available at www.sciencedirect.com
journal homepage: www.europeanurology.com

European Association of Urology

Review – Infections

European Association of Urology Guidelines on Urological Infections: Summary of the 2024 Guidelines

Jennifer Kranz[a,b,*], *Riccardo Bartoletti*[c], *Franck Bruyère*[d,e], *Tommaso Cai*[f], *Suzanne Geerlings*[g,h], *Bela Köves*[i], *Sören Schubert*[j], *Adrian Pilatz*[k], *Rajan Veeratterapillay*[l], *Florian M E Wagenlehner*[k], *Kathrin Bausch*[m], *Wout Devlies*[n], *József Horváth*[o], *Lorenz Leitner*[p], *Guglielmo Mantica*[q], *Tunde Mezei*[r], *Emma J. Smith*[s], *Gernot Bonkat*[t]

[a] *Department of Urology and Pediatric Urology, RWTH Aachen University, Aachen, Germany;* [b] *Department of Urology and Kidney Transplantation, Martin-Luther-University, Halle, Germany;* [c] *Department of Translational Research and New Technologies, University of Pisa, Pisa, Italy;* [d] *Department of Urology, CHRU Bretonneau, Tours, France;* [e] *Université Francois Rabelais, PRES Centre Val de Loire, Tours, France;* [f] *Department of Urology, Santa Chiara, Regional Hospital, Trento, Italy;* [g] *Department of Internal Medicine, Amsterdam Institute for Infection and Immunity, Amsterdam Public Health Research Institute, Amsterdam, The Netherlands;* [h] *Amsterdam University Medical Center, Amsterdam, The Netherlands;* [i] *Department of Urology, University of Szeged, Szeged, Hungary;* [j] *Max von Pettenkofer Institute, Faculty of Medicine, LMU Munich, Munich, Germany;* [k] *Department of Urology, Pediatric Urology and Andrology, Justus-Liebig-University Giessen, Giessen, Germany;* [l] *Freeman Hospital, Newcastle Upon Tyne, UK;* [m] *Department of Urology, University Hospital Basel, University of Basel, Basel, Switzerland;* [n] *Department of Urology, UZ Leuven, Leuven, Belgium;* [o] *BKMK SZTE ÁOK Okt. Kh. Urológiai Osztálya, Kecskemét, Hungary;* [p] *Department of Neuro-Urology, Balgrist University Hospital, University of Zürich, Zürich, Switzerland;* [q] *IRCCS Ospedale Policlinico San Martino, Genova, Italy;* [r] *Department of Urology, Telemark Hospital, Skien, Norway;* [s] *European Association of Urology Guidelines Office, Arnhem, The Netherlands;* [t] *alta Uro AG, Merian Iselin Klinik, Center of Biomechanics & Calorimetry, University of Basel, Basel, Switzerland*

Article info

Article history:
Accepted March 28, 2024

Associate Editor:
Alberto Briganti

Keywords:
Urological infections
Antimicrobial stewardship
Cystitis
Pyelonephritis
Complicated urinary tract infections
Periprocedural antibiotic prophylaxis
Antibiotics
Male accessory gland infections
Urinary tract infections

Abstract

Background and objective: Urological infections significantly impact the wellbeing and quality of life of individuals owing to their widespread occurrence and diverse clinical manifestations. The objective of the guidelines panel was to provide evidence-based guidance on the diagnosis, treatment, and prevention of urinary tract infections (UTIs) and male accessory-gland infections, while addressing crucial public health aspects related to infection control and antimicrobial stewardship.

Methods: For the 2024 guidelines on urological infections, new and relevant evidence was identified, collated, and appraised via a structured assessment of the literature. Databases searched included Medline, EMBASE, and the Cochrane Libraries. Recommendations within the guidelines were developed by the panel to prioritise clinically important care decisions. The strength of each recommendation was determined according to a balance between desirable and undesirable consequences of alternative management strategies, the quality of the evidence (including the certainty of estimates), and the nature and variability of patient values and preferences.

Key findings and limitations: Key recommendations emphasise the importance of a thorough medical history and physical examination for patients with urological infections. The guidelines stress the role of antimicrobial stewardship to combat the rising threat of antimicrobial resistance, providing recommendations for antibiotic selection, dosing, and duration on the basis of the latest evidence.

