



A Rare Pediatric Case of *Morganella morganii* Urinary Tract Infection with Hydronephrosis and Antimicrobial Resistance Challenges: A Case Report

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ABSTRACT

Morganella morganii (*M. morganii*) is a facultative anaerobic, gram-negative bacillus which uncommonly causes urinary tract infections in children. We report a rare pediatric case of *M. morganii*, highlighting the diagnostic and treatment challenges associated with this microorganism, its notable antimicrobial resistance profile, and the importance of antibiotic stewardship in guiding effective therapy.

Keywords: *Morganella morganii*, pediatric urinary tract infection, hydronephrosis, gram-negative bacilli, case report

Introduction

Morganella morganii (*M. morganii*) is a facultative anaerobic, gram-negative bacillus, non-lactose-fermenting, and urease-positive microbe. It belongs to the *Enterobacteriaceae* family, which is normally found in the gastrointestinal flora and is typically considered as an opportunistic pathogen (1). Although *M. morganii* has been more commonly associated with nosocomial infections in adults (2,3), community-acquired cases have also been reported (1,4).

There is limited data reporting on *M. morganii* infections, but studies across different countries have reflected the rarity of this microorganism in the general population and its exceptionally low incidence among pediatric patients. According to a study conducted in Australia from 2000 to

2019, only 709 cases of *M. morganii* were identified in the bloodstream, corresponding to an annual incidence of 9.2 cases per million population, with a median patient age of 75.2 years and also indicating an incidence of almost zero cases per million in children and adolescents. Among the 709 cases reported, only 97 (13.7%) cases were isolated from the renal system (4). Although that study specifically reported on *M. morganii* isolated from bloodstream infections, which does not directly apply to the patient described in this case study, it still serves as an indication of the organism's very low prevalence in the pediatric population.

Another study in Taiwan reviewed 82,861 patients over a 6-year period with gram-negative nosocomial infections, from which, *M. morganii* was isolated in only 1,219 cases (1.47%) (5).

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Between 2015 and 2017, a pediatric clinic in Türkiye conducted a retrospective study on a total of 2,866 cases of urinary tract infection (UTI) in children. *M. morganii* was isolated in the urine cultures of only 11 patients, revealing a prevalence of 0.38% among pediatric UTIs (1).

Given the limited epidemiological data available, the presence of *M. morganii* in the pediatric population remains both a diagnostic and therapeutic challenge, mainly due to its natural and acquired resistance to antibiotics. In this report, we describe a rare case of *M. morganii* pyelonephritis in an 11-year-old boy with newly diagnosed hydronephrosis, highlighting this pathogen's potential to cause serious infection, particularly in medically complex children with communication barriers which may delay diagnosis and obscure the classic symptoms of UTI.

Case Report

An 11-year-old non-verbal, non-ambulatory male with cerebral palsy and developmental delay presented to the emergency department with a 3-day history of persistent fever (maximum temperature of 39.2 °C recorded at home) and irritability. One day prior to admission, he experienced five episodes of watery, non-bloody diarrhea and one episode of vomiting. His past medical history is significant extreme prematurity at 27 weeks' gestation, neonatal intensive care unit stay complicated by necrotizing enterocolitis requiring bowel surgery, and recurrent dental abscesses and infections. His immunizations were up to date, and his family history was unremarkable.

On physical examination, the patient appeared in mild discomfort but otherwise unremarkable. Laboratory workup revealed a low creatinine level of 0.7 mg/dL, a markedly elevated white blood cell (WBC) count of $28.8 \times 10^3/\mu\text{L}$, and a high C-reactive protein (CRP) of 319 mg/L. Urinalysis was abnormal consistent with large leukocytes esterase and WBC, raising concerns for a UTI. Consequently, a urine sample was collected by catheter for culture. Blood cultures and respiratory viral panels were unremarkable. The patient was initially managed for urosepsis and started on empiric ceftriaxone.

On day 2, the urine culture grew gram-negative bacilli, later identified as *M. morganii*. A susceptibility test resulted in admission on day 3 and also revealed resistance to ampicillin-sulbactam, nitrofurantoin, and trimethoprim-sulfamethoxazole, with retained susceptibility to ciprofloxacin, levofloxacin, and meropenem. Upon consultation with the infectious diseases department, ceftriaxone was discontinued, and the patient was

transitioned to intravenous levofloxacin. Despite improvement of the inflammatory markers, WBC count and CRP, and a negative repeat urine culture, the patient's fever persisted until day 13 of admission, which required continuation of intravenous levofloxacin for a total of 21 days.

