CASE REPORT

Serial sonographic findings during progression from acute pyelonephritis to renal abscess: a rare case report

Masayuki Sato¹ · Shigeru Suzuki¹ · Sorachi Shimada¹ · Shiho Yamamoto¹ · Genya Taketazu¹ · Tokuo Mukai¹ · Mio Taketazu¹ · Hiroshi Sakata¹ · Junichi Oki¹

Received: 28 June 2016 / Accepted: 20 September 2016 / Published online: 24 September 2016
© Japanese Society of Nephrology 2016

Abstract Renal abscess, accumulation of infective fluid in the kidney, is a rare pathology. Currently, no reports of the serial imaging changes of acute pyelonephritis (APN) progressing to renal abscess exist. We report clinical and serial sonographic findings of a patient with hyper-immunoglobulin E syndrome, a primary immunodeficiency, who developed APN that progressed to renal abscess. Renal ultrasonography revealed that echogenicity of infectious lesions dramatically changed from isoechoic to hyperechoic and to hypoechoic during progression. These findings are useful for differential diagnosis of APN, acute focal bacterial nephritis, and renal abscess.

Keywords Acute pyelonephritis · Acute focal bacterial nephritis · Renal abscess · Sonography · Echogenicity · Hyperimmunoglobulin E syndrome

Introduction

Upper UTI is classified as acute pyelonephritis (APN), acute focal bacterial nephritis (AFBN) or renal abscesses [1, 2]. From the points of view of imaging studies, these diagnoses are defined as follows: APN appears normal kidney images without mass lesions whereas both AFBN and renal abscesses show mass lesions [3]. The masses seen in AFBN do not present with liquefaction but those in renal abscesses have walled-off cavities with liquefaction [1, 2]. Renal abscesses, mostly believed to be progressive

lesions from urologic infections such as APN and AFBN [4], can occur secondary to hematogenous seeding; they are very rare and are often accidentally detected while examining for idiopathic fever, when the abscesses are completed. Probably for these reasons, there are no reports that demonstrate the serial imaging changes associated with APN progressing to renal abscess. We experienced the case of a 6-month-old boy with hyper-immunoglobulin E syndrome (HIES), a rare primary immunodeficiency, who developed APN that progressed to renal abscess despite the fact that pathogenic bacteria of the first urine culture was susceptible to initial antibiotic therapy which was administered since the early stage of the disease. We describe here the serial sonographic findings of the patient. This report provides valuable information for ultrasonographic staging of urinary tract infection.

Case report

A 6-month-old boy was admitted to our pediatric department because of high fever (39.4 °C) for a few hours. He had been clinically diagnosed previously with HIES on the basis of recurrent cutaneous *Staphylococcus aureus* infection, atopic dermatitis, elevated serum immunoglobulin E level (270 IU/mL at the age of 5 months), and family history of recurrent staphylococcal infection. Physical examination on admission revealed no sign of respiratory tract infection or skin abscess. Urinalysis revealed pyuria (urinary sediments: white blood cells, 5–9 per high power field) and a positive on nitrite test. A gram-negative rod-like bacteria suggesting *Escherichia coli* was detected in a gram-stained smear of centrifuged urine deposit. White blood cell count elevated to 16,800/μL, whereas the levels of C-reactive protein (CRP) and procalcitonin were normal

✉ Masayuki Sato
satou5p@asahikawa-med.ac.jp

¹ Department of Pediatrics, Asahikawa-Kosei General Hospital, 24-111, 1-jodori, Asahikawa 078-8211, Japan

(0.17 mg/dL and 0.22 ng/mL, respectively). We suspected a UTI and initially treated him with cefazolin dosed at 20 mg/kg every 8 h. Sonographic findings on admission revealed small perinephric effusion only (Fig. 1a). The urine culture examination on admission revealed growth of *E. coli* with a density of 10^7 /mL, which confirmed UTI. Although the *E. coli* detected in his urine was sensitive to cefazolin and the blood culture on admission was negative, his high-grade fever persisted with a remarkably elevated CRP level of 14.75 mg/dL. A repeat gram staining of urine on the third hospital day revealed gram-negative rod-like bacteria, suggesting *Pseudomonas aeruginosa*. Thus, we

changed the antibiotic to doripenem dosed at 26 mg/kg every 8 h. Color Doppler sonography on the fourth hospital day revealed a hyperechoic mass with poor vascularity at both upper and lower poles of the right kidney, which indicated AFBN (Fig. 1b). Because the second urine culture on the third hospital day confirmed *P. aeruginosa* infection with a density of 10^4 /mL and the second blood culture on the same day was negative, doripenem was continued. His high-grade fever nevertheless persisted, and we performed a contrast-enhanced computed tomography (CT) scan on the sixth hospital day. CT imaging showed 2 low-density lesions at both upper and lower poles of the