* Corresponding author. Department of Urology and Pediatric Urology, RWTH Aachen University, Pauwelsstrasse 30, 52074 Aachen, Germany. Tel. +49 241 8089374.
E-mail address: jkranz@ukaachen.de (J. Kranz).

https://doi.org/10.1016/j.eururo.2024.03.035

Please cite this article as: J. Kranz, R. Bartoletti, F. Bruyère et al., European Association of Urology Guidelines on Urological Infections: Summary of the 2024 Guidelines, Eur Urol (2024), https://doi.org/10.1016/j.eururo.2024.03.035

Conclusions and clinical implications: This overview of the 2024 EAU guidelines offers valuable insights into managing urological infections and are designed for effective integration into clinical practice.
Patient summary: The European Association of Urology has issued an updated guideline on urological infections. The guidelines provide recommendations for diagnosis, treatment, and prevention, with a particular focus on minimising antibiotic use because of the increasing global threat of antimicrobial resistance.

1. Introduction

Urinary tract infections (UTIs) are among the most frequent bacterial infections, with various medical specialties involved in their diagnosis, treatment, and prevention [1]. Clinical presentations of UTIs vary widely, from rather benign, uncomplicated infections to severe conditions such as complicated UTIs, pyelonephritis, and even urosepsis [2,3]. While the overall incidence of sepsis is decreasing, there has been a notable rise in severe UTIs. Antimicrobial resistance has emerged as a significant global health concern, particularly for complicated UTIs including pyelonephritis, with antibiotic resistance increasingly encountered in daily clinical practice, which poses challenges for effective treatment [4].

The European Association of Urology (EAU) urological infections guidelines panel has compiled these clinical guidelines to equip health care professionals with evidence-based insights and recommendations for the diagnosis, treatment, and prevention of UTIs and male accessory-gland infections. The guidelines also address crucial public health aspects such as infection control and antimicrobial stewardship.

It must be emphasised that clinical guidelines present the best evidence available to experts. However, following guideline recommendations will not necessarily result in the best outcome. Guidelines are not a substitute for clinical expertise in tailoring treatment decisions for individual patients; instead, they serve as a framework for decisions, which should also take into consideration patients' personal values, preferences, and unique circumstances. The EAU urological infections guidelines panel is a multidisciplinary group of urologists with expertise in this area, an infectious disease specialist, and a clinical microbiologist. All contributors to this document have disclosed potential conflicts of interest, accessible for review on the EAU website (http://uroweb.org/guideline/urological-infections/).

2. Methods

For the 2024 urological infections guidelines, new evidence was identified, collated, and appraised via a structured assessment of the literature. Databases searched included Medline, EMBASE, and the Cochrane Libraries. Detailed search strategies are available on the EAU website (https://uroweb.org/guidelines/urological-infections/publications-appendices). Recommendations in the guidelines were developed by the panel to prioritise clinically important care decisions. The strength of each recommendation was determined according to a balance between desirable and undesirable consequences of alternative management strategies, the quality of the evidence [5] (including certainty of estimates), and the nature and variability of patient values and preferences. Recommendations are rated as strong when the evidence quality is high and/or there is a favourable balance of benefits to harms and patient preferences. Recommendations are rated as weak when the evidence is of lower quality and/or benefits and patient preferences are less clear.

3. Guidelines

3.1. *UTI classification*

The clinical spectrum of UTIs is heterogeneous and ranges from benign to life-threatening infections [2,6–9], with important variations in both diagnosis and treatment. Hence, patient stratification is crucial. There are several classification systems for describing and classifying UTIs, with the common rationale that the risks of recurrence, progression, chronic status, and severe outcomes are worse for complicated than for uncomplicated UTIs [2] (Fig. 1).

3.2. *Asymptomatic bacteriuria in adults*

Asymptomatic bacteriuria (ABU) is common and corresponds to commensal colonisation [10]. In an individual without urinary tract symptoms, ABU is defined as a midstream sample of urine showing bacterial growth $>10^5$ cfu/ml in two consecutive samples in women [11] and in a single sample in men [12]. The spectrum of bacteria in ABU is similar to species found in uncomplicated or complicated UTIs, depending on the presence of risk factors . Cystoscopy and/or imaging of the upper urinary tract is not mandatory if the medical history is otherwise unremarkable. If persistent growth of urease-producing bacteria (*Proteus mirabilis*) is detected, stone formation in the urinary tract must be excluded [13]. For men, digital rectal examination should be performed to investigate the possibility of prostate diseases. Clinical studies have shown that ABU may protect against superinfecting symptomatic UTI, and thus ABU should only be treated in cases of proven benefit for the patient to avoid the risk of selecting antimicrobial resistance and eradicating a potentially protective ABU strain [14,15] (Table 1). A meta-analysis of the available evidence revealed that treatment of ABU in pregnant women was beneficial; however, most studies have low method-

