CASE REPORT

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

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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 m² 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,
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, Poznan, 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, thrombocytopenia, and decreased 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 demonstrated Escherichia coli sensitive to sev- triglycine was performed 4 months after the AKI. The study confirmed close to the medial edge of the lower part 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.

Two days after surgery, the CRP concentration increased to 38 of hospitalization. Intermittent catheterisation using latex-free Nelaton catheters was recommended. 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. Anti-biotic 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-sulphamethoxazole was initiated. Voiding cystourethrography did not indicate VUR. Dynamic kidney scintigraphy using Tc-99m mercaptoacetyl triglycine 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
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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 m²; 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.

**TABLE 1. Laboratory test results**

<table>
<thead>
<tr>
<th>Day of treatment</th>
<th>0</th>
<th>6</th>
<th>9</th>
<th>16</th>
<th>18</th>
<th>22</th>
<th>24</th>
<th>38 (hospital discharge)</th>
<th>Control (1 month after discharge)</th>
<th>Control (4 months after discharge)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin [g/dl] (N: 12–16)</td>
<td>9.5</td>
<td>8.2</td>
<td>11.6</td>
<td>12.1</td>
<td>8.7</td>
<td>13.8</td>
<td>10.0</td>
<td>11.1</td>
<td>10.3</td>
<td>14.0</td>
</tr>
<tr>
<td>WBC [g/l] (N: 4–10)</td>
<td>9.1</td>
<td>11.7</td>
<td>12.1</td>
<td>18.4</td>
<td>12.2</td>
<td>31.1</td>
<td>16.1</td>
<td>9.5</td>
<td>9.0</td>
<td>8.1</td>
</tr>
<tr>
<td>PLT [g/l] (N: 150–400)</td>
<td>113</td>
<td>199</td>
<td>246</td>
<td>562</td>
<td>531</td>
<td>793</td>
<td>482</td>
<td>572</td>
<td>312</td>
<td>546</td>
</tr>
<tr>
<td>CRP [mg/dl] (N: &lt; 0.5)</td>
<td>32.4</td>
<td>10.7</td>
<td>24.9</td>
<td>8.2</td>
<td>23.6</td>
<td>2.1</td>
<td>35.3</td>
<td>3.6</td>
<td>0.7</td>
<td>0.4</td>
</tr>
<tr>
<td>PCT [ng/ml] (N: 0.17–0.35)</td>
<td>64.2</td>
<td>4.5</td>
<td>5.2</td>
<td>0.8</td>
<td>2.3</td>
<td>0.4</td>
<td>1.2</td>
<td>0.3</td>
<td>0.2</td>
<td>0.1</td>
</tr>
<tr>
<td>Creatinine [mg/dl] (N: 0.24–0.73)</td>
<td>6.9</td>
<td>3.3</td>
<td>3.3</td>
<td>1.7</td>
<td>0.9</td>
<td>1.0</td>
<td>1.1</td>
<td>1.2</td>
<td>1.0</td>
<td>0.9</td>
</tr>
<tr>
<td>Urea [mg/dl] (N: 11–38)</td>
<td>399</td>
<td>165</td>
<td>169</td>
<td>119</td>
<td>91</td>
<td>87</td>
<td>52</td>
<td>51</td>
<td>30</td>
<td>49</td>
</tr>
<tr>
<td>GFR [ml/min/1.73 m²] (N &gt; 90)</td>
<td>8.6</td>
<td>17.8</td>
<td>17.5</td>
<td>34.8</td>
<td>62.0</td>
<td>56.6</td>
<td>55.5</td>
<td>48.0</td>
<td>58.8</td>
<td>66.0</td>
</tr>
<tr>
<td>K [mmol/l] (N: 3.1–5.1)</td>
<td>5.67</td>
<td>3.34</td>
<td>4.00</td>
<td>4.16</td>
<td>3.60</td>
<td>2.71</td>
<td>2.89</td>
<td>4.06</td>
<td>4.47</td>
<td>4.10</td>
</tr>
<tr>
<td>pH (N: 7.32–7.42)</td>
<td>7.22</td>
<td>7.49</td>
<td>7.44</td>
<td>7.34</td>
<td>7.48</td>
<td>7.52</td>
<td>7.46</td>
<td>7.36</td>
<td>7.31</td>
<td>7.37</td>
</tr>
<tr>
<td>HCO3 [mmol/l] (N: 24–28)</td>
<td>6.3</td>
<td>24.6</td>
<td>21.4</td>
<td>18.7</td>
<td>27.5</td>
<td>25.7</td>
<td>24.4</td>
<td>18.8</td>
<td>18.4</td>
<td>23.5</td>
</tr>
<tr>
<td>BE [mmol/l] (N: –3 to +3)</td>
<td>–20.3</td>
<td>–1.9</td>
<td>–1.7</td>
<td>–5.6</td>
<td>4</td>
<td>2.9</td>
<td>0.9</td>
<td>–5.7</td>
<td>–2.7</td>
<td>–1.2</td>
</tr>
<tr>
<td>ALT [IU/l] (N: &lt; 39)</td>
<td>7</td>
<td>7</td>
<td>–</td>
<td>6</td>
<td>6</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>23</td>
<td>26</td>
</tr>
<tr>
<td>AST [IU/l] (N: &lt; 47)</td>
<td>11</td>
<td>12</td>
<td>–</td>
<td>18</td>
<td>25</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>20</td>
<td>29</td>
</tr>
<tr>
<td>Cyst [mg/l] (N: 0.53–1.01)</td>
<td>3.53</td>
<td>–</td>
<td>–</td>
<td>3.12</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>1.76</td>
</tr>
<tr>
<td>Fibrosis [mg/dl] (N: 180–350)</td>
<td>935</td>
<td>545</td>
<td>–</td>
<td>750</td>
<td>574</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>355</td>
<td>473</td>
</tr>
<tr>
<td>Proteinuria [mg/dl] (N: 0)</td>
<td>320</td>
<td>25</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>0</td>
<td>–</td>
<td>–</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Leukocyturia (HPF) (N: 0–5)</td>
<td>5070</td>
<td>10–15</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>2–4</td>
<td>–</td>
<td>–</td>
<td>8–10</td>
<td>4–8</td>
</tr>
<tr>
<td>Erythrocyturia (HPF) (N: 0–5)</td>
<td>40–60</td>
<td>20–30</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>15–20</td>
<td>–</td>
<td>–</td>
<td>20–30</td>
<td>1–2</td>
</tr>
</tbody>
</table>

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 m², 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 ab-
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 patients with decreased GFR, MRI with a gadolinium-based contrast medium was contraindicated due to kidney failure. In the present case, the use of ionic contrast was contraindicated due to kidney failure. In the present case, the use of ionic contrast was contraindicated due to kidney failure. In the present case, the use of ionic contrast was contraindicated due to kidney failure. In the present case, the use of ionic contrast was contraindicated due to kidney failure. In the present case, the use of ionic contrast was contraindicated due to kidney failure. In the present case, the use of ionic contrast was contraindicated due to kidney failure. 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In the present case, 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.

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.

REFERENCES