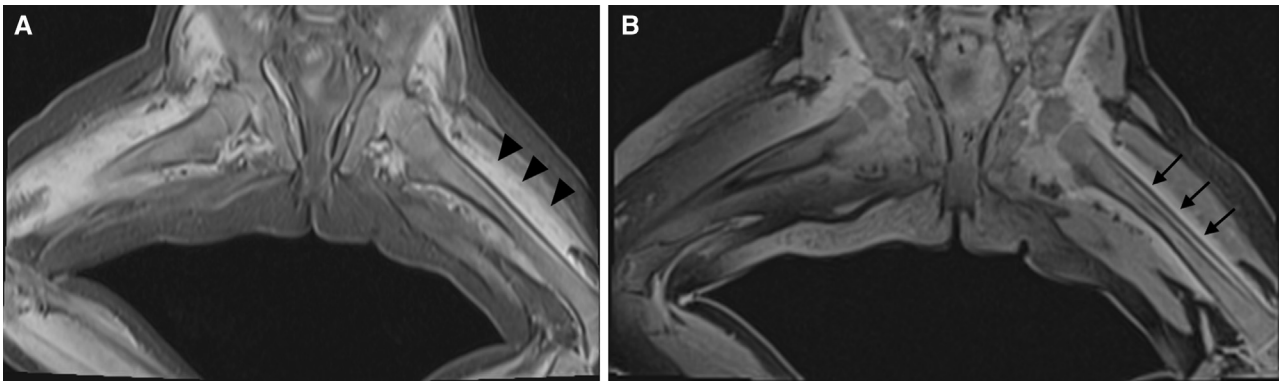

Figure 2. **A,** T2 turbo inversion recovery magnitude (slice thickness 3.0 mm; repetition time 4160 milliseconds; echo time 36 milliseconds; TI 140 milliseconds) image acquired on the coronal plane after 20 days highlights a worsening of the soft-tissue involvement (*black arrowheads*). **B,** T1 volumetric interpolated breath-hold examination 3-dimensional (slice thickness 0.5 mm; repetition time 16 milliseconds; echo time 6.29 milliseconds) image acquired on the coronal plane postcontrast medium administration with fat saturation highlights a diffuse bilateral cortical bone thickening of femur shaft with the typical aspect of "bone in bone" (*black arrows*).

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Reversible Myelofibrosis in Pediatric Renal Osteodystrophy

A 9-year-old female with no medical care for 3 years presented with recurrent emesis, epistaxis, and pallor. Laboratory evaluation showed renal failure, with a blood urea nitrogen level of 216 mg/dL and a serum creatinine concentration of 17.5 mg/dL. A hematologic workup showed severe normocytic anemia, with a hemoglobin concentration of 4.3 g/dL and an initial platelet count of 108×10^9/L that decreased to 60×10^9/L over 5 days. Her total white blood cell count was normal, but she had mild lymphopenia (1.3×10^9/L). Parathyroid hormone (PTH) levels were markedly elevated at 1600 pg/mL (normal range, 15-87 pg/mL). A peripheral blood smear review showed normocytic normochromic anemia with moderate anisopoikilocytosis, including rare acanthocytes, teardrop cells, and rare schistocytes. No blasts were present.

The cause of renal failure was not determined, but the patient had small bilateral kidneys on ultrasound, consistent with long-standing chronic kidney disease (CKD), possibly secondary to renal dysplasia. Bone marrow biopsy revealed hypocellularity (30%) and grade 2 nonuniform, patchy peritrabecular myelofibrosis with increased osteoclastic and osteoblastic activity (**Figure**, A). No immunophenotypic or cytogenetic abnormalities were detected, and testing for *JAK2V617F* was negative. The bone marrow findings in the setting of hyperparathyroidism due to long-standing CKD and the absence of other etiologies were consistent with nonmalignant myelofibrosis due to secondary hyperparathyroidism/renal osteodystrophy.

