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CASE REPORT

A severe urinary tract infection in an 11-year-old girl with a neurogenic bladder

Jolanta Sołtysiak[1], Danuta Ostalska-Nowicka[1], Karolina Frąckowiak[1], Katarzyna Maćkowiak-Lewandowicz[1], Magdalena Patelska-Banaszewska[2], Jacek Zachwieja[1]

[1]Department of Pediatric Nephrology and Hypertension, Poznan University of Medical Sciences, Poznań, Poland
[2]Department of Pediatric Radiology, Poznan University of Medical Sciences, Poznań, Poland

ABSTRACT

This paper presents a case of urosepsis caused by *Escherichia coli* in an 11-year-old girl with a neurogenic bladder. The patient had not previously been supervised by a nephrologist or urologist. On admission, significantly elevated serum markers of inflammation (C-reactive protein 32.4 mg/dl, procalcitonin 64.2 ng/ml), kidney failure (creatinine 6.9 mg/dl, pH 7.22, HCO3 6.3 mmol/l), and increased serum urea (399 mg/dl) were detected. Antibiotic therapy with rehydration was started and improved renal function without dialysis. However, a 9-cm-long perirenal abscess site was diagnosed. It required surgical treatment, which was complicated by an anaphylactic reaction to latex. Colitis caused by Clostridium difficile was also diagnosed. Hospital treatment lasted 38 days. After 4 months the patient's glomerular filtration rate was 66 ml/min/1.73 m^2 and indicated stage 2 chronic kidney disease. Because early diagnosis of urinary tract infection is crucial for preventing complications in children with neurogenic bladder, they should undergo regular nephro-urological care.

KEY WORDS:

acute kidney injury, urinary tract infection, neurogenic bladder.

INTRODUCTION

Urinary tract infections (UTI) are common bacterial infections in children. In a pooled analysis of four studies that included children < 19 years of age (most of whom were older than two years) with urinary symptoms and/or fever, the prevalence of UTI was 7.8% [1]. The prevalence of UTI in children < 2 years of age presenting with fever is in the range 4.5–7.2% [1, 2]. Approximately 80% of cases are caused by the Gram-negative bacterium *Escherichia coli*. Other bacteria that can cause UTI include Gram-negative *Proteus*, *Enterobacter*, *Pseudomonas aeruginosa*, and *Citrobacter* and Gram-positive *Staphylococcus aureus* [3].

Risk factors for UTI include congenital anomalies of the kidney and urinary tract, especially vesicoureteral reflux (VUR), and nephrolithiasis, hypercalciuria, constipation, sexual activity, and bladder dysfunction [4, 5]. Neurogenic bladder involves severe bladder dysfunction resulting from central nervous system defects, such as those caused by a myelomeningocele [6]. In patients with neurogenic bladder, the overall incidence of UTI is high at an estimated 2.5 episodes per patient per year [7].

Urinary tract infections in children with neurogenic bladder are primarily caused by insufficient bladder emptying resulting from detrusor dysfunction (areflexia or hyporeflexia) accompanied by detrusor-sphincter dyssynergia. The absence of contractile activity of the detrusor combined with the closed bladder sphincter significantly impairs urine outflow. Increased intravesical pressure may predispose individuals to VUR, which significantly increases the risk of developing UTI [8]. Furthermore,

ADDRESS FOR CORRESPONDENCE:

Jolanta Sołtysiak, PhD, Department of Pediatric Nephrology and Hypertension, Poznan University of Medical Sciences, Poznań, Poland, e-mail: jsoltysiak1@gmail.com

increased intravesical pressure and excessive bladder distension increase the risk of bladder wall hypoperfusion and ischemia, which can lead to reduced bladder penetration by inflammatory cells and antibiotics [8]. The local defence mechanism, which involves the glycosaminoglycan layer and surface secretory immunoglobulin A, can also become damaged. Due to reduced sensation in the pelvic region, patients rarely report pain due to UTI, which significantly delays the diagnosis.

This report presents a severe UTI in an 11-year-old girl with a neurogenic bladder who developed life-threatening complications.

