Urosepsis

10

Axel Heidenreich and Andrea Thissen

10.1 Definition

Urosepsis is defined as sepsis caused by urinary tract infection (UTI) and accounts for 25 % of all sepsis cases [1]. Patients with anatomical and/or functional abnormalities of the urinary tract and patients with comorbidities such as poor-regulated diabetes mellitus or immunosuppressive therapy are predisposed to sepsis syndrome [2]. Severe sepsis is a potentially life-threatening condition related to a mortality rate ranging between 30 and 50 % [3]. Hence, early goal-directed therapy and empirical antibiotic treatment are crucial aspects of patient care in urosepsis.

10.2 Medical History

Patients should be questioned regarding symptoms of urinary tract infection, such as dysuria, flank pain, or fever and chills (see Fig. 10.1). Pain and its duration, localization, and quality must be carefully gathered and patient's medication reviewed. Furthermore, information about age and underlying illnesses such as diabetes mellitus, cancer, or other forms of compromising disorders contributes to acquiring a realistic assessment of the patient's condition.

10.3 Diagnostics

Physical examination with assessment of vital signs (body temperature, heart and respiratory rate, blood pressure, urine output, vigilance) and results of laboratory data (white blood cell count, c-reactive protein, procalcitonin, AT III, liver enzymes, blood gas analysis) are essential parameters for the evaluation of the disease

A. Heidenreich • A. Thissen (✉)
Department of Urology, RWTH University Aachen, Pauwelsstr. 30, Aachen 52074, Germany
e-mail: aheidenreich@ukaachen.de; anthissen@ukaachen.de

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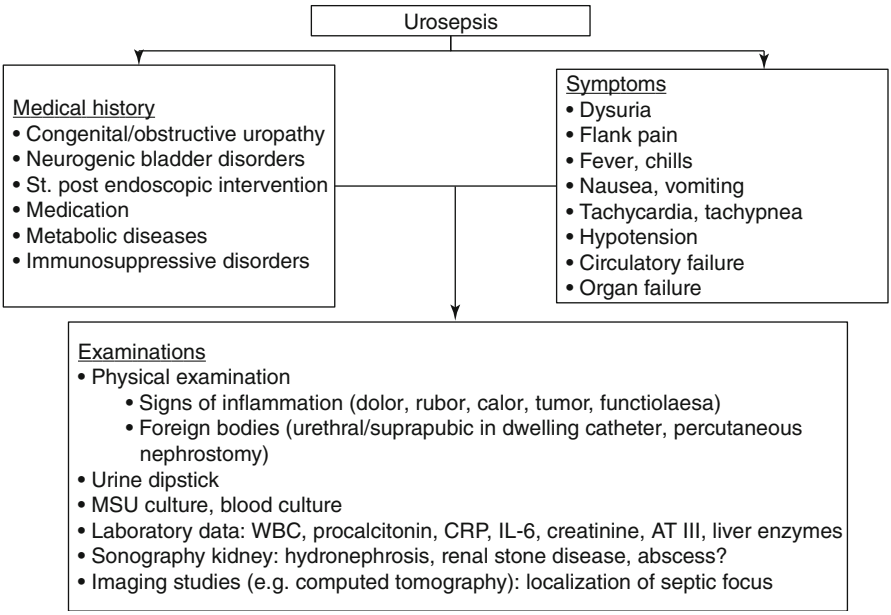

Fig. 10.1 Flow chart demonstrating the pathway from symptoms to diagnosis

severity in sepsis. Procalcitonin has emerged as an early reliable marker suggestive of severe bacterial infection and may help decide if an immediate urological intervention is required or not [4]. Blood and urine cultures as well as local swabs (e.g., of abscesses) ought to be taken before beginning with antibiotic treatment. Evaluation of the upper urinary tract should be performed by sonography to rule out obstructive uropathy. Additional imaging studies such as computed tomography may be helpful to confirm a potential source of infection and localize the septic focus, such as perinephric or intrarenal abscess formation.