J Pediatr 2020;226:303-5.

https://doi.org/10.1016/j.jpeds.2020.07.051

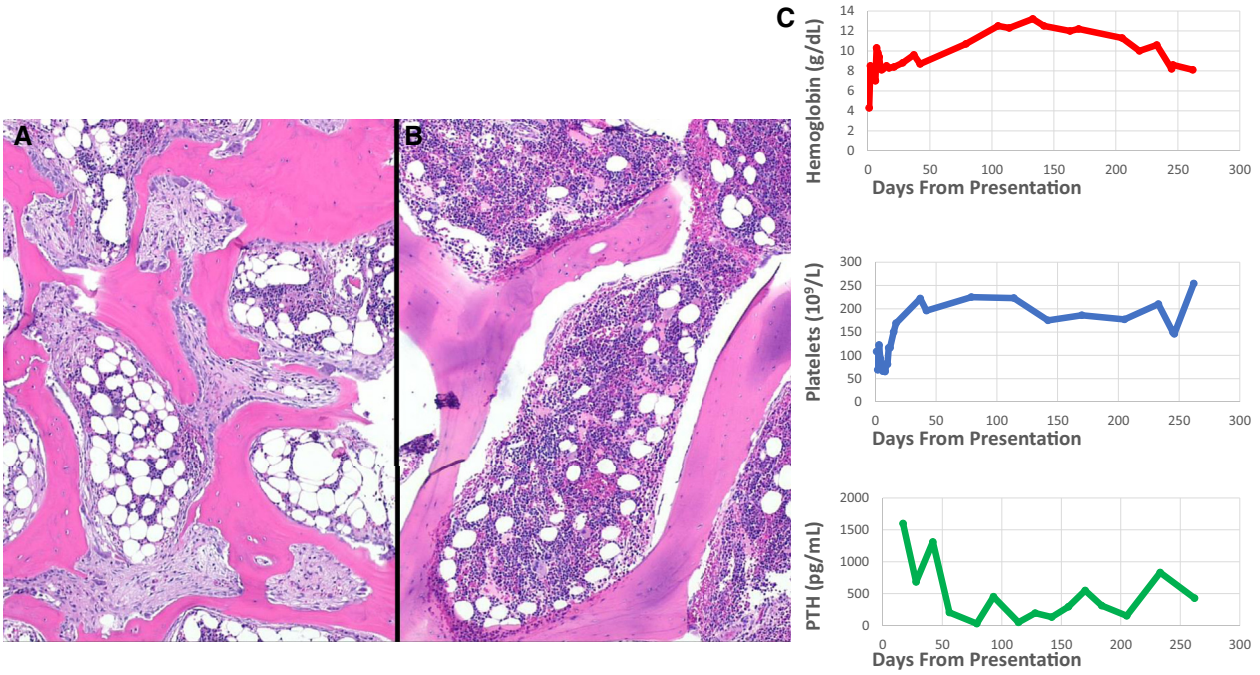

Figure. **A,** Bone marrow biopsy image showing hypocellular marrow with peritrabecular myelofibrosis, increased osteoclastic and osteoblastic activity, and anastomosing trabeculae at time of presentation. **B,** Normocellular marrow with no fibrosis and normal trabeculae after initiation of hemodialysis and control of hyperparathyroidism. **C,** Changes in hemoglobin concentration, platelet count, and PTH level over time.

The patient underwent prompt hemodialysis and was treated with paricalcitol for hyperparathyroidism. Platelet levels improved within 2 weeks, and hemoglobin normalized within 3 months of initiating the erythropoiesis-stimulating agent (**Figure**, C). PTH levels decreased within 2 months and remained mostly below 600 pg/mL. A bone marrow biopsy repeated 10 months later in anticipation of kidney transplantation showed complete resolution of myelofibrosis, improved cellularity (80%) with normal multilineage hematopoiesis, and unremarkable trabeculae (**Figure**, B).

Renal osteodystrophy is a complication of CKD that results from secondary hyperparathyroidism. The effects on the bone marrow and hematopoiesis are poorly understood, and myelofibrosis has been linked to hyperparathyroidism.[1,2] PTH up-regulates cytokine production, including platelet-derived growth factor, and may inhibit erythropoietin (EPO) production, causing myelofibrosis, pancytopenia, and EPO-refractory anemia in some patients with CKD.[3,4] The optimal treatment of myelofibrosis in secondary hyperparathyroidism is variable. Myelofibrosis was found to be ameliorated in patients with CKD after renal transplantation[1] and parathyroidectomy[5,6]; however, myelofibrosis developed or persisted in patients on chronic dialysis.[1,2,5,6] Myelofibrosis was also hypothesized to negatively affect outcomes of kidney transplantation.[7] Awareness of this rare complication in patients with CKD with pancytopenia or EPO-resistant anemia can limit unnecessary workup and should prompt intensive management of hyperparathyroidism, which may lead to reversal of myelofibrosis. ■

