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Clinical Management of Urosepsis in Emergency Settings: Nursing Care, Nutritional Support, and EMS Interventions

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Abstract

Background: Urosepsis is a life-threatening systemic response to urinary tract infections resulting from a dysregulated host reaction leading to inflammation, tissue hypoperfusion, and organ dysfunction. It commonly arises from ascending infections such as pyelonephritis, complicated UTIs, and infections associated with urinary obstruction or indwelling catheters. Gram-negative bacteria, particularly *Escherichia coli*, are the predominant causative organisms.

Aim: This review aims to summarize current evidence on the clinical presentation, diagnostic workup, management strategies, and multidisciplinary roles in improving outcomes in urosepsis.

Methods: The article synthesizes evidence from clinical trials, epidemiologic trends, and contemporary guidelines focusing on etiology, pathophysiology, diagnostic evaluation, emergency management, and interprofessional practices.

Results: Urosepsis continues to exhibit high morbidity and mortality, with death rates reaching up to 40% despite advances in care. Early recognition, rapid initiation of antibiotics within one hour, and aggressive fluid resuscitation significantly improve survival. CT imaging is valuable in identifying obstructive or structural causes requiring source-control procedures such as stenting or nephrostomy. Complications include acute kidney injury, septic shock, and multiorgan dysfunction. Multidisciplinary collaboration, including infectious-disease pharmacists and urology specialists, enhances antimicrobial stewardship and improves outcomes.

Conclusion: Early diagnosis, prompt antimicrobial therapy, aggressive resuscitation, and timely correction of urologic abnormalities are essential for reducing mortality and long-term complications. Interprofessional coordination remains critical to optimal outcomes.

Keywords: Urosepsis, urinary tract infection, sepsis management, antimicrobial therapy, source control, emergency care, multidisciplinary team.

Introduction

Sepsis represents a complex and life-threatening clinical syndrome that arises from a maladaptive host response to infection, resulting in widespread physiologic, cellular, and biochemical disturbances. This dysregulated response extends beyond localized infection and initiates systemic inflammation that can rapidly progress to multiple organ dysfunction syndrome and death if not promptly recognized and treated. The defining feature of sepsis is not merely the presence of an infectious agent, but rather the host's exaggerated and poorly controlled immune reaction, which disrupts normal homeostasis and impairs vital organ function. Within this broad clinical spectrum, urosepsis refers specifically to sepsis that originates from an infection within the urogenital tract, including the bladder, kidneys, prostate, or associated structures [1][2]. Urosepsis constitutes a significant proportion of sepsis cases encountered in both emergency and inpatient settings. Infections of the urinary tract are common, and while many remain localized and uncomplicated, a subset progresses to systemic involvement, particularly in vulnerable populations such as older adults, individuals with urinary obstruction, immunocompromised patients, and those with indwelling urinary devices. Once bacteremia develops, inflammatory mediators are released in large

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quantities, triggering endothelial dysfunction, altered microcirculation, and impaired oxygen delivery to tissues. These pathophysiologic changes contribute to hypotension, metabolic derangements, and progressive organ failure. Clinically, urosepsis poses a diagnostic challenge because early manifestations may be subtle or nonspecific, especially in elderly or critically ill patients. Delay in diagnosis and treatment significantly worsens outcomes, underscoring the importance of early recognition, prompt antimicrobial therapy, and rapid hemodynamic support. Given its high morbidity and mortality, urosepsis remains a major focus of clinical research and quality improvement initiatives. Understanding its underlying mechanisms, risk factors, and clinical presentations is essential for improving patient outcomes and reducing the global burden of sepsis-related complications [1][2].

