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Case Report

Interstitial nephritis: Two pediatric cases with atypical radiological features

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ABSTRACT

Interstitial nephritis (IN) is a relatively rare entity in children and adolescents that can be caused by a range of disorders including infection, medications, inflammatory bowel disease, and sarcoid. There is no proven therapy for this condition. We present 2 cases of biopsy-proven interstitial nephritis, of which 1 case was with granulomatous features that presented with unusual sonographic findings of discrete mass lesions in the kidney parenchyma bilaterally. Although a precise cause could not be identified in either case, 1 patient progressed to end-stage kidney disease (ESKD) and the other is in the early stages of treatment. We suggest that recognition of the atypical imaging features of interstitial nephritis may enable early recognition of this condition and avoid confusion with neoplastic or infectious processes.

Introduction

Interstitial nephritis (IN) is a relatively rare disorder in pediatrics that can be caused by a wide range of infections (eg, mycoplasma), prescription drugs, and autoimmune disorders (eg, sarcoidosis and inflammatory bowel disease; Ref. 1]. In a subset of cases, a discrete cause cannot be identified and they are characterized as idiopathic. The diagnosis is established by kidney biopsy demonstrating interstitial edema and infiltration with inflammatory cells including neutrophils, eosinophils, and plasma cells [1]. Granulomatous interstitial nephritis (GIN) is a subtype of IN accounting for 0.9%-5.9% of cases that also occur in association with numerous medications, infections, and autoimmune disorders [2]. The prognosis of IN depends on the cause, extent, and duration of the disease [3]. Prompt withdrawal of a causative medication is generally associated with a good outcome, whereas poorer outcomes are seen in cases with diffuse inflammation, increased neutrophil infiltration, the presence of granulomas, and extensive fibrosis [1–3].

Renal biopsy remains the gold standard for diagnosis of all forms of IN, including GIN [3]. IN is characterized radiographically by increased kidney size and diffuse increased echogenicity on ultrasound (US) imaging [1,3]. In cases of

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Table 1 – Laboratory data.

	Patient 1		Patient 2	
	Presenting	Last follow-up	Presenting	Last follow-up
Creatinine	1.28	4.75	2.4	2
BUN	22	122	37	28
Urine protein: creatinine ratio	1.82	0.5	0.8	
Albumin	1.9	4.6		4.6
Ca	5.7	7.7	8.4	9.8
QuantiFERON	Indeterminate (A)		Negative	
ANA	<40		1:80	
ANCA	<1:20		Negative	
DsDNA			Negative	
ACE			71	
ASLO			111	

sarcoidosis-related GIN, there can be pseudotumoral masses that are histologically consistent with the granulomatous lesions [4]. In pediatric patients, presentation of IN with mass-like lesions on US is exceedingly rare. There is a single case report describing GIN in a child with sarcoidosis who had mass-like lesions on computerized tomography (CT) and US imaging [5]. We present 2 cases of children with IN, of which one was with GIN, who presented in the last 2 years and in whom US imaging revealed striking bilateral mass-like lesions without diffusely increased echogenicity. We highlight the importance of early recognition and timely diagnosis with a kidney biopsy in these cases because of the risk for progression to end-stage kidney disease (ESKD).

Case reports

Case 1 is a 17-year-old white male with a history of autistic spectrum disorder, Crohn's disease, and pulmonary embolism. He had history of chronic iron-deficiency anemia of unclear etiology and he was treated with intravenous iron infusions. For his Crohn's disease, he received weekly infusions of adalimumab for 1 year. The antibody was discontinued when he was noted to have an elevated serum creatinine and low serum calcium level, 1.28 mg/dL and 5.7 mg/dL, respectively (Table 1). He was referred for a second opinion evaluation because of the abnormal renal function. Testing for sarcoidosis and TB was negative.

A renal ultrasound revealed bilateral, discrete hypovascular echogenic mass-like lesions (Fig. 1a) that were also visualized on axial T2-weighted MRI images (Fig. 1b). A percutaneous kidney biopsy demonstrated IN with widespread interstitial fibrosis and tubular atrophy (Fig. 1c).

He was treated with high-dose corticosteroids for the IN with a partial response. In June 2015, he had worsening of his kidney function and was diagnosed with pulmonary emboli. He was placed on enoxaparin. Over the next 2 years, his kidney function gradually deteriorated, complicated by hypertension. Despite retreatment with steroids in January 2016, his serum creatinine level continued to increase (2.39 mg/dL). His BUN and serum creatinine rose to 120 mg/dL and 4.5 mg/dL, respectively, and he was started on hemodialysis in June 2017. He received a kidney transplant from a living donor in July 2017 and is currently doing well without evidence of disease recurrence.

Case 2 is an 18-year-old black male admitted for a hypertensive crisis, BP 180/120 mm Hg. He had a history of constipation and gastroesophageal reflux. He presented with dizziness, weakness, and headache for 2 days prior to admission. There was no prior history of renal disease, UTI, gross hematuria, abnormal urinalysis, or edema. Testing for sarcoidosis and TB was negative. A renal ultrasound showed bilateral hypovascular echogenic mass-like lesions (Fig. 2a). There was no diffuse increased echogenicity. The lesions were also seen in axial T2-weighted MRI images (Fig. 2b) A kidney biopsy showed GIN (Fig. 2c). IgG4-related tubulointerstitial nephritis was excluded [6]. He was started on prednisone 1 mg/kg per day and mycophenolate mofetil was added to his regimen to facilitate resolution of the interstitial inflammation. He developed a UTI 1 week after discharge and was treated with amoxicillin. At his last follow-up, his BUN and serum creatinine concentrations were 24 mg/dL and 1.9 mg/dL, respectively (Table 1).

Discussion

We present 2 cases of IN in pediatric patients, of which 1 was with GIN that presented with impaired kidney function in the absence of evidence of glomerular disease. In both cases, a renal ultrasound revealed striking evidence of discrete mass-like lesions distributed heterogeneously throughout both kidneys without diffusely enhanced echogenicity. The findings were confirmed by MRI. The differential diagnosis of multiple bilateral solid renal lesions in pediatric patients includes nephroblastomatosis, angiomyolipomas, and lymphoma. In both cases, the age of the patients spoke against nephroblastomatosis and the echogenicity of the lesions against angiomyolipomas or lymphoma. Finally, in the setting of Crohn's disease, granulomatous renal inflammation is a consideration. The appearance of IN as echogenic mass-like lesions is exceedingly rare and it is not on the standard list of possible causes of this ultrasound finding.