10.4 Differential Diagnosis

Nosocomial infection	Indwelling foreign body infections, HAP (hospital-acquired pneumonia), wound infection
Respiratory system	Pneumonia, pyothorax, lung abscess
Cardiac system	Endocarditis
Gastrointestinal tract	Cholecystitis, pancreatitis, perforated sigma diverticulitis, peritonitis
Central nervous system	Meningococcal disease

10.5 General Facts

Predominant pathogens identified on urine culture in uroseptic patients are enterobacteria, with *Escherichia coli* being the most common microorganism. *Pseudomonas aeruginosa* and *Serratia* sp. are likely to be multiresistant and difficult to treat [2]. Risk factors for bacteremia are complicated UTIs, such as pyelonephritis or obstructive uropathy. Elderly and immunocompromised patients (diabetes mellitus, cancer diseases, or immunosuppressive therapy) are at higher risk to experience severe complications of genitourinary infections, for instance, perinephric (emphysematous) or renal abscesses or xanthogranulomatous pyelonephritis [5–7]. Depending on the severity of the condition, sepsis can be classified into four groups: SIRS (systemic inflammatory response syndrome), sepsis, severe sepsis, and septic shock, first defined in 1991 by the American College of Chest Physicians (ACCP) and Society of Critical Care Medicine (SCCM) [8]. SIRS is a clinical syndrome resulting from a noninfectious insult and can be diagnosed when two or more of the criteria below are present (Table 10.1).

Severe sepsis, defined by the presence of symptoms of organ dysfunction, is related to UTI in 5 % [2], causing mortality in 30–50 % [3]. Main cause of mortality is a hemodynamic collapse due to the release of pro-inflammatory cytokines, such as interleukin-1 and interleukin-6 (IL-1, IL-6), and tumor necrosis factor-alpha (TNF-α), which is activated by bacterial components. The excessive production of pro-inflammatory cytokines increases the permeability of endothelial cells leading to blood shift into the interstitial space and triggers proanticoagulant mechanisms with occurrence of multiple intravascular thrombi [3]. Endothelial damage prevents the activation of protein C, which is important for the downregulation of proanticoagulant mechanisms. As a consequence of the homeostatic imbalance toward coagulation, impaired blood flow to vital organs may lead to circulatory and organ failure. Hence, interdisciplinary management, including early transfer to intensive care unit, is essential to prevent fatal outcome.

Table 10.1 Clinical diagnostic criteria of SIRS, sepsis, and septic shock [2]

Disorder	Definition
Systemic inflammatory response syndrome (SIRS)	Temperature >38° or <36°
	Heart rate >90 bpm
	Respiratory rate >20 breaths/min or $PaCO_2$ <32 mmHg
	WBC >12,000 cells/mm^3 or <4,000 cells/mm^3
Sepsis	Activation of the inflammatory process due to infection
Severe sepsis	Organ dysfunction, hypoperfusion, or hypotension
Septic shock	Sepsis with hypoperfusion despite adequate fluid resuscitation

10.6 Symptoms, Classification, and Grading

Symptoms of urosepsis can range from fever and chills, flank pain, abdominal tenderness, tachypnea, and tachycardia to more severe symptoms such as hypothermia and confusion. Further complications are drop of blood pressure, circulatory failure, and oliguria/anuria. SIRS can evolve into sepsis and septic shock, with endothelial injury being one of the hallmarks. As a result of capillary leakage, venous pooling and peripheral vasodilatation may occur, leading to intravascular volume depletion, tissue hypoxia, and consecutive organ failure. Refractory septic shock presents the most severe complication in patients with urosepsis, with non-response to fluid and pharmacological interventions causing a significant increase of morbidity and mortality.