CASE REPORT

An 11-year-old girl with a congenital myelomeningocele and a ventriculoperitoneal shunt was referred from a regional hospital to the Department of Paediatric Nephrology and Hypertension, Poznań, due to renal failure. Approximately one week before admission, the patient experienced diarrhoea, loss of appetite, and a fever of 39°C. Prior to presentation, the patient had only been managed under neurosurgery care, without nephro-urological supervision. On admission to the hospital, anuria was observed. The patient was in a severe general condition; she had signs of dehydration, tachycardia, and tachypnoea. Laboratory tests revealed anaemia, thrombocytopaenia, significantly increased inflammatory markers and indicators of impaired renal failure (elevated serum creatinine and severe metabolic acidosis). Following bladder catheterisation, 1100 ml of bloody urine was obtained. Urine analysis confirmed erythrocyturia, leukocyturia, and proteinuria (Table 1). Ultrasound revealed enlarged kidneys with increased echogenicity of the cortex and enhanced corticomedullary differentiation. The right kidney was 11.2 cm long, with a dilated pelvicalyceal system; the pelvis dimensions were 2.3 × 2.7 × 0.7 cm and the calyces were up to 1.1 cm wide. The left kidney was 14.1 cm long, with a dilated pelvicalyceal system; the pelvis dimensions were 5.9 × 2.4 × 1.3 cm and calyces were up to 1.7 cm wide with blunted fornices, and a thickened pelvic wall up to 0.45 cm.

The initial diagnosis was urosepsis with acute kidney injury (AKI) in a child with probable chronic kidney disease (CKD) due to a neurogenic bladder. The treatment included cefotaxime, rehydration of the patient, and diuretics to enhance urinary output. Urinary culture revealed the presence of *Escherichia coli* sensitive to several antibiotics, including cefotaxime. On the sixth day of hospitalisation, inflammatory and renal failure indicators had decreased (Table 1); however, C-reactive protein (CRP) increased on the ninth day. A repeated urine culture showed no bacterial growth, while abdominal ultrasound revealed a hypoechoic, heterogeneous area with no detectable vascular flow in the left kidney next to the pelvis and upper part of the abdominal ureter. The total cranio-caudal length of this change measured 9.1 cm, and detailed diameters were 5.4 × 4.5 × 5.2 cm in the part adjacent to the renal pelvis and 2.9 × 2.8 × 1.7 cm in the part contiguous to the ureter.

A perinephric abscess was suspected and was confirmed with magnetic resonance imaging (MRI) (Figure 1). The abscess compressed the pelvis but did not demonstrate communication with the collecting system. The upper spherical portion of the abscess measured 4.7 × 3.7 × 3.8 cm. A narrow, 6-mm-wide channel opened into the lower part, located medially to the ureter, with dimensions of 2.1 × 3.7 × 1.9 cm. In the parenchyma of the upper pole of the left kidney, numerous small cystic changes 0.2–0.7 cm in diameter were observed in addition to an inflammatory reaction within the iliopsoas muscle.

Due to difficult access to the abscess, percutaneous drainage was not possible, and surgical treatment was necessary. The operation was complicated with anaphylactic shock that occurred during placement of the silicone tube near the abscess. As at the same time meropenem was administered, an allergic reaction to this antibiotic was also suspected. The operation was aborted, and the patient was transferred to the intensive care unit for two days. Antibiotic treatment was changed to piperacillin/tazobactam. The surgery resumed the next day following stabilisation of the patient. After the left perirenal space was opened under ultrasound guidance, a structure corresponding to an abscess was accessed. The lesion was punctured and incised, and a non-latex drain was inserted.

Escherichia coli was grown in a culture of the abscess contents. Piperacillin/tazobactam treatment was continued. Two days after surgery, the CRP concentration increased from 2.0 to 30.9 mg/dl. The patient experienced abdominal pain and bloody diarrhoea. *Clostridium difficile* glutamate dehydrogenase antigens were detected in the stool; however, *Clostridium difficile* toxin was negative. Oral vancomycin was added to the treatment regimen, and over several days, a gradual reduction in CRP was observed.

Sonographic examination revealed a partially visible empty abscess with dimensions of 5.5 × 2.0 × 1.9 cm, situated close to the medial edge of the lower part of the left kidney.

Despite the residual volume observed during the sonographic examination, the drain was removed. Antibiotic treatment was continued, and serum CRP gradually decreased to 4.6 mg/dl. The patient was discharged on day 38 of hospitalization. Intermittent catheterisation using latex-free Nelaton catheters was recommended.

