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Renal osteodystrophy presenting as ankylosing spondylitis: a case report

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Abstract

Renal osteodystrophy is a common long term complication of end stage renal disease. We here by present a case of unusual presentation of renal osteodystrophy in young female who reported as chronic backache with stiffness.Case report- A 21 year's old, unmarried female presented with progressive low back pain and gradual restriction of movements of spine with no history of trauma. On basis of clinical presentation she was admitted with an impression of Ankylosing spondylitis. Her investigations revealed raised blood urea, serum creatinine and alkaline phosphatase with low calcium. In X-rays she was found to have pseudo fractures involving inferior ischiopubic ramii, dense vertebrae, mild coarsening of trabeculae and subperiosteal resorption in proximal phalanx of index and middle fingers. Ultrasound of KUB region revealed bilateral hypo plastic kidneys. All this led us to the diagnosis of Renal Osteodystrophy.In cases of ROD the children are always stunted in growth, often to a degree not equaled by any other form, our patient's growth was also stunted. It is stated that osteomalacia is a common component. This feature was also present in our case. By the time dialysis is required almost all patients are affected with ROD. This stood true in our case also as patient was referred for dialysis after establishing the diagnosis. In a normal scenario, patients of chronic renal disease are referred cases in orthopedics and usually on dialysis and then ROD is labeled.

Key Words: Renal Osteodystrophy, Renal failure, Osteitis fibrosa, Osteomalacia

INTRODUCTION

Renal Osteodystrophy (ROD) is the term given to the bone changes which accompany chronic renal failure.[1] 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.[2] In children age 5 yrs 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 etiologies occur more frequently.[3]

We here by present a case of unusual presentation of renal osteodystrophy in young female who reported as chronic backache with stiffness.

CASE HISTORY

A 21 years old, unmarried female of low social status presented to our orthopedic outpatient department with progressive low back pain and gradual restriction of movements of back and both hips for one year. She also complained of lethargy since three weeks. For the same she had been taking treatment from quacks and doing fomentations with no avail. Her family history was not significant and menstrual history normal. There was no history of trauma, fever, night sweats, night pains, loss of appetite, loss of weight, morning stiffness, involvement of smaller joints of hand and feet, bowel disturbances, recurrent UTI's, jaundice, breathlessness, cough with haemoptysis.

On examination, she was short statured with height of four feet four inches and also physically underdeveloped. All movements of her back were restricted, especially flexion (Figure 1). Abduction and rotatary movements of both hips were also restricted. She was unable to sit cross legged and squat. Her cardiovascular, respiratory and abdominal examinations were normal. Subsequently she was admitted with a provisional diagnosis of Ankylosing Spondylitis.

Patient was investigated for–hemoglobin , total and differential leukocyte counts, ESR, random blood sugar, S.G.P.T., RA- Factor, C-reactive protein, serum uric acid, blood urea, serum creatinine, serum calcium, serum phosphorus, serum alkaline phosphatase, urine calcium and phosphorus estimation. She was found to have deranged renal function, as blood urea and serum creatinine were raised to 148 mg % and 5.0mg% .Her serum alkaline phosphatase markedly raised to 511 I.U./LIT. Her serum calcium was low 7.2 mg% and phosphate normal 6.0 mg %.(Table 1). Her X-ray pelvis revealed pseudo fractures involving both inferior ishiopubic rami (Figure 2). X-ray dorsolumbar spine revealed generalized increase in density (osteosclerosis) (Figure 3). Hand X-ray revealed early changes of renal osteodystrophy-coarsening of trabeculae and subperiosteal resorption in proximal phalanx of index and middle fingers (Figure 4).

Clinicoradiological diagnosis of Renal Osteodystrophy was made. Patient was then made to undergo ultrasound of KUB region which further revealed bilateral hypo plastic kidneys which further added to our diagnosis.

DISCUSSION

According to the clinical features described in Mercer's Orthopedics Surgery in cases of ROD the children are always stunted in growth, often to a degree not equaled by any other form of infantilism. The body weight is correspondingly small, though malnutrition is not present. Patients surviving beyond puberty may show infantilism as well as dwarfism [1]. Our patient's growth was also stunted as height at 21 years was just four feet and four inches.

