

Case report

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소아에서 방광 요관역류가 동반된 급성 신우신염 및 신장 농양으로 인한 급성 신부전 1례

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Acute Kidney Injury Accompanied by Acute Pylonephritis and Renal Abscess in a Child with Vesicoureteral Reflux

Acute pyelonephritis (APN) is a relatively common bacterial infection in children. In previously healthy children, acute kidney injury (AKI) is very rare in the course of APN without urinary tract obstruction, renal hypoperfusion due to hypotension or exposure to nephrotoxic agents. We describe a case of AKI secondary to APN and renal abscess in a child with vesicoureteral reflux. With antibiotic treatment and adequate hydration, the patient was improved. APN should be included in the differential diagnosis of AKI and adequate treatment by proper antibiotics is crucial for full recovery of renal function.

Key Words: Abscess, Acute kidney injury, Pyelonephritis

Introduction

Acute pyelonephritis (APN) is one of the most severe form of urinary tract infection (UTI) in children. It is an unusual cause of acute kidney injury (AKI) in children with an anatomically normal urinary tract and no other predisposing conditions such as the presence of an indwelling catheter, use of immunosuppressive drugs, renal stones, solitary kidney, or non-steroidal analgesic use [1]. According to our knowledge, only a few cases of AKI due to APN have been described in children in the literature [1-4]. Here, we report a 9-year-old girl presenting with AKI secondary to APN and renal abscess who was successfully recovered with conventional antibiotics and supportive therapy.

Case report

A previously healthy 9-year-old girl was referred to our hospital because of fever, vomiting and oliguria without other urinary symptoms for two days. There was no frequent history of febrile illness nor family history of kidney disease. The history of antibiotic use prior to admission was absent. On admission, her temperature was 38°C, blood pressure was 100/70 mmHg. Physical examination was unremarkable and costovertebral angle tenderness was absent. Peripheral blood analysis indicated as follows: hemoglobin 11.7 g/dL, white blood cell (WBC) counts 26,400/mm³, platelet 185,000/mm³, erythrocyte sedimentation rate 86 mm/hr, C-reactive protein 41.1 mg/dL (normal range <0.3 mg/dL), blood urea nitrogen 50 mg/dL, creatinine 1.5 mg/dL, AST 37 IU/L, ALT 27 IU/L, protein 6.2 g/dL, albumin 3.7 g/dL. Electrolyte values were sodium 136.7 mmol/L, potassium 3.92 mmol/L, chloride 101.2 mmol/L and total CO₂ 21.2 mmol/L. The urine analysis showed protein 2+, 5-9 WBC/high power field, nitrites negative, pH 5.0 and specific gravity 1.030. Random urine electrolytes were sodium 39.3 mmol/L, potassium 57.3 mmol/L, chloride 10.0 mmol/L and osmolality 325 mosml/kg. The fractional excretion of sodium (FE_{Na}) was 0.37%. C3 and C4 were normal. Antinuclear antibody, anti-neutrophil cytoplasmic antibody, anti-streptolysin O, and anti-extractable nuclear antigen profile were

all negative. Creatinine clearance was 51.3 mL/min/1.73m² and no bacteria was grown on blood and urine cultures. Abdomen ultrasonogram (US) showed enlarged left kidney with a hydrocalyx (the length of the left kidney was 11.29 cm and of the right kidney was 7.85 cm). On abdomen color doppler examination, the result was normal. ^{99m}Tc-dimercaptosuccinic acid (DMSA) renal scan showed multiple cortical defects of both kidneys and decreased relative uptake of the right kidney (right kidney 35.87% and left kidney 64.13%, respectively) (Fig. 1A). We suspected a renal abscess because of DMSA renal scan defect pattern, longer duration of fever and continued pyuria. In 10 days after hospitalization, the blood analysis results (e.g. blood urea nitrogen, creatinine) improved to normal and we conducted abdominal computed tomography (CT) with sufficient hydration. CT showed two renal abscesses on the left kidney (size 1.7 cm and 1.2 cm, respectively) (Fig. 1B). The patient was hydrated and treated with diuretics and intravenous antibiotics (cefotaxime and gentamicin). About three weeks later, clinical symptoms were subsided and blood analysis also improved as follows (Fig. 2): hemoglobin 10.9 g/dL, white blood cell 7,700/mm³, platelet 393,000/mm³, C-reactive protein 0.4 mg/dL, blood urea nitrogen 10 mg/dL, and creatinine 0.7 mg/dL. Likewise, urine analysis did not show proteinuria and pyuria. Ultrasonographically, renal abscesses also disappeared. Voiding cystourethrography showed grade III/V of right vesicoureteral reflux (VUR) (Fig.