10.7 Therapy

The optimal approach to uroseptic patients involves three goals: early recognition, causal treatment with relief of urinary tract obstruction, and timely administration of antimicrobial agents [2]. Any delay in the initiation of adequate therapy is potentially lethal. Causal treatment requires urological interventions, such as performance of percutaneous or surgical drainage of renal abscesses, placement of percutaneous nephrostomy for infected hydronephrosis, or insertion of suprapubic catheter in bacterial prostatitis. Prostate abscesses need to be drained immediately either by perineal placement of a drain or by TURP depending on the patient's general condition. Empirical antibiotic treatment should be promptly initiated with reassessment of microbiological and clinical data to narrow antimicrobial coverage in the course of the therapy. Critically ill patients have to be provided with life-sustaining circulatory and respiratory support at the intensive care unit, where maintenance of balance between oxygen delivery and oxygen demand is crucial in the prevention of metabolic acidosis and consecutive organ failure. It should be constantly monitored by the assessment of central venous oxygenation saturation, lactate concentration, serum pH, and base excess.

Supportive and adjunctive therapy, including volume resuscitation with crystalloids and catecholamines, remain the mainstay in the strategy of septic shock and contribute to a significant reduction of mortality [2]. The administration of human recombinant activated protein C with its anticoagulant properties aims at improving sepsis-induced coagulopathy and outcome [9]. Interdisciplinary management, early goal-directed therapy, source control, continuous monitoring, management of fluid and electrolyte balance, and correction of coagulation abnormalities present the crucial therapeutic challenges of urosepsis (Fig. 10.2).

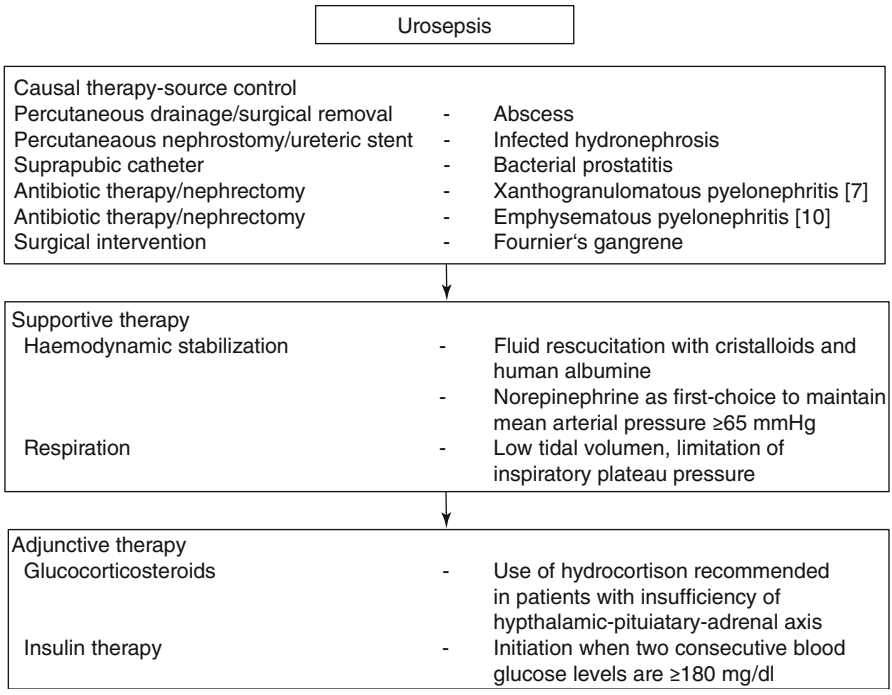

Fig. 10.2 Flow chart demonstrating the pathway from diagnosis to therapy [2, 7, 10, 11]

10.8 Complications

Fournier's gangrene, a necrotizing fasciitis of the perineal, perianal, and genital region, presents a rare but life-threatening complication of urogenital infections [2]. Since it is a rapidly progressing disorder of the fascia and subcutaneous soft tissue, mortality and morbidity rate is high [12]. Hence, urgent surgical debridement with wide excision of the necrotic tissue and the immediate administration of broad-spectrum antibiotic therapy present the only way of improving the chances for patient's survival [12].

In general, mortality rate of severe sepsis has decreased in recent years, although it is still a very serious condition. The degree of organ dysfunction and coagulopathy, microbiological etiology, and comorbidities are essential prognostic factors for an adverse outcome. As sepsis develops rapidly, with symptoms frequently imitating those of other conditions, early and accurate diagnosis and prompt therapy remain significant clinical challenges and key elements in the care of septic patients.

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