Etiology

The etiological agents responsible for urosepsis largely reflect the microbial spectrum associated with urinary tract infections. Gram-negative bacteria predominate, with Escherichia coli being the most frequently isolated pathogen, accounting for approximately half of all cases. This organism possesses several virulence factors, including adhesins and endotoxins, that facilitate colonization of the urinary epithelium and promote systemic inflammatory responses once bacteremia occurs. Other commonly implicated gram-negative organisms include Proteus species, Enterobacter species, and Klebsiella species, each contributing a substantial proportion of urosepsis cases [3][4]. These pathogens are particularly associated with complicated urinary tract infections, including those related to urinary obstruction, catheterization, or structural abnormalities of the urinary tract. Pseudomonas aeruginosa, although less frequently encountered, is of particular clinical concern due to its intrinsic resistance to multiple antimicrobial agents and its association with healthcare-associated infections. Gram-positive organisms also contribute to the etiologic landscape of urosepsis, especially in patients with indwelling devices or underlying comorbidities. The distribution of causative organisms may vary based on patient demographics, healthcare exposure, and local antimicrobial resistance patterns, emphasizing the importance of region-specific surveillance data when selecting empiric therapy [3][4]. The progression from localized urinary infection to systemic sepsis is influenced not only by microbial virulence but also by host factors such as immune status, age, and the presence of chronic disease. Conditions that impair urinary drainage or compromise mucosal defenses create an environment conducive to bacterial proliferation and bloodstream invasion. Consequently, urosepsis often reflects an interaction between pathogenic organisms and host vulnerability rather than the presence of a single causative agent alone.

Epidemiology

Over recent decades, the incidence of sepsis in the United States has demonstrated a consistent upward trend, a phenomenon attributed to population aging, increased survival from chronic illnesses, greater use of invasive medical devices, and improved recognition and reporting of sepsis cases. Despite this rise in incidence, overall mortality from sepsis has declined, reflecting advances in early diagnosis, antimicrobial therapy, and critical care management. Nevertheless, urosepsis remains associated with substantial mortality, with reported death rates ranging from 30% to 40%, highlighting its clinical severity [5][6]. Urosepsis accounts for a significant proportion of sepsis cases, particularly among hospitalized and elderly patients. The disease burden is further amplified by its frequent association with severe complications, including acute respiratory distress syndrome, acute kidney injury, and disseminated intravascular coagulopathy. These complications not only increase mortality risk but also contribute to prolonged hospital stays, higher healthcare costs, and long-term functional impairment among survivors [5][6]. Epidemiologic patterns suggest that the severity of urosepsis has increased over time, with patients presenting with more advanced organ dysfunction at the time of diagnosis. This trend may reflect delayed presentation, increasing antimicrobial resistance, or a higher prevalence of comorbid conditions. Urosepsis therefore represents an ongoing public health challenge, requiring sustained efforts in prevention, early detection, and comprehensive management strategies to reduce its impact on patient outcomes and healthcare systems.

Pathophysiology

The pathogenesis of urosepsis begins with the development of a urinary tract infection, most commonly initiated by colonization of the urethral meatus or vaginal introitus with uropathogenic or fecal flora. These organisms ascend through the urethra to the bladder, where they may remain localized or continue upward through the ureters to involve the renal parenchyma, resulting in pyelonephritis. In some cases, renal infection may also arise through hematogenous or lymphatic spread, particularly in patients with systemic bacteremia or compromised host defenses [7][8]. While microbial invasion initiates the disease process, the host immune response ultimately determines the progression and severity of urosepsis. Recognition of microbial components by host cell surface receptors triggers an intense pro-inflammatory cascade characterized by the release of cytokines, chemokines, and other mediators. This response leads to endothelial activation, increased vascular permeability, and the recruitment of neutrophils that release bactericidal substances. Although initially protective, this exaggerated inflammatory response can result in tissue injury, cellular necrosis, and widespread edema [7][8]. Following this hyperinflammatory phase, a

compensatory anti-inflammatory response often develops, leading to immune dysfunction and increased susceptibility to secondary infections. Neutrophil impairment, altered coagulation pathways, and dysregulation of autonomic and endocrine systems further exacerbate organ dysfunction. The interplay between these opposing immune responses creates a dynamic and unstable physiologic state, explaining the rapid clinical deterioration observed in severe urosepsis.