Bladder treatment with oxybutynin and UTI prophylaxis with trimethoprim-sulfamethoxazole was initiated. Voiding cystourethrography did not indicate VUR. Dynamic kidney scintigraphy using Tc-99m mercaptoacetyltriglycine was performed 4 months after the AKI. The study revealed blocked urine outflow from the collecting systems of both kidneys (no reaction to furosemide administration) and scintigraphic features of bilateral parenchymal

TABLE 1. Laboratory test results

Day of treatment	0	6	9	16	18	22	24	38 (hospital discharge)	Control (1 month after discharge)	Control (4 months after discharge)
Hemoglobin [g/dl] (N: 12–16)	9.5	8.2	11.6	12.1	8.7	13.8	10.0	11.1	10.3	14.0
WBC [g/l] (N: 4–10)	9.1	11.7	12.1	18.4	12.2	31.1	16.1	9.5	9.0	8.1
PLT [g/l] (N: 150–400)	113	199	246	562	531	793	482	572	312	546
CRP [mg/dl] (N: < 0.5)	32.4	10.7	24.9	8.2	23.6	2.1	35.3	3.6	0.7	0.4
PCT [ng/ml] (N: 0.17–0.35)	64.2	4.5	5.2	0.8	2.3	0.4	1.2	0.3	0.2	0.1
Creatinine [mg/dl] (N: 0.24–0.73)	6.9	3.3	3.3	1.7	0.9	1.0	1.1	1.2	1.0	0.9
Urea [mg/dl] (N: 11–38)	399	165	169	119	91	87	52	51	30	49
GFR [ml/min/1.73 m^2] (N > 90)	8.6	17.8	17.5	34.8	62.0	56.6	55.5	48.0	58.8	66.0
Na [mmol/l] (N: 132–145)	136	136	136	137	148	136	138	134	138	142
K [mmol/l] (N: 3.1–5.1]	5.67	3.34	4.00	4.16	3.60	2.71	2.89	4.06	4.47	4.10
pH (N: 7.32–7.42)	7.22	7.49	7.44	7.34	7.48	7.52	7.46	7.36	7.31	7.37
HC03 [mmol/l] (N: 24–28)	6.3	24.6	21.4	18.7	27.5	25.7	24.4	18.8	18.4	23.5
BE [mmol/l] (N: –3 to +3)	–20.3	–1.9	–1.7	–5.6	4	2.9	0.9	–5.7	–2.7	–1.2
ALT [IU/l] (N: < 39)	7	7	–	6	6	–	–	–	23	26
AST [IU/l] (N: < 47)	11	12	–	18	25	–	–	–	20	29
Cyst [mg/l] (N: 0.53–1.01)	3.53	–	–	3.12	–	–	–	–	–	1.76
Fibrosis [mg/dl] (N: 180–350)	935	545	–	750	574	–	–	–	355	473
D-dimer [mg/dl] (N: < 0.55)	11.15	4.26	6.61	10.59	2.32	1.67	–	0.59	–	0.58
Proteinuria [mg/dl] (N: 0)	320	25	–	–	–	0	–	–	0	0
Leukocyturia (HPF) (N: 0–5)	5070	10–15	–	–	–	2–4	–	–	8–10	4–8
Erythrocyturia (HPF) (N: 0–5)	40–60	20–30	–	–	–	15–20	–	–	20–30	1–2

ALT – alanine aminotransferase, AST – aspartate aminotransferase, BE – base excess, CRP – C-reactive protein, GFR – glomerular filtration rate, HPF – per average high power field, N – normal range, PCT – procalcitonin, PLT – platelet, WBC – white blood cells

lesions. The proportional participation of kidneys in tracer extraction was within the normal range.

Four months after the acute episode, serum creatinine concentration was 0.9 mg/dl (estimated glomerular filtration rate according to Schwartz formula = 66 ml/min/1.73 m^2; stage 2 CKD). The patient was referred to a urologist for further treatment.

DISCUSSION

Urinary tract infection can lead to serious, life-threatening complications, including AKI. According to the Kidney Disease: Improving Global Outcomes AKI definition, AKI is suspected when blood creatinine increases by more than 0.3 mg/dl in 48 hours or 1.5 times

