Case Report

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# A rare complication of *Escherichia coli* induced urosepsis; is Guillain-Barre syndrome

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## ABSTRACT

Guillain Barre syndrome (GBS) is the most common neurological cause of acute flaccid paralysis worldwide. Early diagnosis and treatment of GBS are vital due to possible deadly consequences. Awareness of the silent neurological symptoms in patients preparing for upcoming surgery may have critically crucial for a urologist. Developing GBS after relieving urosepsis is rarely addressed in the literature. Therefore, this report presents an infrequent complication of *Escherichia coli* (*E. coli*)-induced urosepsis, GBS. A 47-year-old female patient was admitted to the emergency department in a septic state. During the intensive care unit management with the preliminary diagnosis of sepsis-related hypovolemic shock, a nephrostomy catheter was placed in an obstructed left kidney due to impacted upper ureteral calculus with 1.5 cm in size. Following the improvement of the patient's condition in intensive care unit with proper management, the patient was transferred to the urology ward for definitive treatment. During the follow-up, however, the patient showed some neurological signs and symptoms considering GBS. An obtained cerebral spinal fluid analysis revealed an albumin-cytologic dissociation and examining the patient underpinned the diagnosis. The patient was treated with intravenous immunoglobulin for five days, according to the guidlines. After the treatment, the patient's condition improved rapidly following two weeks. The left obstructed ureteral stones were removed with ureteroscopy. A stone-free status was achieved the following month. GBS is the most common cause of acute flaccid paralysis worldwide, and proper management is essential due to poor prognosis. GBS after a uroseptic condition is sporadic, but any surgery on patients who experience active GBS would bode for severe consequences, so awareness of the silent neurological symptoms in patients prepared for upcoming surgery is vital for a urologist. We aimed to remind with this report of the possibility of GBS for a patient who expresses neurological symptoms following a septic state.

**Keywords:** Guillain Barre syndrome, urosepsis, ureteral stones, hydronephrosis

## INTRODUCTION

Early diagnosis and proper management of urosepsis are vital. About one out of four adult sepsis cases worldwide are caused by urosepsis, and septic shock related to urosepsis is responsible for 20-30 % of mortality (1). From the urologist's aspect, with limited data available to evaluate the urosceptic patients' surgical outcomes, the rate of an underlying uro-surgical condition is 12-37% (1-3) In most cases, the main culprits are hydronephrosis/ pyonephrosis, benign prostatic hyperplasia, and obstructing ureteral/ renal stones (2-3). After the initial intervention with a nephrostomy tube or double j (D-J) stent placement and proper management of the condition in the Intensive care unit (ICU), these patients generally return to the urologic ward for definitive treatment after a while. However, a urologist might run into some unusual late downsides of sepsis that cause prolonged hospital stays and deter planned therapy. Therefore, this report presents an infrequent complication of *Escherichia coli* (*E. coli*)-induced urosepsis, Guillan Barre syndrome (GBS). According to our research, only two case reports (7,8) in the literature show GBS after *E. coli* infection. So, we also aimed to support the literature with this report.

## CASE REPORT

A 47-year-old female patient was admitted to the emergency department in a confused state for an hour. Her initial examination revealed low blood pressure (90/50 mm/hg), increased heartbeat (114 bpm), and a low temperature (35.4°C), in addition to low $O_2$ saturation (82 mm Hg). Her husband reported that she had complained of left flank pain, dysuria, vomiting, and hematuria for the previous two days. She also has

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a history of bilateral renal stone surgery. An abdominal computed tomography scan showed an obstructed left upper ureteral calculus 1.5 cm in size associated with mild hydronephrosis in the left kidney and atrophic signs in the right kidney. The laboratory results showed deranged kidney functions with raised creatinine levels to 2.2, left-shifted FBC, mildly elevated liver functions, and metabolic acidosis signs in blood gas analysis, as well as leucocytosis in the urine sample (**Table 1**). After the initial management, the patient was transferred to ICU for the preliminary diagnosis of a sepsis-related hypovolemic shock. Therefore, the patient has resuscitated accordingly with intravenous fluids, $O_2$, and empiric antibiotic therapy. A nephrostomy catheter placement was carried out after hemodynamic stability had been ensured. According to urine and blood culture analyses, demonstrating that extended-spectrum beta-lactamase (ESBL) positive *E. coli* was the causing organism, the treatment was changed to meropenem 1 mg gr daily and gentamicin 160 mg daily for ten days.