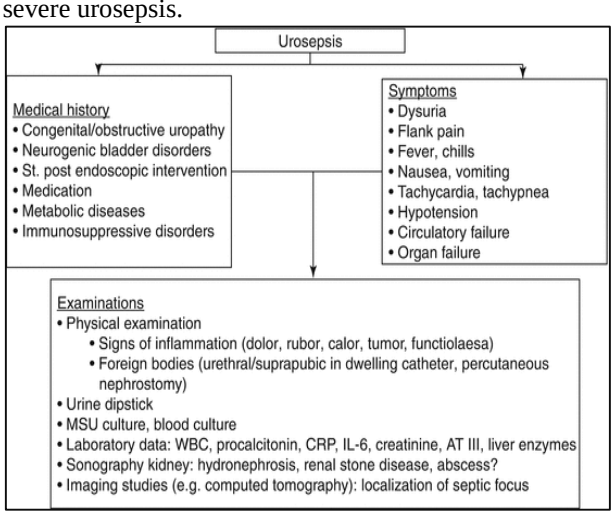

Fig. 1: Urosepsis.

History and Physical

The clinical presentation of urosepsis varies widely and depends on the site of infection within the urogenital tract, the causative organism, and host factors. Infections confined to the bladder typically manifest as cystitis, characterized by dysuria, urinary frequency and urgency, suprapubic discomfort, and hematuria. The presence of fever, chills, rigors, or profound fatigue suggests progression beyond localized infection and raises concern for systemic involvement [9]. Upper urinary tract infections such as pyelonephritis are associated with more pronounced systemic symptoms. Patients commonly present with high-grade fever, flank pain, costovertebral angle tenderness, and gastrointestinal symptoms including nausea and vomiting. Lower urinary tract symptoms may coexist, and atypical presentations, such as epigastric or lower abdominal pain, may obscure the diagnosis, particularly in older adults [10]. Acute bacterial prostatitis represents another potential source of urosepsis, particularly in men. This condition often presents with fever, chills, dysuria, pelvic or perineal pain, and cloudy urine. Obstructive urinary symptoms may occur, and physical examination typically reveals a tender, edematous, and firm prostate gland [11]. A comprehensive history should also explore predisposing factors such as recurrent urinary tract infections, nephrolithiasis, immunosuppression, and the presence of chronic indwelling urinary catheters. These factors increase the likelihood of complicated infection and systemic progression, underscoring the need for heightened clinical vigilance and early intervention.

Evaluation

The evaluation of urosepsis requires a structured and systematic approach that integrates clinical suspicion of sepsis with objective evidence of a urinary tract source. Early recognition is critical, as delayed diagnosis is strongly associated with increased morbidity and mortality. Clinicians must first consider sepsis in any patient presenting with signs of systemic illness in the context of urinary symptoms or known risk factors for complicated urinary tract infection. Although systemic inflammatory response syndrome criteria were historically central to diagnosis, contemporary definitions no longer require their presence. Nevertheless, abnormalities in temperature regulation, cardiovascular status, respiratory function, and white blood cell count remain clinically relevant indicators of systemic infection and physiological stress. Sepsis is established when infection is accompanied by evidence of organ dysfunction. In urosepsis, this commonly includes acute kidney injury manifested by reduced urine output or rising serum creatinine, acute encephalopathy characterized by altered mental status, or hematologic abnormalities such as thrombocytopenia. These findings reflect the systemic consequences of infection-induced inflammation and impaired tissue perfusion. When such features coexist with urinary symptoms such as dysuria, flank pain, or suprapubic discomfort, a diagnosis of complicated urinary tract infection with systemic involvement should be strongly considered. Physical examination findings including costovertebral angle tenderness, bladder distension, or signs of urinary retention further support this assessment. Radiologic findings and laboratory evidence of bacteriuria and leukocyturia play a confirmatory role in establishing the urinary tract as the source of infection [12][13].