Fig. 1 Serial sonographic findings of the right kidney. **a** On admission, perinephric effusion (arrow) was detected. No masses were detected. **b** On the fourth hospital day, high-echogenic masses were detected at upper (arrow) and lower poles (arrowhead) of the right kidney. **c** On the sixth hospital day, echogenicity of the masses

decreased. **d** On the 10th hospital day, echogenicity changed to hypoechoic. **e** On the 16th hospital day, the size of masses reduced. **f** On the 23rd hospital day, masses almost regressed and echogenicity returned to normal

right kidney (Fig. 2) which indicated liquefaction. On the basis of the finding of CT, we concluded that his AFBN progressed to renal abscess. The hyperechoic masses on the fourth hospital day changed to isoechoic avascular masses on the same day on which CT was performed (Fig. 1c). These masses did not show low echogenicity though CT showed liquefaction. Therefore, we speculate that the sonographic finding in Fig. 1c showed the transition from AFBN to renal abscess because the echogenicity became lower in Fig. 1c than in Fig. 1b. Administration dose of doripenem was increased to 39 mg/kg every 8 h, and intravenous immunoglobulin was administered for 3 days. His fever alleviated after the seventh hospital day and normalized on the 11th hospital day. Doripenem was changed to oral tosufloxacin dosed at 6 mg/kg every 12 h on the 14th hospital day. His CRP levels normalized on the 17th hospital day, and tosufloxacin was discontinued on the 20th hospital day. The masses on sonography changed to hypoechoic with well-defined borders on the 10th hospital day (Fig. 1d) which indicated progression of liquefaction. Then, the mass on lower pole of kidney decreased in size and changed to isoechoic on the 16th hospital day though

the hypoechoic mass on upper pole of kidney was still detected (Fig. 1e). We supposed the liquefaction was ceasing, and renal abscesses were on healing process. Renal abscesses were almost regressed by the 23rd hospital day (Fig. 1f). Voiding cystourethrography performed 2 weeks later revealed grade 2 vesicoureteral reflux in his right ureter. Therefore, we administered continuous antibiotic prophylaxis of trimethoprim/sulfamethoxazole.

Discussion

Upper UTI is a crucial bacterial infection in infants. It includes various conditions such as APN, AFBN, and renal abscess [1], and APN is the most common form of upper UTI. As the infection progressed, APN deteriorates to AFBN and renal abscess. The severity of disease is mainly determined using imaging studies such as sonography and contrast-enhanced CT [1, 2]. On ultrasonography, most APN appears normal; however, various findings can be revealed, such as single or multiple hyperechoic areas, kidney enlargement, pelvic wall thickening, hypoechoic areas, and perirenal fat involvement [3]. AFBN is characterized by renal masses without liquefaction on sonography or CT, and it can have either hyperechoic or hypoechoic echogenicity [1, 5–8]. Rathaus et al. reported that 47 of 52 cases of AFBN showed a hyperechoic mass, whereas 5 cases showed hypoechoic [1]. Similarly, Farmer et al. reported 17 cases; 12 cases were hyperechoic, 3 were hypoechoic, and 2 were mixed [5]. AFBN further progresses to renal abscess, which is the most severe upper UTI [1]. Renal abscess is seen as intrarenal walled-off cavities with liquefaction on sonography [2].

In our case, APN deteriorated to renal abscess despite the fact that the *E. coli* detected by urine culture on admission was sensitive to cefazolin. In the course of disease progression, we obtained serial sonographic findings of different stages of UTI, echogenicity of which changed from isoechoic to hyperechoic, and finally hypoechoic. Considering our serial sonographic findings in the same patient, previous conflicting reports of AFBN echogenicity may simply be because of a different phase of the disease. That is, a hyperechoic lesion may indicate the incipient stage of AFBN and its echogenicity may change to hypoechoic with progression. Rigsby et al. suggested that increased echogenicity reflects an acute hemorrhage resulting from the venous abnormalities and obstruction [2, 8], which was evidenced by one of the histopathological findings of AFBN [2]. Although there are no reports on serial histopathological findings of AFBN, low echogenicity followed by high echogenicity may reflect liquefied lesions progressing to renal abscess.