Eknoyan G. et al in their study on bone metabolism and disease in chronic kidney disease have stated that the onset of

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TABLE 1- Results of hematological and biochemical tests.

Investigation	Result	Normal value
Hemoglobin	10.6	13-18 gm%
Total Leukocyte Count	5,100	4-11000 / cu.m.m.
Differential Leukocyte Count	P-71, L-26, E-3, M-0	P-40-75%, L-20-40%, E-1-6%, M-2-8%.
ESR (WINTROBE)after 1 hour	21mm	0.9
Random Blood Sugar	117.0	70-120 mg%
S.G.P.T.	19.16	5-35 IU/L
RA- Factor, serum by Nephelometry	11.00	Below 15.9 IU/mL
C-Reactive Protein	2.10	Adults : 0-3 mg/L
Serum Uric acid	5.6	3 to 6.5 mg%
Blood Urea	148.0	24-45 mg%
Serum Creatinine	5.0	0.7-1.4 mg%
Serum Calcium	7.2	8.5-10.5 mg%
Serum Phosphorus	6.0	2.5-4.8 mg%
Serum Alkaline Phosphatase	511.0	25-100 I.U./LIT.
Urine Calcium(Cresolphthalein complexone)	4.5	mg/dL
Urine Phosphorus (phosphomomolybdate method)	14.2	mg/dL

TABLE 2- Histological Classification of Renal Osteodystrophy.

Disorder	Description	Frequency %
Osteitis Fibrosa	Peritrabecular fibrosis, increased remodeling-resorption and formation.	50
Osteomalacia	Increased osteiod, defective mineralization.	7
Mixed disease	Features of both osteitis fibrosa and osteomalacia.	13
Mild disease	Slightly increased remodeling.	3
Adynamic renal bone disease	Hypocellular bone surfaces, no remodeling.	27

Figure 1-showing restricted flexion of spine.

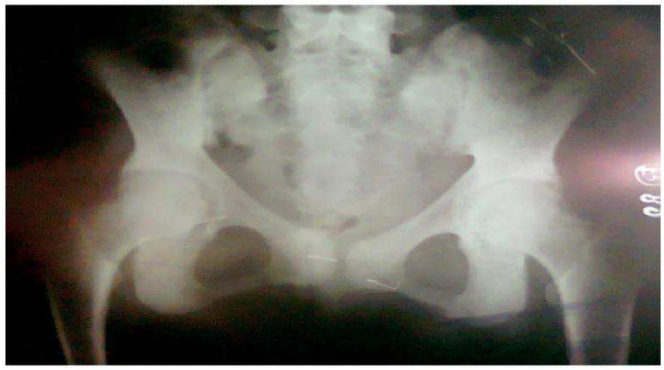

Figure 2-X-ray of pelvis showing pseudo fractures involving both inferior ishiopubic rami.

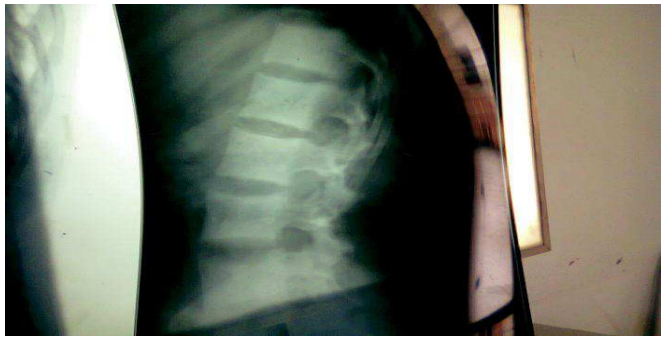

Figure 3- X-ray of dorsolumbar spine showing generalized increase in density.