Once urosepsis is suspected, prompt laboratory evaluation is essential. Urinalysis and urine culture are fundamental investigations, as they provide evidence of infection and allow subsequent pathogen identification and antimicrobial susceptibility testing. Blood cultures should be obtained prior to antibiotic administration to detect bacteremia and guide targeted therapy. A complete blood cell count assists in identifying leukocytosis, leukopenia, or thrombocytopenia, while a comprehensive metabolic panel provides critical information regarding renal and hepatic function, electrolyte disturbances, and metabolic derangements. Measurement of serum lactate is particularly important, as elevated levels indicate tissue hypoperfusion and correlate with disease severity and prognosis. Imaging plays a central role in identifying structural or obstructive causes that may precipitate or perpetuate urosepsis. Postrenal obstruction represents one of the most frequent and clinically significant contributors. Ultrasonography is

capable of identifying many common etiologies, including hydronephrosis and prostatic abscess, with high sensitivity. However, its effectiveness is limited by operator dependency and availability, particularly in emergency settings. Computed tomography has therefore become the preferred imaging modality in many institutions due to its widespread availability, reproducibility, and ability to detect subtle anatomic abnormalities. CT imaging can identify obstructing calculi, abscesses, emphysematous infections, and other complications that may not be readily apparent on ultrasound, making it invaluable in the comprehensive evaluation of suspected urosepsis [14].

Treatment / Management

The management of urosepsis is complex and time-sensitive, requiring immediate intervention aimed at infection control, hemodynamic stabilization, and prevention of progressive organ dysfunction. Over recent decades, treatment has become increasingly challenging due to the rising prevalence of multidrug-resistant organisms causing urinary tract infections. This trend is largely attributed to widespread and often empiric antibiotic use in suspected infections, as definitive culture and susceptibility results are typically unavailable for several days. As a result, inappropriate or suboptimal antimicrobial exposure has contributed significantly to resistance patterns observed in clinical practice [15][16]. Evidence from landmark clinical trials has demonstrated that early and aggressive treatment strategies reduce mortality in sepsis. Early goal-directed therapy established foundational principles that continue to guide modern management despite ongoing refinements. Central to these principles is the rapid initiation of empiric antimicrobial therapy directed toward the suspected source of infection, combined with supportive measures to stabilize organ function and adjunctive therapies tailored to disease severity. Antimicrobial therapy should be administered as soon as possible, ideally within one hour of establishing the diagnosis, but only after appropriate urine and blood cultures have been obtained. Delays in antibiotic administration have a profound impact on outcomes, with each hour of delay beyond the initial critical period associated with a measurable decrease in survival. Selection of empiric antibiotics should be guided by local antimicrobial susceptibility patterns, patient-specific risk factors, and the likelihood of resistant organisms. Common empiric regimens often include a third-generation cephalosporin, a beta-lactam combined with a beta-lactamase inhibitor such as piperacillin-tazobactam, or a fluoroquinolone. In patients with prior healthcare exposure or known colonization with resistant pathogens, broader coverage may be required initially. Dosing must be carefully adjusted to account for organ dysfunction, particularly renal and hepatic impairment, which frequently accompanies severe sepsis and alters drug clearance [2].

Beyond antimicrobial therapy, supportive management is a cornerstone of treatment. Early and adequate fluid resuscitation with isotonic crystalloids is strongly recommended, with an initial target of at least 30 mL per kilogram of body weight. This intervention aims to restore intravascular volume, improve tissue perfusion, and mitigate the effects of sepsis-induced vasodilation. When hypotension persists despite adequate fluid resuscitation, early initiation of vasopressor therapy is indicated to maintain a mean arterial pressure of at least 65 mm Hg. Norepinephrine is the first-line vasopressor, with epinephrine and vasopressin reserved for refractory cases. Additional supportive measures include careful glucose control and individualized consideration of corticosteroids and blood products, although the benefits of these interventions remain subjects of ongoing debate in the literature [17]. Definitive management of urosepsis also requires prompt correction of underlying urologic abnormalities that serve as ongoing sources of infection. Relief of urinary obstruction is critical and may involve bladder catheterization for acute retention or interventional procedures such as ureteral stenting or nephrostomy placement to bypass obstructing calculi. Delayed source control significantly compromises the effectiveness of medical therapy. Optimal outcomes in urosepsis therefore depend on timely diagnosis, early antimicrobial administration, aggressive supportive care, and rapid resolution of contributory anatomic factors through coordinated multidisciplinary intervention.