Please cite this article as: J. Kranz, R. Bartoletti, F. Bruyère et al., European Association of Urology Guidelines on Urological Infections: Summary of the 2024 Guidelines, Eur Urol (2024), https://doi.org/10.1016/j.eururo.2024.03.035

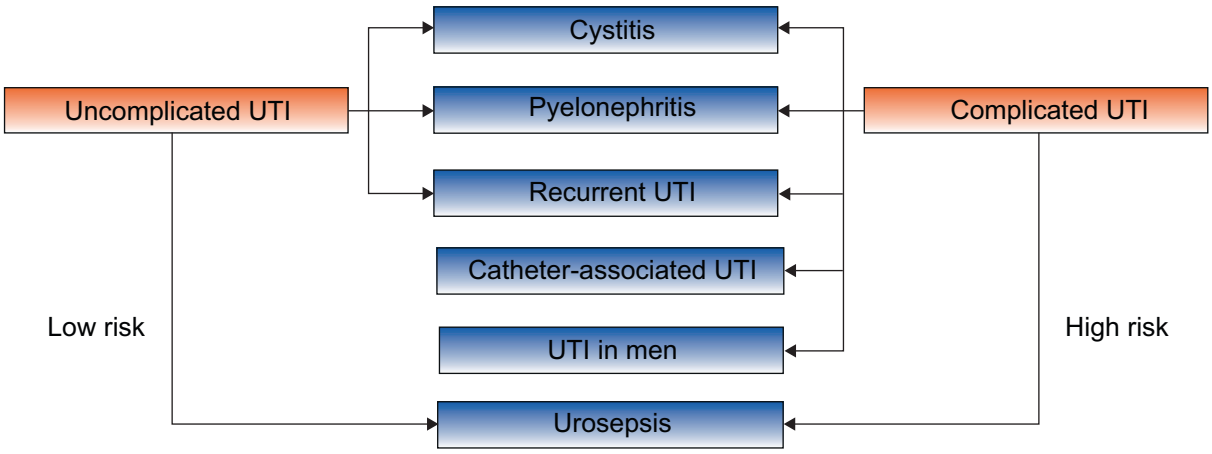

Fig. 1 – Concept of uncomplicated and complicated urinary tract infection (UTI).

Table 1 – Recommendations for the management of asymptomatic bacteriuria

Recommendation	Strength rating
Do not screen or treat asymptomatic bacteriuria in the following conditions: • Women without risk factors • Patients with well-regulated diabetes mellitus • Postmenopausal women • Elderly institutionalised patients • Patients with a dysfunctional and/or reconstructed lower urinary tract • Patients with a renal transplant • Patients before arthroplasty surgery • Patients with recurrent urinary tract infections	Strong
Do not screen or treat asymptomatic bacteriuria in patients before cardiovascular surgeries.	Weak
Screen for and treat asymptomatic bacteriuria before urological procedures breaching the mucosa.	Strong
Screen for and treat asymptomatic bacteriuria in pregnant women with standard short-course treatment or single-dose fosfomycin trometamol.	Weak

ological quality and are from the 1960s to 1980s. Diagnostic and treatment protocols and access to medical services have dramatically changed since then; therefore, the quality of evidence for this recommendation is low. In a newer study of higher methodological quality, the beneficial effects of antibiotic treatment are not as evident [16].

3.3. *Uncomplicated cystitis*

Uncomplicated cystitis is defined as acute, sporadic, or recurrent cystitis limited to nonpregnant women with no known relevant anatomic or functional abnormalities in the urinary tract and no comorbidities. Risk factors include sexual intercourse, use of spermicide, a new sexual partner, a mother with a history of UTI, and a history of UTI during childhood. Most cases of uncomplicated cystitis are caused by *Escherichia coli*. Diagnosis of uncomplicated cystitis can be made with a high probability on the basis of a focused history of lower urinary tract symptoms (dysuria, frequency, and urgency) and the absence of vaginal discharge [17,18]. In elderly women, genitourinary symptoms are not necessarily related to cystitis [19,20]. In patients presenting with typical symptoms of uncomplicated cystitis, urine analysis (urine culture, dipstick testing) leads to only a minimal increase in diagnostic accuracy [21]. However, if the diagnosis is unclear, dipstick analysis can increase the likelihood of a diagnosis of uncomplicated cystitis [22,23]. A urine culture is recommended in the following situations: suspected acute pyelonephritis; symptoms that do not resolve or recur within 4 wk after completion of treatment; women who present with atypical symptoms; and/or pregnant women.