After the patient's septic state had improved, cultures cleared up, and the vital signs returned to normal, she was transferred to the urologic ward for definitive renal stone surgery. However, the patient showed some steadily increased neurological signs and symptoms during the observation, including hypoesthesia in both hands and feet, difficulty walking, and swallowing problems. The neurological examination showed symmetrical weakness in her lower extremities, rapidly spreading to her upper extremities over the following days. Muscle strength testing revealed 2/5 strength in the arms and 2/5 in the legs. Generalized hyporeflexia was present. Mental status and cranial nerves II-XII, however, were intact. The result of the imaging study with magnetic resonance imaging of the cervical, thoracic, and lumbar spine was unremarkable.

Serum electrolytes, including calcium, magnesium, and sodium levels, were also normal. The suspicion of acute polyneuropathy related to Guillan barre syndrome leads to obtaining Cerebrospinal fluid (CSF). The CSF testing revealed albumin-cytologic dissociation with an elevated protein level (863 mg/dl) but an average glucose level (52 mg/dl). Cell counts showed no leukocyte/ $mm^2$ and e few dysmorphic erythrocytes. No organisms were cultured from CSF. Therefore, the diagnosis of Guillan barre syndrome was strongly suspected, and the patient was treated with a five-day course of intravenous immunoglobulin (IVIG). The patient's condition has dramatically improved following the treatment, and she regains muscle strength bilaterally over the seven days, confirming the diagnosis. Electromyography (EMG) study could not be conducted at that time due to technical problems. After two weeks, the patient recovered with oral antibiotics and physical therapy, and her muscle weakness and dyspnea gradually improved over one month.

Consequently, at her follow-up, three months after discharge, the patient's neurologıcal symptoms ultimately enhanced and did not recur. Regarding stone status, the patient's left obstructed ureteral stones were removed with ureteroscopic surgery first and followed for 15 days with a bilateral D-J stent. Finally, the stone-free state was achieved with bilateral retrograde intrarenal surgery.

**Table 1.** CSF, Urine and blood analyses

| Parameters | Causative organisms | Antibiotic résistance + | Antibiotic susceptible + |
|---|---|---|---|
| Urine culture results | *Escherichia coli* ESBL + 50.000 CFU/mL | Cefazolin > 32<br>Ciprofloxacin >1<br>Trimethoprim/Sulplamethoxazole > 8/152<br>Cefixime >4<br>Ampicilin >16<br>Ceftazidime 8<br>Ceftriaxone >4<br>Levofloxacin >4<br>Nitrofrontain 128 | Piperacilin/tazobactam 4/4<br>Meropenem <0.13<br>Gentamicin <2<br>Fosfomycin <16<br>Imıpenem 0.25 |
| Blood Culture results | *Escherichia coli* ESBL + | Cefazolin > 32<br>Cefepime 8<br>Ciprofloxacin >1<br>Trimethoprim/Sulplamethoxazole > 8/152<br>Ampicilin >16<br>Ceftazidime 8<br>Ceftriaxone >4<br>Levofloxacin >4<br>Cefuroxime Sodium >16 | Piperacilin/tazobactam 4/4<br>Meropenem <0.13<br>Gentamicin <2<br>Fosfomycin <16<br>Imıpenem 0.25<br>Amikasin <8<br>Amoxicilin/Clavulanate 8/2<br>Ampicilin/ sulbactam 2/8 |
| CSF analyses | **No** | - | - |
| | **levels of some parameters in CSF** | | |
| LDH | 12 U/L | | |
| Glucose | 52 mg/dl | | |
| Protein | **863 mg/L** ( 150-450 Normal ranges) | | |
| Sodium | 143 mmol/L | | |
| Chloric | 121 | | |

CSF: cerebral spinal fluid, ESBL: extended spectrum beta lactamases, LDH: Lactate dehydrogenase