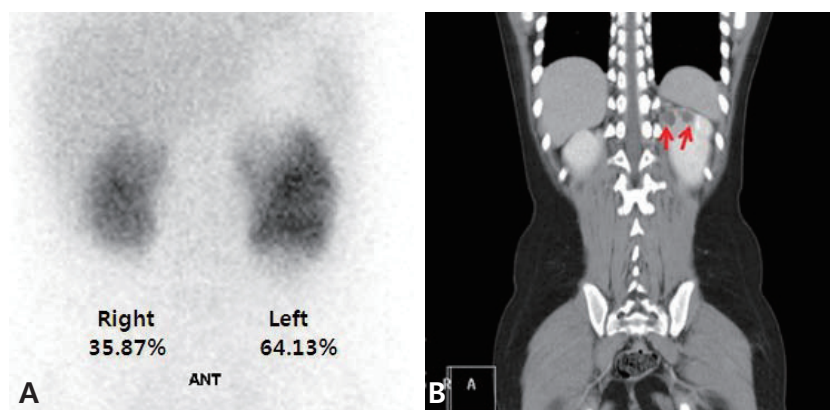


Fig. 1. (A) ^{99m}Tc-dimercaptosuccinic acid renal scans showing a smaller right kidney contributing 35.87% relative uptake and multiple cortical defects of both kidneys (anterior view). (B) Abdominal computed tomography demonstrating two renal abscesses on left kidney (arrows) (size 1.7 cm and 1.2 cm, respectively).

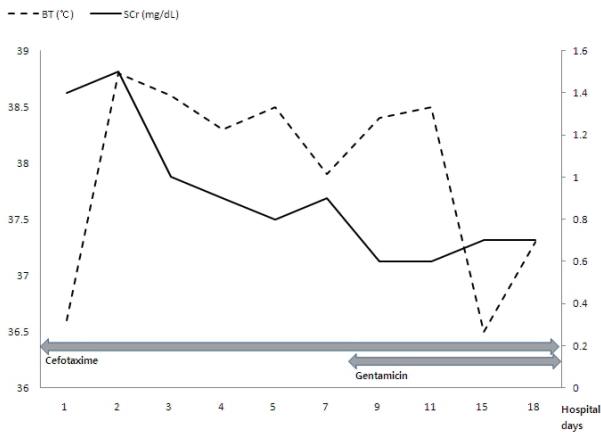


Fig. 2. Duration of antibiotic use (cefotaxime, gentamicin) and clinical course : sequential changes of body temperature (BT) and serum creatinine (SCr).

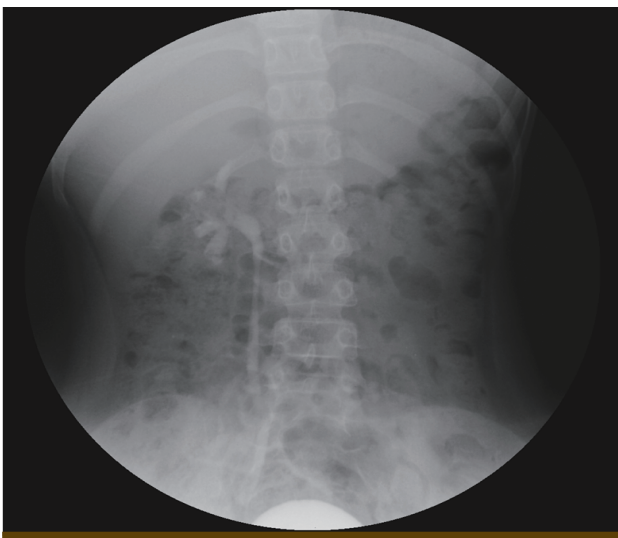


Fig. 3. Voiding cystourethrography showing grade III/IV of right vesicoureteral reflux.

3). We recommended an operation for VUR, however, her parents refused and she has been followed up with prophylactic antibiotics after discharge. In outpatient department, we performed follow-up DMSA renal scan at 6 months after discharge. There was no cortical defect of both kidneys and almost no interval change in relative uptake of both kidneys (right kidney 36.16% and left kidney 63.84%, respectively) (Fig. 4).