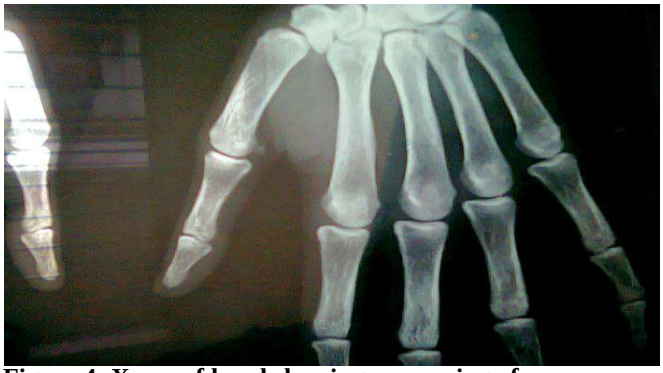

Figure 4- X-ray of hand showing coarsening of trabeculae and subperiosteal resorption.

ROD is generally noted when about 50% of kidney function is lost, and by the time dialysis is required almost all patients are affected [4]. This stood true in our case also as patient was referred for dialysis after establishing the diagnosis.

Moriniere P. et al in their study on disappearance of aluminic bone disease in a long term asymptomatic dialysis population have stated that osteomalacia is a common component of bone disease in patients with end stage renal disease, although its prevalence is decreasing.[5] The disorder is characterized by low rates of bone turnover, a mineralization defect, and an accumulation of unmineralized osteoid (bone matrix). The type of osteomalacia differs from that caused by Vitamin D deficiency, and the role of 1, 25-dihydrooxy-cholecalciferol deficiency in osteomalacia associated with end stage renal disease is unclear.[6] X-ray pelvis AP view in our patient clearly revealed looser's zones (pseudo fractures) bilaterally in inferior ischiopubic ramii.

According to the study of C Lewis et al, Osteoslerosis is seen in 20% of patients because the bony trabeculae look prominent in relation to decreased mineralization.[7] X-ray spine lateral view of our patient revealed dense vertebrae.

ROD is classified as osteitis fibrosa, osteomalacia, or mixed, mild, or adynamic disease; according to the histological features (Table 2).The frequency at the start of therapy for end stage renal disease is shown in this table and has been reported by Hutchison et al [8]. In our case no biopsy was done as case was diagnosed already by ultrasound as well as biochemical study. For the same she was referred to a higher centre for dialysis and further work up. On review we have placed our case in variety of mixed disease according to this classification as features of osteitis fibrosa were coexisting with osteomalacia.

CONCLUSION

The patient presented with simple backache without history of trauma. On paying attention to her associated complaint of stiffness initially it was thought she has ankylosing spondylitis (though less in females). It was only after persistent investigations along with clinicoradiological findings she emerged to be a case of renal osteodystrophy.

REFERENCES

1. Duthie R B, Bentley G. Metabolic, Hormonal and Bone Marrow Diseases. Mercer's Orthopedic Surgery, 9th Edition. New Delhi, Jaypee Brothers Medical Publishers (P) Ltd, 302-306.
2. Foreman JW, Chan JCM. Chronic renal failure in infants and children. Journal Paediatr 1988; 113:793-799.
3. Moxey-Mims M. Renal tubular disorders in the neonate. Clin Perinatol 1992; 19:159-178.
4. Eknoyan G, Levin A, Levin NW. Bone metabolism and disease in chronic kidney disease. Am J Kidney Dis 2003; 42 Suppl: 1130-3.
5. Moriniere P, Cohen-Solal A, Belbrik S, et al. Disappearance of aluminic bone disease in a long term asymptomatic dialysis population restricting AL(OH),intake: emergence of an idiopathic adynamic bone disease not related to aluminum. Nephron1989; 53:93-101.
6. Coburn JW, Slatopolsky E. Vitamin D, parathyroid hormone, and the renal osteodystrophies. The Kidney 4th edition, volume 2.Phildelphia: WB Saunders, 1991:2036-120.
7. C. Lewis, M. Brewster, A. Gadgil, R. Dias: Chronic Renal Failure Presenting Primarily With Renal Osteodystrophy: A Case Report. *The Internet Journal of Orthopedic Surgery*. 2005 Volume 2 Number 2. DOI: 10.5580/2928.
8. Hutchison AJ, Whitehouse RW, Boultan HF, et al. Correlation of bone histology with parathyroid hormone, vitaminD3, and radiology in end stage renal disease. Kidney Int 1933; 44:1071-7.