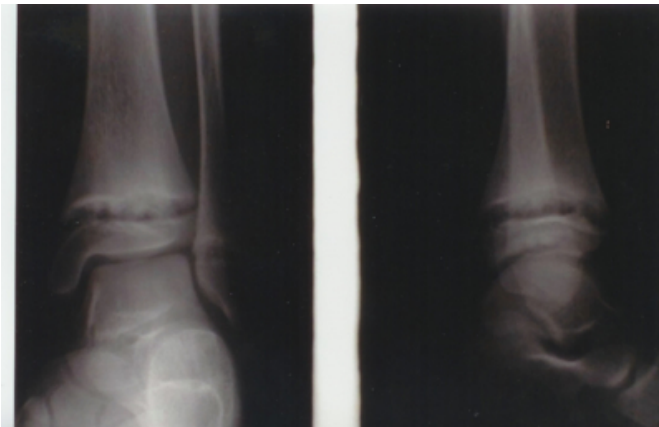


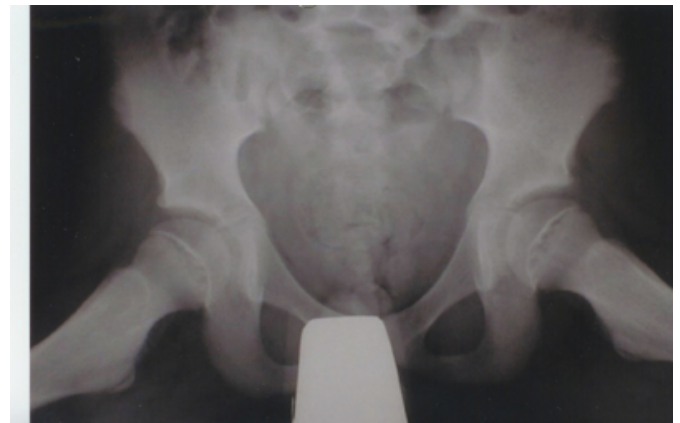
Figure 3

Figure 2: Anteroposterior and lateral radiographs of right knee.



Figure 4

Figure 3: Frog leg view radiograph of both hips.



Paediatric input was sought at an early stage for the further investigation of the normochromic anaemia and renal failure. An ultrasound scan of the abdomen confirmed the presence of bilateral hypoplastic kidneys.

The child was subsequently started on the appropriate medical treatment, and listed on the transplant register.

DISCUSSION

Davies Colley reported the first description of renal rickets in 1883. In renal osteodystrophy, glomerular damage leads to phosphate retention, and tubular injury causes decreased production of the active form of vitamin D (1,25 dihydroxycholecalciferol). The resultant hyperphosphataemia / hypocalcaemia triggers secondary hyperparathyroidism. Parathyroid hormone causes increased bone resorption. ^{5,7}

In a case of established renal osteodystrophy, the following features may be seen:

1. Deficient mineralization of the osteoid secondary to hypocalcaemia
2. Severe lytic changes in the skeleton (osteitis fibrosa cystica and Brown tumours) due to secondary hyperparathyroidism.
3. Osteosclerosis is seen in 20% of patients because the bony trabeculae look prominent in relation to decreased mineralization.
4. Slipped epiphyses. Resorption of metaphyseal regions and epiphysiolysis occurs as a result of hyperparathyroidism giving rise to slipped epiphyses. The proximal femur is the commonest site reported.^{6,9} It has also been reported to occur in proximal humerus, distal femur, distal tibia and in distal radius and ulna.⁴

CONCLUSION

This patient presented with simple ankle pain without history of trauma. Persistent investigation led to prompt diagnosis of renal osteodystrophy before serum phosphate had increased enough to stimulate hypersecretion of parathyroid hormone.

All the cases with skeletal lesions reported previously in the

literature are cases of well established renal rickets.

This is the first case reported where a previous well patient presented initially for an orthopaedic consultation, which then led to the diagnosis of renal osteodystrophy.

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