Discussion

AKI is characterized by the inability of the kidney to

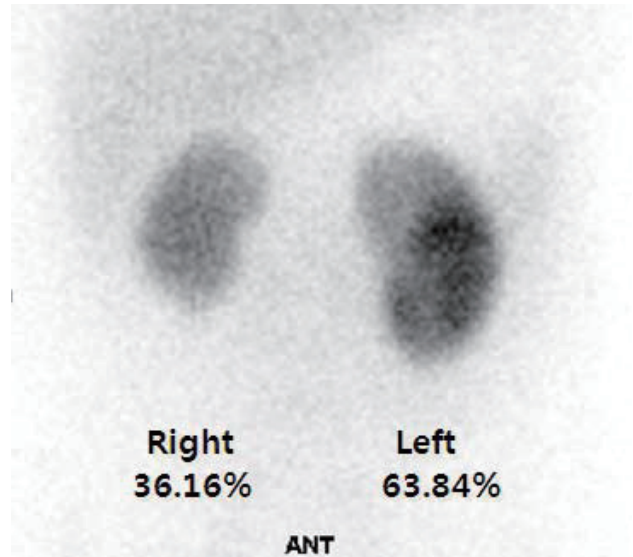


Fig. 4. Follow-up ^{99m}Tc-dimercaptosuccinic acid renal scan showing smaller right kidney contributing 36.16% relative uptake and no cortical defect of both kidneys at 6 months after discharge (anterior view).

regulate fluid and electrolyte homeostasis appropriately [5]. AKI occurs in 2-3% of children admitted to pediatric tertiary care centers and in as many as 8% of infants in neonatal intensive care units [6]. The etiology of AKI over the past decades has been shifted from primary renal disease to multifactorial causes, particularly in hospitalized children. Hui-Stickle S et al [7] conducted a retrospective review of 254 AKI episodes in 248 children discharged from a tertiary referral center between January 1998 and June 2001. Data showed that AKI most often occurred a morbidity associated with other systemic illness rather than primary renal disease which accounted for only 17 cases (7%). Systemic diseases led to AKI in children from either nephrotoxic medication use in their treatment or multiorgan dysfunction syndrome. In our case, APN due to presumably, a bacterial urinary infection caused AKI.

AKI has been conventionally classified into 3 categories: prerenal, intrinsic renal, and postrenal [6]. Pre-renal AKI is caused by renal hypoperfusion secondary to volume contraction or a decrease in effective blood volume. Intrinsic renal AKI includes a variety of disorders characterized by renal parenchymal damage, and obstruction of the urinary tract can cause postrenal AKI [6]. These three processes are not mutually exclusive, and any two of them or all three of them

may be present at the same time. Especially, if renal hypoperfusion and ischemia is sustained, intrinsic renal parenchymal damage may also develop. In our case, although patient presented with vomiting and oliguria, she was not hypotensive or volume depleted on admission. Radiologic and ultrasonographic studies showed no evidence of obstructive uropathy. Thus, AKI was thought to be the result of APN with an intense acute inflammatory reaction within tubules which resulted in disruption of tubular function by intense interstitial infiltrates of neutrophils and phagocytes, interstitial edema, tubular obstruction by cellular debris and glomerular hypercellularity possibly compromised intrarenal blood flow [1, 2, 4]. These tubular dysfunction and compromised intrarenal blood flow would induce renal hypoperfusion and ischemia in turn. If hypoxic/ischemic insults were sufficient to cause a decrease in medullary blood flow, then acute tubular necrosis might be developed. In other words, intrinsic renal AKI due to a bacterial infection can induce prerenal AKI and these two mechanisms might affect the progression of AKI each other. Laboratory studies also showed both prerenal and intrinsic renal AKI features. Serum blood urea nitrogen to creatinine ratio (33.3) and FE_{Na} (0.37%) suggested the presence of prerenal AKI where as urine osmolality (325 mOsm/kg) and urine sodium (39.3 mmol/L) suggested that of intrinsic renal AKI.