Differential Diagnosis

The differential diagnosis of urosepsis encompasses a wide range of infectious and noninfectious conditions affecting the urogenital tract, many of which may present with overlapping clinical features. Accurate differentiation is essential, as management strategies and prognostic implications vary depending on the underlying cause and the anatomical extent of infection. Urosepsis represents the systemic manifestation of infection originating anywhere along the urinary tract, from the lower urinary system to the kidneys and associated structures. Consequently, clinicians must carefully evaluate the site of infection, the severity of local involvement, and the presence of complicating factors such as obstruction or abscess formation. Uncomplicated urinary tract infection and cystitis typically present with localized lower urinary tract symptoms, including dysuria, urinary frequency, urgency, and suprapubic discomfort, and usually lack systemic features unless progression occurs. In contrast, pyelonephritis involves upper urinary tract structures and is more likely to produce fever, flank pain, costovertebral angle tenderness, nausea, and vomiting, reflecting parenchymal renal involvement. When bacteremia and systemic inflammatory responses develop, pyelonephritis becomes a leading precursor to urosepsis. Acute bacterial prostatitis

should be considered in male patients presenting with fever, pelvic or perineal pain, obstructive urinary symptoms, and a tender, edematous prostate on examination. Failure to recognize and adequately treat prostatitis may lead to complications such as prostatic abscess, which can act as a persistent nidus of infection and significantly worsen systemic illness. Renal and prostatic abscesses represent more advanced and localized infectious processes that may mimic or coexist with urosepsis. These conditions often present with persistent fever despite antibiotic therapy and require imaging for diagnosis. Urolithiasis, particularly when associated with urinary obstruction, is a critical differential diagnosis, as obstructed infected systems predispose patients to rapid clinical deterioration. Obstructive calculi can impair urinary drainage, promote bacterial proliferation, and facilitate the progression from localized infection to sepsis. A thorough diagnostic evaluation is therefore required to identify the precise etiology, as effective management of urosepsis depends on addressing both the infectious process and any underlying structural or functional abnormalities of the urinary tract [17][18].

Complications

Urosepsis is a potentially reversible condition when identified early and managed appropriately; however, delays in recognition or treatment can result in severe and life-threatening complications. The systemic inflammatory response associated with sepsis leads to widespread endothelial dysfunction, impaired tissue perfusion, and progressive organ injury. Among the most common and clinically significant complications is acute kidney injury, which may arise from a combination of hypotension, inflammatory-mediated microvascular damage, and direct renal parenchymal infection. Renal failure not only worsens patient outcomes but also complicates antimicrobial dosing and fluid management, thereby increasing overall morbidity. Septic shock represents one of the most severe complications of urosepsis and is characterized by persistent hypotension requiring vasopressor support despite adequate fluid resuscitation. This state reflects profound circulatory and cellular dysfunction and is associated with a markedly increased risk of mortality. Multiorgan dysfunction syndrome may follow, involving the respiratory, cardiovascular, hepatic, hematologic, and central nervous systems. Patients may develop acute respiratory failure requiring mechanical ventilation, coagulopathies due to dysregulated clotting pathways, and altered mental status secondary to encephalopathy. These complications often necessitate prolonged intensive care unit admission and are associated with long-term functional impairment in survivors. In addition to systemic consequences, uncontrolled urosepsis may lead to localized complications, including renal cortical necrosis, abscess formation, and progression of obstructive uropathy. Persistent infection can also predispose patients to recurrent urinary tract infections and chronic kidney disease. Ultimately, without timely intervention, urosepsis can culminate in death, underscoring the importance of early diagnosis and aggressive management [18]. Preventing these complications relies on rapid source control, appropriate antimicrobial therapy, and meticulous supportive care tailored to the patient's evolving clinical status.

