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The Pediatric Orthopaedic Society of North America

Renal Osteodystrophy

Objectives

1. Describe the skeletal features of renal osteodystrophy
2. Describe the chain of metabolic events responsible for the disordered skeletal features characteristic of renal osteodystrophy
3. Describe the clinical manifestations of renal osteodystrophy in the skeletally immature patient
4. Discuss laboratory values associated with renal osteodystrophy

Discussion points

1. What is the role of the orthopaedist in managing patients with renal osteodystrophy?
2. What is the rugger jersey spine?
3. Why do patients with renal osteodystrophy usually develop genu valgum and not genu varum?

Discussion

Renal osteodystrophy is a complex metabolic disorder that is seen much more frequently at present due to advances in the medical management of renal failure and the much more frequent occurrence of renal transplantation. The initial event is glomerular damage leading to phosphate retention and a reduction in production of 1,25 dihydroxyvitamin D. In addition hyperphosphatemia decreases the production of 1,25 dihydroxyvitamin D. Gut absorption of calcium is reduced, and perhaps even actively secreted. Marked secondary hyperparathyroidism results, which is ineffective in

increasing intestinal absorption of calcium or renal tubular reabsorption because vitamin D is present and hyperphosphatemia is already present. The end result to the skeleton is then a combination of rickets and secondary hyperparathyroidism. Ectopic calcification may occur if the solubility of serum calcium and phosphorus is exceeded; acidosis may further aggravate negative calcium balance. Skeletal manifestations include a mixture of osteomalacia, osteitis fibrosa cystica, osteosclerosis (poorly understood) and osteoporosis. Aluminum containing phosphate binders are often administered to control hyperphosphatemia, and a form of osteodystrophy resembling pure osteomalacia has been identified with aluminum administration has been identified.

The marked hyperparathyroidism results in significant reabsorption in metaphyseal bone, and this has been incriminated in the frequent occurrence of slipped capital femoral epiphysis in the skeletally immature population with renal osteodystrophy. Patterns of bone absorption are variable and together with osteitis fibrosa cystica can resemble neoplasia. A peculiar manifestation of renal osteodystrophy is the so-called rugger jersey spine, with alternating bands of osteosclerosis and rarefaction. Ectopic calcification may limit joint mobility.

Creatinine and BUN will obviously be elevated in patients with renal osteodystrophy. The serum calcium is low, and the phosphorus is elevated. Alkaline phosphatase and PTH are elevated. Vitamin D levels are reduced, urinary calcium is reduced, and fecal calcium is elevated.

Younger children at the age of physiologic bowing usually do not develop renal osteodystrophy; it is much more common in older children who have already developed physiologic genu valgum. The weakened bone of the lower extremity will tend to fail toward a worsening deformity as a result of weightbearing, thus genu valgum commonly accompanies renal osteodystrophy. If medial management or successful transplantation results in improved renal function, some improvement in alignment may occur. Obviously, internal fixation of bone weakened by renal osteodystrophy is tenuous, and prudence dictates a very discretionary approach to the correction of skeletal deformity in the patient with active renal osteodystrophy.

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