Following the guidelines for UTI management in pediatrics, a retroperitoneal ultrasound was performed on day 4 of admission, which revealed dilation of the right renal pelvis and calyces (Figure 1 area between red x) with mild thinning of the right kidney's cortex (Figure 1 green arrow), indicating severe right hydronephrosis consistent with ureteropelvic junction (UPJ) obstruction. A mercaptoacetyltriglycine (MAG-3) scan showed significant delayed excretion on the right side, and a subsequent voiding cystourethrogram confirmed vesicoureteral reflux (Figure 2 a red arrow). Pediatric urology recommended ureteral stent placement to relieve the obstruction and facilitate urine drainage, but the procedure was deferred until the patient was no longer febrile. Two days after the stent placement (Figure 2b red arrow), an ultrasound revealed mildly improved hydronephrosis with a decrease in the right kidney's length from 13 cm to 11.5 cm.

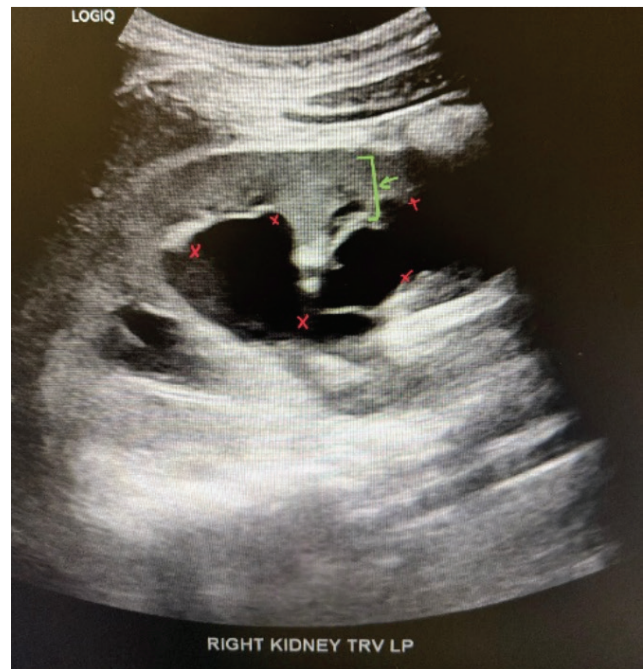


Figure 1. Retroperitoneal ultrasound demonstrating right-sided hydronephrosis. Retroperitoneal ultrasound showing severe right-sided hydronephrosis due to ureteropelvic junction obstruction, with marked dilatation of the renal pelvis and calyces (red x) and cortical thinning (green arrow)

TRV: Transverse, LP: Lower pole

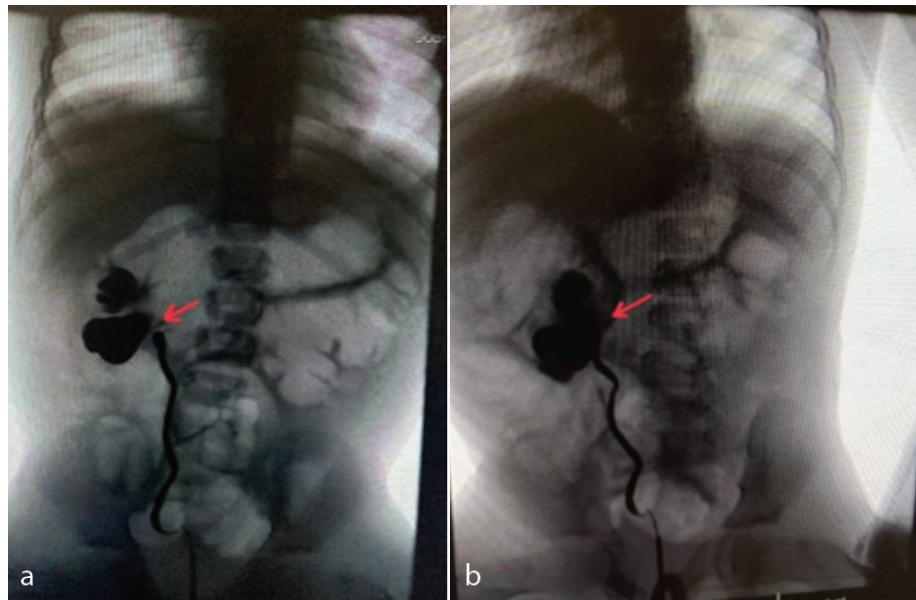


Figure 2. Voiding cystourethrogram (VCUG) images of the right kidney and ureter before and after double-J stent placement **(a)** VCUG prior to stent placement showing right-sided vesicoureteral reflux and proximal ureteropelvic junction (UPJ) obstruction (red arrow). **(b)** VCUG after double J stent placement showing relief of the obstruction at the right proximal UPJ

According to the patient's mother, this was his first known UTI. However, given his non-verbal status and the absence of typical symptoms such as dysuria, it was difficult to determine whether previous UTIs had gone undetected. A review of his medical records revealed two pelvic ultrasounds were performed two and 10 years earlier, both documenting normal renal anatomies.