We did not perform renal biopsy because renal dysfunction was recovered quickly. Söylemezođlu et al [2] and Krishnamurthy et al [3] reported the cases of children presenting with AKI after APN and renal abscesses. They conducted renal biopsies because of rapidly worsening kidney function and confirmed the diagnosis. Renal biopsies showed extensive destruction of the parenchyma by the acute inflammatory process with diffuse congestion and focal hemorrhage [2] or a dense lymphoplasmacytic infiltrate and neutrophils with focal areas of necrosis in the interstitium [3].

DMSA renal scan is currently considered as the most sensitive technique for the identification of the renal parenchymal change in APN as well as in the detection of renal scarring [8]. It has been compared with histopathologic findings, and there has been 97%

agreement between them [9]. Segmental or generalized decrease of renal uptake in DMSA renal scan suggests infection of kidney including renal abscess. Rus et al [4] conducted DMSA renal scan in a case of renal abscess and AKI secondary to APN, which showed focal defects in the right kidney and smaller left kidney with multiple focal defects. Wang et al [10] has found that DMSA renal scan is the best and the most sensitive examination for detecting the extension area of renal inflammation and that it is well correlated with the renal outcome in eight cases of renal abscess in children.

We could not find renal abscesses on US, but CT showed two renal abscesses on left kidney. When the infection has not progressed to a distinct mass of sufficient size, US may just show an enlarged kidney [11]. A CT scan is more sensitive for detection of both intrarenal abscesses and focal bacterial nephritis, especially when abnormalities are small (less than 2 cm) [12]. In a series of patients assessed by Soulen et al [13], US failed to detect seven of 15 intrarenal and extrarenal abscesses revealed by CT.

Our patient had grade III/V of right VUR. VUR is a known risk factor for renal scarring and several studies demonstrated that VUR is an important risk factor for permanent renal damage after UTI [14]. Shimizu et al [15] reported a girl with APN associated with VUR that evolved into a renal abscess despite intravenous antibiotic therapy. They indicated that pre-existing malformative uropathy, especially VUR, is common with APN and renal abscess like our case.

Decision regarding the duration of antibiotic treatment and additional treatment options should be based on the patient's clinical status. Our patient was completely treated with only intravenous antibiotics and supportive treatment for three weeks. Dialysis can be considered as the management strategy of choice to rapidly restore fluid and electrolyte homeostasis because of the complexity of AKI [16]. Söylemezođlu et al [2] and Krishnamurthy et al [3] treated their patients with intravenous antibiotics and repeated sessions of peritoneal dialysis [2] and hemodialysis [3], respectively. Rus et al [4] described a child presenting with UTI, renal abscess and AKI after salmonella infection, who

was treated with antibiotics for six weeks. Sqalli et al [17] reported two cases of severe AKI complicating bacterial pyelonephritis, which were successfully treated the inflammation with corticosteroids in association with conventional antibiotics and dialysis in adults.

Clinical outcomes vary from complete recovery like our case to chronic kidney disease or death in AKI secondary to APN [2-4]. It has been thought that AKI due to hypoxic/ischemic insults were reversible with a return of renal function to normal. However, recent studies have shown that recovery may be partial and that the patient is at higher risk for later chronic kidney disease [18]. In addition, hypoxic/ischemic insults can result in physiologic and morphological alterations in the kidney that can lead to kidney disease at a later time [19]. Thus, children with a history of AKI from any cause, including APN, need life-long monitoring of their renal function.

This case report emphasizes that pediatricians should consider APN in the differential diagnosis of AKI. Prompt diagnosis and appropriate antimicrobial treatment may lead to full recovery of renal function and avoid a poor outcome on long-term dialysis. Also, such children need long-term follow-up.

한글요약

소아에서 급성 신우신염은 상대적으로 흔한 세균 감염 질환이다. 이전에 건강했던 소아에서 요로계의 폐쇄, 저혈압에 의한 신장 허혈, 신장 독성물질에의 노출 등이 없이는 급성 신우신염의 결과로 급성 신부전이 생기는 매우 드물다. 저자들은 이전에 건강했던 소아에서 방광요관 역류가 동반된 급성 신우신염과 신장 농양에 따른 급성 신부전이 발생한 예를 보고하는 바이다. 환자는 적절한 수액 치료와 항생제 치료를 통해 호전되었다. 증례를 통해 저자들은 급성 신우신염이 급성 신부전의 감별진단에 포함되어야 하며, 신장기능의 완전한 회복을 위해서는 적절한 항생제 치료가 수반되어야 함을 제시하는 바이다.

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