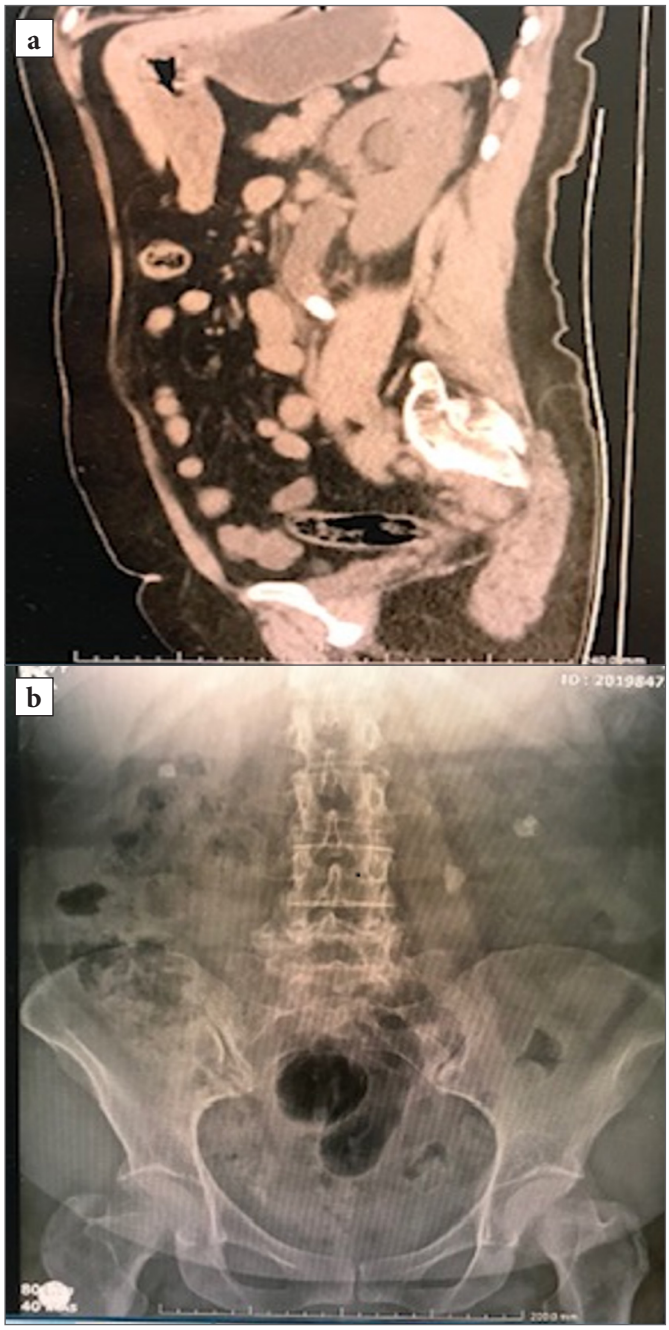


**Figure 1.** Stone status at first presentation. A left obstructed ureteral stone and bilateral renal stones is seen. **a)** Preoperative CT scan. **b)** Preoperative KUB film.

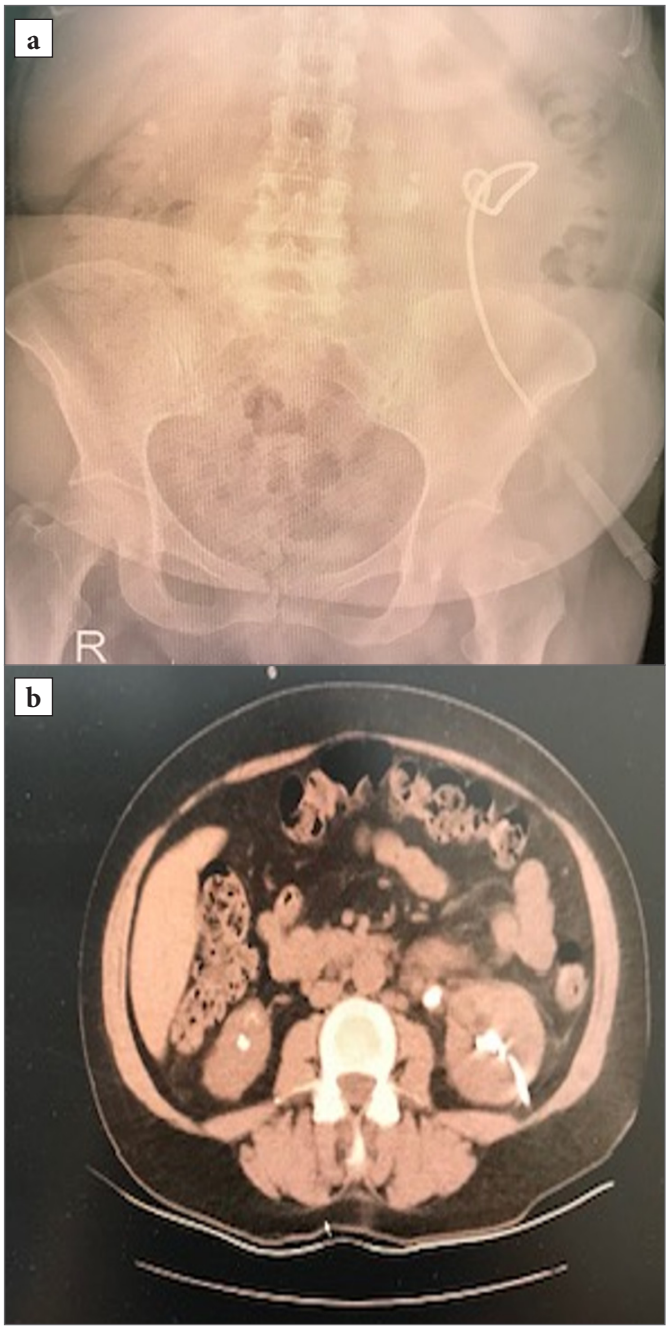


**Figure 2.** A nephrostomy tube was placed in the left kidney for decompression. **a)** Nephrostomy Tube on KUB, **b)** Nephrostomy Tube on CT scan