Three months later, the stent was removed and a follow-up ultrasound revealed persistent severe right hydronephrosis with further shrinking in renal length to 8.9 cm. Despite the progressive decrease in renal measurements, a subsequent MAG-3 scan showed persistent delayed excretion and partial UPJ obstruction, suggesting chronic obstructive injury and cortical atrophy.

Discussion

One of the major challenges faced in managing *M. morganii* infections lies in its extensive antimicrobial resistance profile. Similar to other pathogens belonging to the *Enterobacteriaceae* family known collectively as the *Enterobacter*, *Serratia*, *Citrobacter*, *Providencia*, *Morganella* (ESCPM) group, *M. morganii* possesses an inducible chromosomal AmpC β -lactamase gene encoding the enzyme responsible for hydrolysis of many β -lactam antibiotics upon exposure (3,6). This mechanism confers natural resistance to penicillin, ampicillin, ampicillin/sulbactam, oxacillin,

first- and second-generation cephalosporins, and narrow-spectrum β -lactam/ β -lactamase inhibitor combinations (7). In addition, resistance to agents such as tetracyclines, tigecycline, polymyxins, and nitrofurantoin has been well-documented, further limiting empirical treatment options (8).

In many cases, *M. morganii* has been shown to retain susceptibility to third- and fourth-generation cephalosporins. Yet, clinical studies have suggested that treatment with these agents may result in a rapid emergence of resistance (6). A study by Mizrahi et al. (6) focused on the role of third-generation cephalosporins in inducing or selecting resistance among certain *Enterobacteriaceae* species. Their study demonstrated that the resistance developed is primarily attributable to the induction of AmpC β -lactamases enzymes, which exhibit stronger induction properties compared to those in other cephalosporins (6). This phenomenon explains why third- and fourth-generation cephalosporins are often ineffective against ESCPM organisms, which aligns with the lack of clinical improvement observed in our patient during the first three days of hospitalization.

Meanwhile, carbapenems, aztreonam, aminoglycosides, and chloramphenicol generally demonstrate good *in vitro* activity. However, reports of multidrug-resistant *M. morganii* strains emphasize the need for careful antimicrobial use and close monitoring (9). With the variability in resistance and

the risk of new resistant strains developing, susceptibility testing is vital in order to guide appropriate antimicrobial therapy.

A recent study by Tasanapak et al. (10) reviewed the geographic prevalence of fluoroquinolone resistance in *M. morganii* from 1998 to 2024. Their study reported a rise in resistance from 2% between 1993-1997 to 30% between 2013-2024. The highest resistance rates were in Africa (55%), followed by South America (36%), Asia (35%), and North America (11%). The rapid increase in resistance was mainly attributed to plasmid-mediated resistance mechanisms, which protect DNA gyrase and topoisomerase IV, encode drug-modifying enzymes, and sometimes promote efflux pumping, thereby reducing fluoroquinolone efficacy. Despite limitations such as regional variations in antibiotic use, their study provided valuable insights into global resistance trends (10).

Aside from the challenges posed by *M. morganii*'s drug resistance, the presence of right-sided hydronephrosis in our patient raised concerns about the chronicity of urinary tract involvement. Although imaging carried out two and 10 years earlier demonstrated normal renal anatomy, the development of hydronephrosis and mild cortical thinning in the current study indicated an underlying chronic or congenital obstruction rather than a purely acute process. The absence of renal congenital malformations, along with the exclusion of extrinsic crossing vessels on imaging, supported the diagnosis of congenital UPJ obstruction. Since the blockage may have been partial or subclinical initially, the current infection and inflammation likely contributed to symptomatic presentation and may have exacerbated the obstruction.

In pediatric patients, particularly those with limited communication abilities, early identification of pathogens with unusual resistance patterns is crucial. Prompt initiation of targeted therapy based on culture and sensitivity results has been shown to markedly prevent complications such as persistent infection, renal damage, progression to sepsis, and even death (3,4). This case serves as a reminder to consider less common organisms such as *M. morganii* in pediatric UTIs and highlights the importance of timely imaging, appropriate antimicrobial selection, and continuous microbiological monitoring.

Ethics

Informed Consent: This case report was a retrospective descriptive observation describing a single patient. Written

informed consent from the patient's guardian could not be obtained. All identifying information has been removed to protect confidentiality.

Footnotes

Authorship Contributions

Surgical and Medical Practices: K.G., V.E., Concept: Y.A., V.E., Design: Y.A., Data Collection or Processing: Y.A., Analysis or Interpretation: Y.A., K.G., V.E., Literature Search: Y.A., Writing: Y.A.

Conflict of Interest: The authors declare they have no conflicts of interest regarding the publication of this case report.

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