Fig. 2 Enhanced CT on the sixth hospital day. Renal abscesses were detected at upper (sized 20 × 27 × 32 mm) and lower (sized 23 × 26 × 25 mm) pole of the right kidney

AFBN is also known to show poor vascularity on ultrasonographic color Doppler imaging [1], which was confirmed with our findings of poor vascularity during both hyperechoic and hypoechoic AFBN phases. Previous reports suggested that poor vascularity of the lesions could be due to the compression of veins by the mass itself or due to the narrowing of the veins by interstitial inflammation of the kidney [1, 2]. Evaluation of vascularity in renal lesions is useful for differential diagnosis of AFBN and APN, particularly when patients with UTI are refractory to antibiotics.

Our patient had been diagnosed with HIES, which is characterized by recurrent staphylococcal infection of the skin and respiratory tract, atopic dermatitis, and elevated IgE levels [9]. Patients with HIES are more susceptible to infection with extracellular pathogens. While *S aureus* is the most frequent etiology, other organisms such as *Streptococcus pneumoniae*, *Haemophilus* species, *P. aeruginosa*, and *Aspergillus* can also cause infections. The commonly affected organs are skin, lung, sinus, and ear. There are a few reports of HIES complicated with renal abscess [10, 11]. Our patient also developed renal abscess despite appropriate initial therapy for APN. Although the initial treatment failed, the antibiotic therapy might have slowed disease progression, which made sonographic evaluation of each stage possible.

In conclusion, this is the first report of serial sonographic findings of APN progressing to renal abscess in the same patient. We demonstrated that echogenicity of the infectious lesions dramatically changed from isoechoic to hyperechoic, and finally hypoechoic during its progression. These findings should emphasize the utility of ultrasonography in the diagnosis and follow-up of UTI at different stages.

Acknowledgments The authors thank Mr. Takahiro Yamagishi and Mr. Shingo Okazaki who performed ultrasonography. They also thank Dr. Hironori Takahashi from the Department of Pediatrics,

Asahikawa Medical University, for his expert advice on managing renal abscess.

Compliance with ethical standards

Conflict of interest The authors have declared that no Conflict of interest exists.

This article does not contain any studies with animals performed by any authors.

Informed consent was obtained from all individuals participants included in the study.

References

1. Rathaus V, Werner M. Acute focal nephritis: its true sonographic face. *Isr Med Assoc J*. 2007;9:729–31.
2. Rosenfield AT, Glickman MG, Taylor KJ, Crade M, Hodson J. Acute focal bacterial nephritis (acute lobar nephronia). *Radiology*. 1979;132:553–61.
3. Rollino C, Beltrame G, Ferro M, Quattrocchio G, Sandrone M, Quarello F. Acute pyelonephritis in adults: a case series of 223 patients. *Nephrol Dial Transplant*. 2012;27:3488–93.
4. Saiki J, Vaziri ND, Barton C. Perinephric and intranephric abscesses: a review of the literature. *West J Med*. 1982;136:95–102.
5. Farmer KD, Gellett LR, Dubbins PA. The sonographic appearance of acute focal pyelonephritis: 8 years experience. *Clin Radiol*. 2002;57:483–7.
6. Lee JKT, McClennan BL, Melson GL, Stanley RJ. Acute focal bacterial nephritis: emphasis on gray scale sonography and computed tomography. *AJR Am J Roentgenol*. 1980;135:87–92.
7. Siegel ML, Glasier CM. Acute focal bacterial nephritis in children: significance of the ureteral reflux. *Am J Roentgenol*. 1981;137:257–60.
8. Rigsby CM, Rosenfield AT, Glickman MG, Hodson J. Hemorrhagic focal lobar nephritis: findings on gray-scale sonography and CT. *AJR Am J Roentgenol*. 1986;146:1173–7.
9. Freeman AF, Holland SM. Clinical manifestations of hyper IgE syndromes. *Dis Mark*. 2010;29:123–30.
10. Roxo P, Menezes UP, Tucci S Jr, Andrade MF, Silva GE, Melo JM. Renal abscess in hyper-IgE syndrome. *Urology*. 2013;81:414–6.
11. Yükksekaya H, Ozbek O, Goncu F, Keser M, Hasibe A, Harun P, Keles S. Hyperimmunoglobulin E syndrome presenting with renal abscess. *J Invest Allergol Clin Immunol*. 2012;22:143–5.