structures, capable of detecting small UDs and identifying neoplasms[1,2,4–6,8]. In T-2 weighted MRI sequences, UDs show hyperintense signals, although they can be hypointense if they have thick content[1,2,4,6]. Solid tumor components present as vegetative lesions with intermediate signals on T1- and T2-weighted sequences, potentially restricting the diffusion, and show significant enhancement after intravenous administration of contrast[1,2].

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Chronic kernicterus: magnetic resonance imaging findings

Dear Editor,

A 3-year-old male child who had developed bilirubin encephalopathy in the neonatal period, due to Rh incompatibility, presented with delayed neuromotor/psychomotor development and involuntary movements. The prenatal and perinatal periods had been free of complications. Serology for cytomegalovirus, toxoplasmosis, and HIV were negative, as was the VDRL test. The results of a complete blood count, serum ceruloplasmin, electrolytes, and thyroid function were all within the limits of normality. Magnetic resonance imaging (MRI) of the brain showed bilateral, symmetrical hyperintense signals on FLAIR and T2-weighted sequences, affecting the globus pallidus and subthalamic nuclei, with no mass effect, with no diffusion restriction or evidence of gadolinium enhancement (Figure 1). Those imaging findings, together with the clinical and biochemical history, confirmed the suspected diagnosis of chronic kernicterus.

Recent studies conducted in Brazil have highlighted the importance of MRI studies to improving the diagnosis of central nervous system disorders[1–5]. Kernicterus, also known as bilirubin encephalopathy, is a rare complication of hyperbilirubinemia in childhood, occurring when serum bilirubin levels in the neonate are in excess of 20 mg/dL at term or even lower values in premature infants, which result in bilirubin deposition in the globus pallidus, subthalamic nuclei, hippocampus, putamen, thalamus, and cranial nerves, primarily the third, fourth, and sixth cranial nerves[6]. Symptoms include drowsiness, hypotonia, opisthotonus, rigidity, and seizures. The factors involved in its pathogenesis are hyperbilirubinemia, reduced serum bilirubin binding capacity, changes in the permeability of blood-brain barrier, and neurotoxicity. Although the main causes of kernicterus are ABO and Rh mismatches, it can also be caused by sepsis and other types of hemolytic anemia such as glucose-6-phosphate dehydrogenase deficiency[7]. The clinical symptoms and signs can regress completely if properly treated with phototherapy and blood transfusions[6]; without treatment, permanent damage can occur, generating encephalopathy with symptoms related to the basal nuclei, including involuntary movements, asymmetric spasticity, rigidity, ataxia, and hearing loss[8].

The MRI findings in kernicterus are characterized by a hyperintense signal on T1-weighted sequences in the globus pallidus, progressing chronically to a shift from a hyperintense signal on T1-weighted sequences to a bilateral, symmetrical hyperintense signal on T2-weighted and FLAIR sequences in the globus pallidus and subthalamic nuclei[7,9–11], corresponding to the areas of preferential deposition of unconjugated bilirubin, characterizing chronic kernicterus, as in the case presented here.

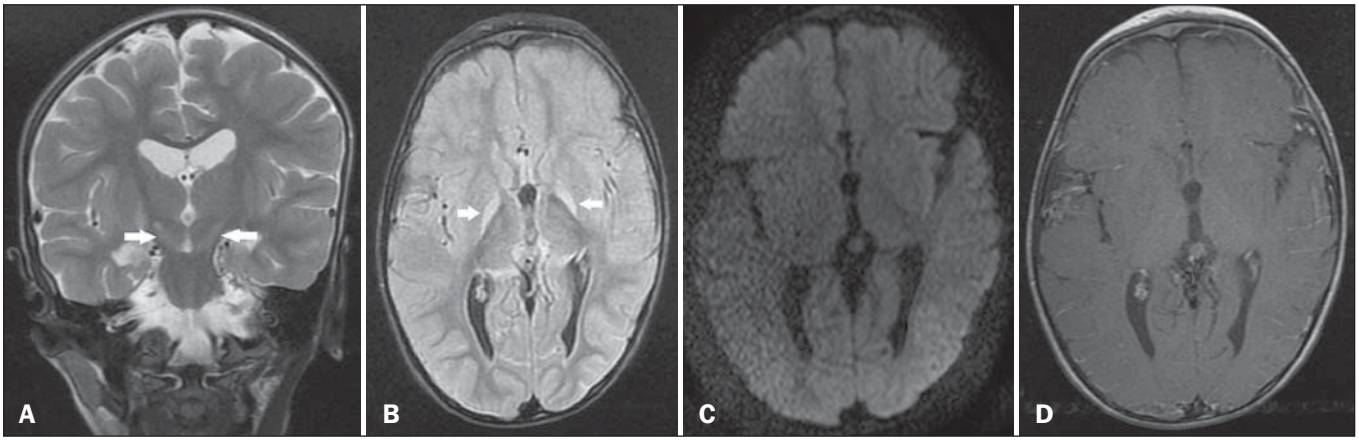

Figure 1. A: Coronal T2-weighted MRI sequence showing a bilateral, symmetrical hyperintense signal in the subthalamic nuclei (arrows), without a mass effect. **B:** Axial FLAIR MRI sequence showing a bilateral, symmetrical hyperintense signal in the globus pallidus (arrows). **C:** Axial diffusion-weighted MRI sequence showing no diffusion restriction. **D:** Axial T1-weighted MRI sequence showing no evidence of gadolinium enhancement.