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Epiphyseal Cleft: A Misleading Radiologic Finding

A 2-year-old boy was referred to the emergency department because of temporary limping after falling from a wall (~80 cm). At admission, his physical examination was unremarkable; neither abnormal gait, pain, nor joint limitation in passive and active range of motion were observed. Due to the high-energy trauma, he underwent plain radiography, which showed a small, bipartite, and irregular epiphyseal nucleus of the right femoral head (**Figure 1**, A). Computed tomography and magnetic resonance imaging (MRI) were performed (**Figure 1**, B and C). Radiologic imaging demonstrated a double epiphyseal nucleus in which cortical bone and growth plate were preserved, and no lytic lesions were observed. Therefore, a diagnosis of epiphyseal cleft was made. After 6 months, the toddler underwent a second radiograph, showing the same, unchanged, radiologic findings. No pain or other complaints were reported.

Epiphyseal clefts or defects are uncommon anatomical variants of the growth plates. They can potentially occur in each epiphysis but are most frequently seen on the basal epiphysis of the proximal phalanx of the big toe. Epiphyseal clefts are accidentally found on radiographs in an otherwise-healthy child as a clear, shining line dividing the epiphysis into 2 parts.[1] A tomography scan and/or an MRI are usually needed to exclude cartilaginous or cortical bone disruption, as well as bone malignancies. The main differential diagnosis (**Figure 2**) encompasses epiphyseal fracture, Legg–Calve–Perthes disease, and Langerhans cell histiocytosis. The rare

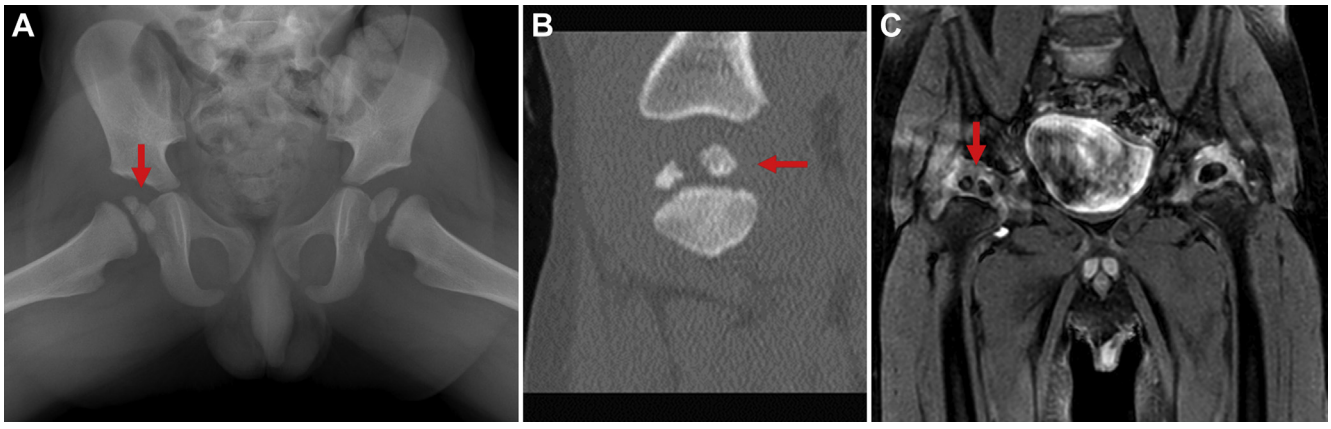

Figure 1. Epiphyseal cleft. **A,** On the hip radiograph, a radiolucent cleft (*red arrow*) divides into an irregular and flattened epiphyseal nucleus of the proximal femur. **B,** Computed tomography scan demonstrates a double epiphyseal nucleus (*red arrow*) without periosteal reaction, cortical disruption, or lytic lesion. **C,** Coronal MRI scan does not show any alteration of signal intensity (*red arrow*).

J Pediatr 2020;226:305-6.

https://doi.org/10.1016/j.jpeds.2020.07.068