Patient Education

Effective prevention of urosepsis begins with patient education and early engagement with healthcare services. Individuals with known risk factors for urinary tract infections, such as recurrent UTIs, urinary obstruction, indwelling catheters, diabetes mellitus, or immunocompromised states, should be counseled regarding symptom recognition and the importance of seeking prompt medical evaluation. Early identification and treatment of uncomplicated urinary infections significantly reduce the risk of progression to systemic infection and sepsis. Patients should be educated about common warning signs of urinary tract infection, including dysuria, urinary urgency, frequency, flank pain, fever, and malaise, and advised not to delay care when these symptoms arise. Emphasis should also be placed on adherence to prescribed antimicrobial regimens, as incomplete or inappropriate antibiotic use contributes to treatment failure, recurrence, and the development of resistant organisms. Clear communication regarding dosing schedules, treatment duration, and potential adverse effects enhances compliance and therapeutic effectiveness. Preventive counseling should additionally address modifiable risk factors, such as adequate hydration, proper perineal hygiene, and timely management of underlying urologic conditions. For patients with chronic urinary devices, education on catheter care and early signs of infection is essential. By empowering patients with knowledge and reinforcing the importance of adherence and early intervention, healthcare providers play a critical role in reducing the incidence, severity, and complications of urosepsis [18].

Enhancing Healthcare Team Outcomes

Optimal outcomes in urosepsis depend on coordinated care delivered by an interprofessional healthcare team. Given the complexity of the condition and the potential for rapid clinical deterioration, collaboration among multiple specialties is essential. Physicians from nephrology, infectious diseases, urology, and critical care contribute specialized expertise in managing organ dysfunction, selecting appropriate antimicrobial therapy, addressing anatomic abnormalities, and providing advanced life support when necessary. Early involvement of these specialists facilitates comprehensive evaluation and timely intervention. Nurses play a central role in monitoring patients for early signs of sepsis progression, administering

therapies, and maintaining close communication with the treating team. Their continuous bedside assessment is critical for detecting changes in vital signs, urine output, and mental status that may signal worsening disease. Pharmacists contribute by ensuring the timely preparation and administration of antibiotics, optimizing dosing in the presence of renal or hepatic impairment, and identifying potential drug interactions or adverse effects that could compromise patient safety. Many patients with urosepsis require intensive care unit admission, where frequent laboratory testing, hemodynamic monitoring, and repeated imaging studies are often necessary. Effective communication and role clarity among team members improve efficiency, reduce delays in care, and enhance patient safety. Addressing predisposing urologic abnormalities through timely procedural intervention is equally important, as definitive source control significantly reduces morbidity. Evidence supports that interprofessional collaboration improves clinical outcomes, shortens hospital length of stay, and reduces mortality in severe infections, highlighting the critical value of team-based care in the management of urosepsis [19][20].

Outcomes

Clinical outcomes following urosepsis are highly variable and are determined by a complex interaction between the severity of the infectious process, the timeliness of diagnosis, the effectiveness of source control, and patient-specific factors. The anatomical origin of infection within the urinary tract and the extent of systemic involvement at presentation strongly influence prognosis. Patients with uncomplicated infections who receive early, appropriate antimicrobial therapy generally experience favorable outcomes, whereas those presenting with advanced sepsis or septic shock face substantially higher risks of morbidity and mortality. Delays in recognition and treatment allow for progression of the dysregulated inflammatory response, resulting in widespread endothelial injury, tissue hypoperfusion, and multiorgan dysfunction. The microbiological profile of the causative organism is another critical determinant of outcome. Infections caused by multidrug-resistant pathogens are associated with longer hospital stays, increased need for intensive care, and higher mortality rates, particularly when initial empiric therapy fails to provide adequate coverage. Antimicrobial resistance complicates therapeutic decision-making and increases the likelihood of persistent infection or relapse. Patient-related factors, including advanced age, diabetes mellitus, chronic kidney disease, immunosuppression, and the presence of indwelling urinary devices, further worsen prognosis by impairing host defense mechanisms and limiting physiologic reserve [21].

Interprofessional collaboration plays a decisive role in improving outcomes. The involvement of a board-certified infectious disease pharmacist is especially valuable in optimizing antimicrobial management. Pharmacist expertise supports the selection of empiric and targeted antimicrobial regimens based on local susceptibility patterns, ensures appropriate dosing in the setting of renal or hepatic dysfunction, identifies clinically significant drug–drug interactions, and provides ongoing monitoring for toxicity or therapeutic failure. This specialized input enhances antimicrobial stewardship efforts and contributes to improved clinical response and reduced complications. When urosepsis is not promptly or adequately treated, mortality rates remain unacceptably high. Even among survivors, recovery is often prolonged and may be complicated by persistent functional impairment. Residual renal dysfunction is a common long-term consequence, reflecting the vulnerability of the kidneys to ischemic and inflammatory injury during sepsis. Some patients experience incomplete recovery of renal function, predisposing them to chronic kidney disease and increased susceptibility to future infections. Additionally, survivors may face prolonged weakness, reduced quality of life, and recurrent hospitalizations. These outcomes highlight the importance of early recognition, aggressive management, and coordinated multidisciplinary care in reducing the long-term burden of urosepsis and improving patient survival and recovery [21][22].