There is a broad spectrum of diagnoses of bilateral lesions in the basal ganglia in the pediatric population. The main causes cited are hypoxic-ischemic encephalopathy; hypoglycemia; encephalitis; inborn errors of metabolism; water and electrolyte disturbances; carbon monoxide poisoning; and demyelinating disorders. The correlation with clinical and laboratory data is fundamental for making the definitive diagnosis[7,12,13].

In conclusion, the possibility of acute or chronic kernicterus should be considered when clinical symptoms, biochemical data, and MRI findings are suggestive of the disease, the chronic presentation and permanent, irreversible profile being promoted by bilirubin neurotoxicity.

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Renal lymphangiectasia: know it in order to diagnose it

Dear Editor,

Here, we report the case of a 9-year-old girl with hyperparathyroidism. Ultrasound showed renal cysts and increased echogenicity of the parenchyma in both kidneys. The diagnostic hypothesis was hyperparathyroidism secondary to chronic/polycystic kidney disease. The patient presented with gradually worsening kidney function and hypertension, and new imaging scans were requested. The ultrasound showed anechoic, multiloculated images in the pyelocaliceal region of both kidneys, and perirenal, subcapsular cysts. A computed tomography (CT) scan was acquired, although no contrast agent was used, which precluded an accurate characterization. Nevertheless, the CT scan revealed changes similar to those observed on ultrasound. We also performed magnetic resonance imaging (MRI), which showed pyelocaliceal, perirenal cysts, with altered intensity of the signal of the renal parenchyma and loss of corticomedullary differentiation (Figure 1A), confirming, in conjunction with the clinical and biochemical data, the diagnosis of renal lymphangiectasia (RL).

RL is a rare benign disease that occurs because of miscommunication between the renal lymphatic drainage system and the retroperitoneal lymphatic system[1]. As a result, there is accumulation of lymph in the renal lymph ducts, making them ectatic and forming simple or multiloculated, typically asymmetric and bilateral, collections in the pyelocaliceal, perinephric, or parenchymal regions, although, in some cases, only a part of one kidney is affected (Figure 1—B,C). There is no predilection for a given gender or age group. As of 2005, only 40 cases had been described[1,2].

In most cases, RL is an incidental finding, with or without signs and symptoms of pain, increased abdominal volume, hematuria, ascites, edema of the lower limbs, hypertension, erythrocytosis with renal vein thrombosis, and, rarely, chyluria[3]. Such manifestations can be explained by the distention of the renal fascia and compression of the renal parenchyma by cysts, fistulization to the pelvic cavity, and changes in the renin-angiotensin system[2–4]. In rare cases, chronic kidney disease has been reported[5]. To our knowledge, there have been no specific reports of clinical evolution to hyperparathyroidism, although a relationship with chronic kidney disease can be assumed.

A CT scan can reveal expansive perirenal formations with fluid attenuation, bounded by the renal fascia, that conform to

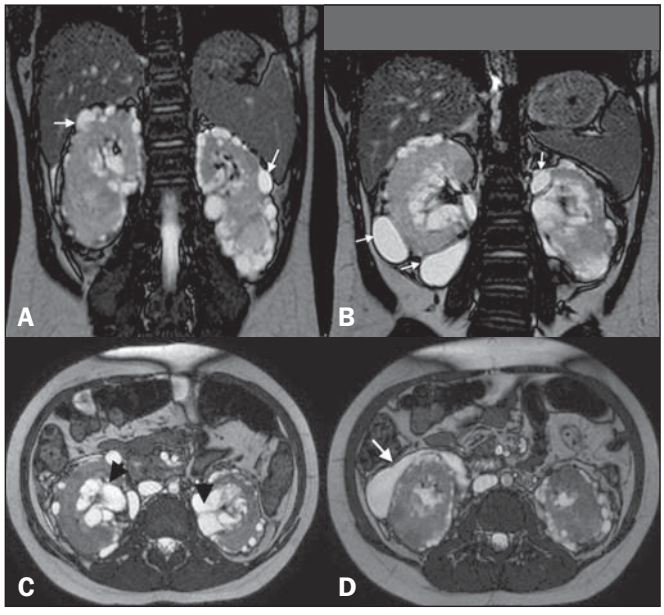

Figure 1. A: Coronal T2-weighted MRI sequence showing a loss of corticomedullary differentiation in both kidneys and multiple cystic lesions, with thin walls, located in the cortex (arrows). **B:** Cystic formations in the subcapsular cortex (arrows). **C:** Axial T2-weighted MRI sequence showing cysts located in the renal sinuses (arrowheads) and perinephric spaces, simulating pelvic dilatation. **D:** The same images simulating cystic collections in the subcapsular cortex (arrow).