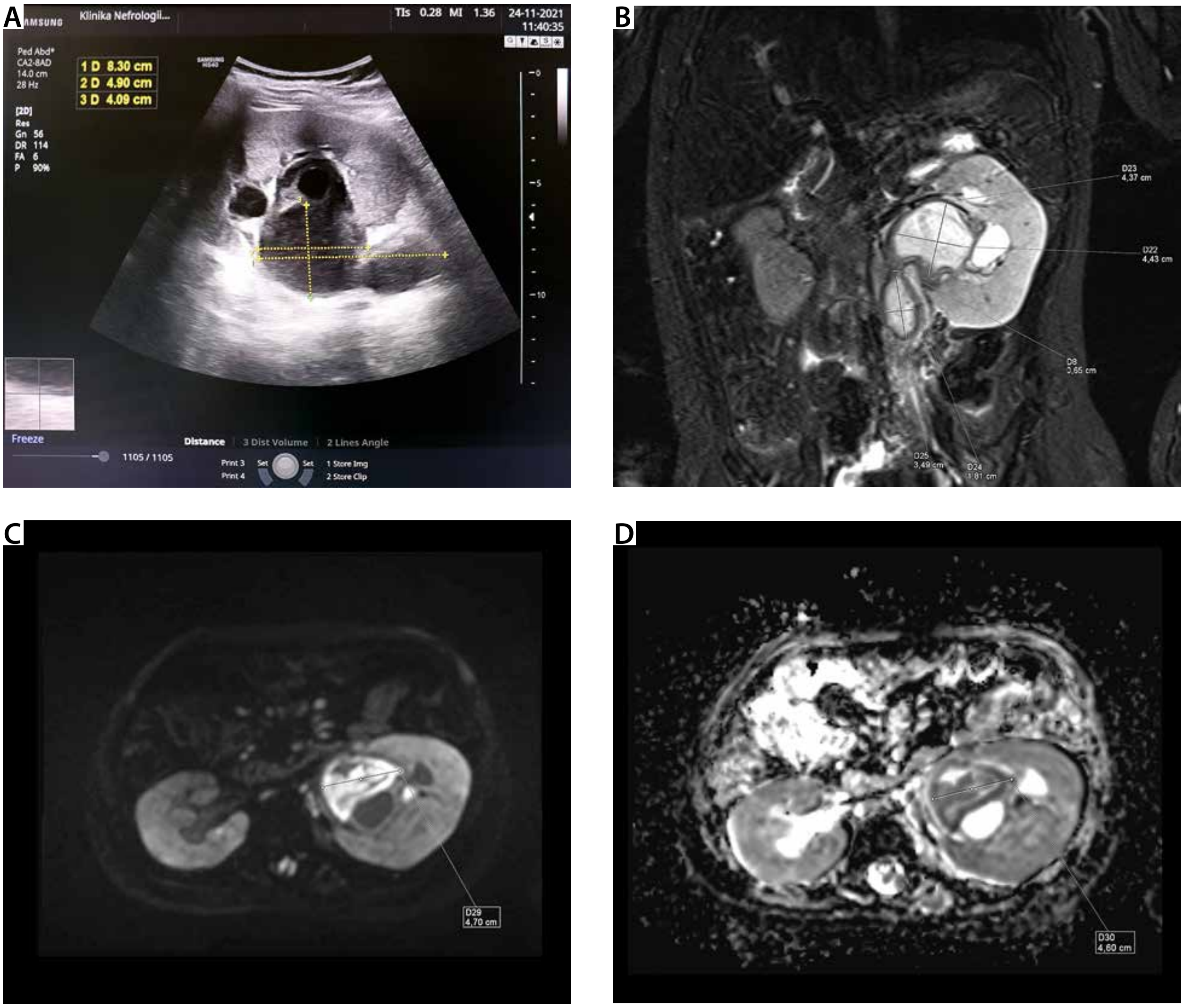

FIGURE 1. Ultrasound and magnetic resonance imaging (MRI) of the perirenal abscess. **A**) The ultrasound image shows a well-defined, hourglass-shaped, hypoechoic focus with a limited area of concentrated fluid. **B**) MRI examination with T2-weighted images in the coronal view allowed more accurate visualisation of the hourglass-shaped focus, which was localised in the hilum, near the pelvis of the left kidney and close to the lower pole of the kidney, with a high signal that indicated a fluid nature. **C**) examination in the axial view showing a high diffusion-weighted imaging (DWI) signal. **D**) MRI examination in the axial view showing a low apparent diffusion coefficient (ADC) map signal. DWI and ADC maps indicate restriction of water diffusion, which implies the presence of an abscess

the baseline value from the previous 7 days or when diuresis decreases below 0.5 ml/kg/h within 6 hours [9].

Baseline blood creatinine levels were unknown in the present case. Research findings on admission might suggest that the patient was in end-stage renal failure; however, rapid improvement in renal function following antibiotic therapy and rehydration confirmed the diagnosis of AKI.

The frequency of AKI during UTI is difficult to assess due to the low number of reported cases, especially in children. In a study involving 790 adult patients hospitalised for UTI, 12.3% had concomitant AKI [10]. The mean baseline glomerular filtration rate (GFR) was 53 ±23 ml/min/1.73 m^2, and most patients (74.2%) were female. Among the AKI patients, septic shock occurred in 22.7%, bacteraemia in 39.2%, diabetes in 56.7%, and a fever-free UTI course in 59.8%.