Conclusion:

Urosepsis remains a major clinical challenge due to its rapid progression from localized urinary tract infection to systemic inflammatory dysfunction and multiorgan involvement. The condition's high mortality underscores the need for immediate recognition and intervention. Early diagnosis supported by targeted laboratory testing, blood and urine cultures, and appropriate imaging is central to identifying both the infectious source and any anatomical contributors such as obstruction or abscess formation. Timely administration of empiric antibiotics—ideally within the first hour—combined with aggressive fluid resuscitation and vasopressor therapy when needed, significantly improves patient survival. Equally important is timely source control through catheterization, ureteral stenting, or nephrostomy to relieve obstruction and prevent ongoing bacterial proliferation. Complications such as acute kidney injury, septic shock, respiratory failure, and coagulopathies further highlight the importance of rapid and comprehensive care. Long-term consequences—including persistent renal dysfunction, recurrent infections, and reduced functional status—demonstrate that the impact of urosepsis extends well beyond initial hospitalization. Interprofessional collaboration, particularly the involvement of pharmacists, infectious-disease specialists, and urologists, improves antimicrobial stewardship, optimizes patient monitoring, and leads to better outcomes. Overall, reducing morbidity and mortality from urosepsis depends on rapid diagnosis,

timely therapy, and coordinated multidisciplinary management.

References:

1. Björck V, Påhlman LI, Bodelsson M, Petersson AC, Kander T. Morbidity and mortality in critically ill patients with invasive group A streptococcus infection: an observational study. Critical care (London, England). 2020 Jun 6:24(1):302. doi: 10.1186/s13054-020-03008-z. Epub 2020 Jun 6
2. Shimoni Z, Salah M, Kasem A, Hermush V, Froom P. Bacterial Resistance to Cephalosporin Treatment in Elderly Stable Patients Hospitalized With a Urinary Tract Infection. The American journal of the medical sciences. 2020 Sep:360(3):243-247. doi: 10.1016/j.amjms.2020.05.008.
3. Vikrant S, Gupta D, Singh M. Epidemiology and outcome of acute kidney injury from a tertiary care hospital in India. Saudi journal of kidney diseases and transplantation : an official publication of the Saudi Center for Organ Transplantation, Saudi Arabia. 2018 Jul-Aug:29(4):956-966. doi: 10.4103/1319-2442.239633.
4. Scotland KB, Lange D. Prevention and management of urosepsis triggered by ureteroscopy. Research and reports in urology. 2018:10():43-49. doi: 10.2147/RRU.S128071.
5. Liu YQ, Lu J, Hao YC, Xiao CL, Ma LL. [Predicting model based on risk factors for urosepsis after percutaneous nephrolithotomy]. Beijing da xue xue bao. Yi xue ban = Journal of Peking University. Health sciences. 2018 Jun 18:50(3):507-513
6. Kozyrakis D, Perikleous S, Chatzistamou SE, Kateris D, Soukias G, Karatzas A, Dimitriadis I. Is There a Role for Double J Stent Culture in Contemporary Urology? Urologia internationalis. 2018:100(2):203-208. doi: 10.1159/000486798.
7. Sarhan M, von Mässenhausen A, Hugo C, Oberbauer R, Linkermann A. Immunological consequences of kidney cell death. Cell death & disease. 2018 Jan 25:9(2):114. doi: 10.1038/s41419-017-0057-9.
8. Kodner CM, Thomas Gupton EK. Recurrent urinary tract infections in women: diagnosis and management. American family physician. 2010 Sep 15:82(6):638-43
9. Saade A, Styczynski J, Cesaro S, Infectious Disease Working party of EBMT. BK virus infection in allogeneic hematopoietic cell transplantation: An update on pathogenesis, immune responses, diagnosis and treatments. The Journal of infection. 2020 Sep:81(3):372-382. doi: 10.1016/j.jinf.2020.06.009.
10. Parlak E. A case of glomerulonephritis caused by brucellosis. Tropical doctor. 2020 Oct:50(4):360-361. doi: 10.1177/0049475520929505.
11. Reddivari AKR, Mehta P. Prostatic Abscess. StatPearls. 2025 Jan
12. Vigil HR, Hickling DR. Urinary tract infection in the neurogenic bladder. Translational andrology and urology. 2016 Feb:5(1):72-87. doi: 10.3978/j.issn.2223-4683.2016.01.06.
13. Dreger NM, Degener S, Ahmad-Nejad P, Wöbker G, Roth S. Urosepsis--Etiology, Diagnosis, and Treatment. Deutsches Arzteblatt international. 2015 Dec 4:112(49):837-47; quiz 848. doi: 10.3238/arztebl.2015.0837.
14. Morin A, Bergevin M, Rivest N, Lapointe SP. Antibiotic prophylaxis for transrectal ultrasound-guided prostate needle biopsy: Compared efficacy of ciprofloxacin vs. the ciprofloxacin/fosfomycin tromethamine combination. Canadian Urological Association journal = Journal de l'Association des urologues du Canada. 2020 Aug:14(8):267-272. doi: 10.5489/cuaj.6248.
15. Caron F, Galperine T, Flateau C, Azria R, Bonacorsi S, Bruyère F, Cariou G, Clouqueur E, Cohen R, Doco-Lecompte T, Elefant E, Faure K, Gauzit R, Gavazzi G, Lemaitre L, Raymond J, Senneville E, Sotto A, Subtil D, Trivalle C, Merens A, Etienne M. Practice guidelines for the management of adult community-acquired urinary tract infections. Medecine et maladies infectieuses. 2018 Aug:48(5):327-358. doi: 10.1016/j.medmal.2018.03.005.
16. Wagenlehner FM, Tandogdu Z, Bjerklund Johansen TE. An update on classification and management of urosepsis. Current opinion in urology. 2017 Mar:27(2):133-137. doi: 10.1097/MOU.0000000000000364.
17. Reynolds LF, Kroczak T, Pace KT, D'Arcy Honey RJ, Ordon M, Lee JY. Are Routine Laboratory Investigations Necessary Following Percutaneous Nephrolithotomy? Urology. 2020 Sep:143():80-84. doi: 10.1016/j.urology.2020.05.025.
18. Grüne B, Viehweger V, Waldbillig F, Nientiedt M, Kriegmair MC, Rassweiler-Seyfried MC, Ritter M. Preoperative urine culture - Is it necessary to prevent infectious complications following ureterorenoscopy? Journal of microbiological methods. 2020 Jun:173():105933. doi: 10.1016/j.mimet.2020.105933.
19. Heppner HJ, Yapan F, Wiedemann A. [Urosepsis in Geriatric Patients]. Aktuelle Urologie. 2016 Feb:47(1):54-9. doi: 10.1055/s-0041-106184.
20. Goveas B. Urosepsis: A simple infection turns toxic. The Nurse practitioner. 2017 Jul 15:42(7):53-54. doi: 10.1097/01.NPR.0000520425.91534.b8.
21. Pietropaolo A, Hendry J, Kyriakides R, Geraghty R, Jones P, Aboumarzouk O, Somani BK. Outcomes of Elective Ureteroscopy for Ureteric Stones in Patients with Prior Urosepsis and Emergency Drainage: Prospective Study over 5 yr from a Tertiary Endourology Centre. European

urology focus. 2020 Jan 15:6(1):151-156. doi: 10.1016/j.euf.2018.09.001.

22. Zhu Z, Cui Y, Zeng F, Li Y, Chen Z, Hequn C. Comparison of suctioning and traditional ureteral access sheath during flexible ureteroscopy in the treatment of renal stones. World journal of urology. 2019 May:37(5):921-929. doi: 10.1007/s00345-018-2455-8.