## DISCUSSION

GBS is the most common cause of acute flaccid paralysis worldwide (4). This autoimmune disease is characterized by rapidly progressive and acute inflammatory features, which lead to polyradiculoneuropathy. The condition requires prompt diagnosis and treatment; otherwise, the patient's state may deteriorate quickly, and respiratory failure may develop. It is estimated that 3–10% of patients with GBS experience autonomic nervous system involvement, which may cause mortality (5). Diagnosis of GBS is based on the patient history and neurological, cerebrospinal fluid, and electrophysiological examinations. Although the exact cause of the disease is unknown, about 75 percent of patients experience preceding infections. The majority of reasons for conditions include *Campylobacter jejuni*, cytomegalovirus, hepatitis E virus, *Mycoplasma pneumonia*, Epstein–Barr, and Zika virus. Other rare infectious sources have also been reported in the literature, such as Human Immunodeficiency Virus (HIV), Haemophilus influenza, herpes simplex, rubella, and varicella-zoster (6). It has been suggested that an aberrant immune response induced by these infections may cause GBS (7). Molecular imitation is a mechanism by which infectious agents may cause an immune response against autoantigens of gangliosides.

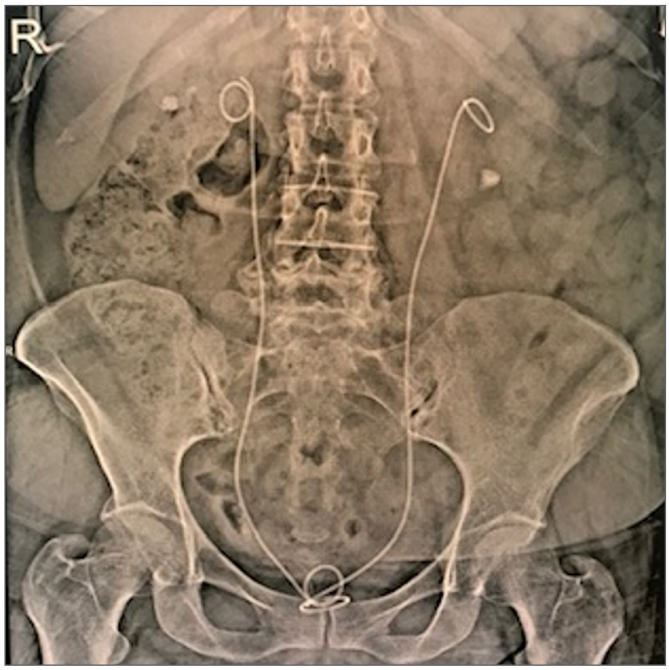

**Figure 3:** The left ureteral stone was removed and bilateral D-J stents were placed.

Anti-Gangliosidoz molecule 1 (GM1) IgG antibody is positive in about 30% GBS after *C. jejuni* infection (10). Because the *E. coli* capsule incorporates LPS, like all gram-negative bacilli, the most probable mechanism for developing GBS after *E. coli* infection is the same immune response as the *C. jejuni* infection. However, there is a lack of available data about the homology between *E. coli* lipopolysaccharide (LPS) and the GM1 ganglioside. More resources need to be done on this issue.

A surgery on patients who experience active GBS would bode for severe consequences, so awareness of the silent neurological symptoms in patients prepared for upcoming surgery is vital for a urologist. From that point of view, we aimed to remind the possibility of GBS for a patient who expresses neurological symptoms following a septic state.

Urosepsis is a well-known complication of GBS, but it is hard to say the reverse. Only two case reports in the literature show GBS after *E. coli* infection. One of them has been associated with *E. coli* related urinary tract infections (8) and the other one is related to a perirenal abscess formation which has not been required a surgical intervention (9). These reports are related to non-surgical causes, both of which were published in neurological journals. Our case is a unique report presenting GBS in a patient with an uro-surgical condition.

## CONCLUSION

Urosepsis is a well-known complication of GBS, but it is hard to say the reverse. Only two case reports in the literature show GBS after E. coli infection. One of them has been associated with E. coli related urinary tract infections (8) and the other one is related to a perirenal abscess formation which has not been required a surgical intervention (9).

## ETHICAL DECLARATIONS

**Informed Consent:** Written informed consent was obtained from all participants who participated in this study.

**Referee Evaluation Process:** Externally peer-reviewed.

**Conflict of Interest Statement:** The authors have no conflicts of interest to declare.

**Financial Disclosure:** The authors declared that this study has received no financial support.

**Author Contributions**: All of the authors declare that they have all participated in the design, execution, and analysis of the paper and that they have approved the final version

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