Several cases of AKI associated with renal biopsy changes during UTI have been described [11, 12]. In one study, two patients were HIV positive [12]. In a 3-year-old boy with acute pyelonephritis and AKI, kidney biopsy revealed acute interstitial infiltration of neutrophils and macrophages. There were also glomerulitis and capillary tuft thrombosis [13]. In some cases, temporary dialysis was necessary [13, 14].

Most studies have emphasised the role of hypovolemia, hypotension, sepsis, nephrotoxic drugs, contrast agents, CKD, urinary tract obstruction, urolithiasis, and chronic bladder catheterisation in the development of AKI during UTI [5, 10–12].

In the present case, several risk factors for AKI development during UTI were present. Chronic kidney disease, bladder dysfunction, reduced pelvic sensation, and the absence of dysuria could have delayed the diagnosis of UTI and were significant risk factors for AKI and sepsis.

Sepsis is defined as a systemic inflammatory response syndrome (SIRS) in response to a suspected or confirmed infection [15]. The present case demonstrated SIRS associated with UTI; therefore, urosepsis was initially diagnosed. Sepsis is one of the most common causes of AKI [16]; approximately 60% of septic shock patients develop AKI [17]. Despite the present case being at high risk of developing septic shock, no relevant signs were observed. Therefore, the AKI was likely caused by a severe UTI.

A severe course of UTI was further confirmed by the presence of intrarenal and extrarenal abscesses. In pyelonephritis, local activation of the complement system results in vasoconstriction and inflammatory oedema, which may lead to kidney tissue necrosis and the development of a pus-filled cavity [18, 19]. Intrarenal abscesses are associated with lobar necrosis, while perirenal abscesses are associated with adipose tissue necrosis. Kidney abscesses form cavities surrounded by walls, while perirenal abscesses involve more diffuse structures between the kidney capsule and Gerota's fascia. In the present case, the perirenal abscess had two parts: a wider, spherical part located close to the kidney and a tail-shaped part formed due to the force of gravity.

Computed tomography (CT) is considered the gold standard imaging method for the diagnosis of renal and perirenal abscesses [20, 21]. In the present case, the use of ionic contrast was contraindicated due to kidney failure. In patients with decreased GFR, MRI with a gadolinium-based contrast medium is preferred. However, in the case of GFR < 30 ml/min/1.73 m^2 or AKI, the use of gadolinium is associated with a risk of nephrogenic systemic fibrosis (NSF) and nephrotoxicity [22]. Moreover, MRI has shown limited relevance in the diagnosis of perirenal abscesses. However, in a study of 170 adults with acute pyelonephritis, 21 out of 26 patients assessed by MRI presented accurate positive results. Furthermore, in one patient, an abscess that was not visualised on the CT scan was detected by MRI [23].

In the present case, due to kidney failure, we performed MRI without contrast media. Magnetic resonance imaging revealed necrosis in the lesion previously visualized in ultrasonography, with a low signal on the T1-weighted images and a high signal on the T2-weighted images. Diffusion-weighted images are helpful during MRI examinations. Diffusion-weighted images of abscesses demonstrate water diffusion restriction as an increased signal in the lesion and low signal maps of the apparent diffusion coefficient. Using this method in the present case helped us to visualise the perirenal abscess and its precise location (Figure 1).

Antibiotic therapy is primarily used for the treatment of small abscesses (< 5 cm); larger changes require percutaneous drainage or surgical excision [24, 25]. Compared with percutaneous drainage, surgical drainage is associated with a higher risk of complications. In the present case, due to the location of the abscess, the only possible method of treatment was surgical excision. The operation was successful. Anaphylactic shock might be the consequence of chronic latex stimulation of the ventriculoperitoneal shunt, while *Clostridium difficile* infection resulted from intensive antibiotic therapy.

The complications associated with AKI during UTI can have numerous long-term effects, such as cortical scars and permanent renal parenchymal damage. This may result in a decrease in the filtration function of the kidney and progression to end stage kidney failure requiring dialysis. Furthermore, there is an increased risk of hypertension. Both AKI and UTI can lead to premature death.

CONCLUSIONS

In the present case, a significant improvement in GFR was achieved, and CKD was finally diagnosed as stage 2; however, the severe UTI with AKI might have intensified the pre-existing renal damage. This report proves that patients with neurogenic bladder are at a high risk of developing severe UTI with life-threatening complications. Therefore, early diagnosis and proper management with nephro-urological supervision are crucial for the prevention of complications in children with neurogenic bladder.

DISCLOSURE

The authors declare no conflict of interest.

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