For females with mild to moderate symptoms, symptomatic therapy (eg, ibuprofen) may be considered as an alternative to antimicrobial treatment in consultation with individual patients [24–27]. The choice of antimicrobial therapy should be guided by the spectrum and susceptibility patterns of the aetiological pathogens; efficacy for the particular indication in clinical studies; tolerability and adverse reactions; adverse ecological effects; and costs and/or availability [17]. Suggested regimens for antimicrobial therapy for uncomplicated cystitis are listed in Table 2.

Routine post-treatment urinalysis or urine cultures are not indicated for asymptomatic patients [6]. For women whose symptoms do not resolve by the end of treatment, and for those whose symptoms resolve but recur within 2 wk, a urine culture and antimicrobial susceptibility testing should be performed [28]. For therapy in this situation, it should be assumed that the infecting organism is not susceptible to the agent originally used. Retreatment with a 7-d regimen using another agent should be considered [28].

3.4. *Recurrent UTIs*

Recurrent UTIs (rUTIs) are recurrences of uncomplicated and/or complicated UTIs, with a frequency of at least three UTIs/yr or two UTIs in the last 6 mo. Although rUTIs include both lower tract infection (cystitis) and upper tract infection (pyelonephritis), repeated pyelonephritis should prompt consideration of a complicated aetiology. rUTIs negatively impact a patient's quality of life, with reductions in the quality of social and sexual relationships, self-esteem, and capacity for work [29]. Risk factors for rUTI are outlined in Table 3.

Please cite this article as: J. Kranz, R. Bartoletti, F. Bruyère et al., European Association of Urology Guidelines on Urological Infections: Summary of the 2024 Guidelines, Eur Urol (2024), https://doi.org/10.1016/j.eururo.2024.03.035

Table 2 – Suggested antimicrobial therapy regimens for uncomplicated cystitis

Antimicrobial	Daily dose	Therapyduration	Comments
First-line treatment in women			
Fosfomycin trometamol	3 g SD	1 d	Recommended only in women with uncomplicated cystitis.
Nitrofurantoin macrocrystals	50–100 mg q.i.d.	5 d	
Nitrofurantoin monohydrate or macrocrystals	100 mg b.i.d.	5 d	
Nitrofurantoin macrocrystals prolonged release	100 mg b.i.d.	5 d	
Pivmecillinam	400 mg t.i.d.	3–5 d	
Alternatives			
Cephalosporins (eg, cefadroxil)	500 mg b.i.d.	3 d	Or comparable
If the local resistance pattern for *Escherichia coli* is <20%			
Trimethoprim	200 mg b.i.d.	5 d	Not in the first trimester of pregnancy
Trimethoprim-sulfamethoxazole	160/800 mg b.i.d.	3 d	Not in the last trimester of pregnancy
Treatment in men			
Trimethoprim-sulfamethoxazole	160/800 mg b.i.d.	7 d	Restricted to men, fluoroquinolones can also be prescribed in accordance with local susceptibility testing.

SD = single dose; b.i.d. = twice daily; q.i.d. = four times daily; t.i.d = three times daily.

Table 4 – Recommendations for the diagnostic evaluation and treatment of recurrent UTIs

Recommendation	Strength rating
Diagnose recurrent UTI via a urine culture.	Strong
Do not perform an extensive routine workup (eg, cystoscopy, full abdominal ultrasound) in women younger than 40 yr with recurrent UTI and no risk factors.	Weak
Advise premenopausal women to increase their fluid intake, as this might reduce the risk of recurrent UTI.	Weak
Use vaginal oestrogen replacement in postmenopausal women to prevent recurrent UTI.	Strong
Use immunoactive prophylaxis to reduce recurrent UTI in all age groups.	Strong
Advise patients on the use of local or oral probiotic-containing strains of proven efficacy for vaginal flora regeneration to prevent UTIs.	Weak
Advise patients on the use of cranberry products to reduce recurrent UTI episodes; however, patients should be informed that the quality of evidence underpinning this is low, with contradictory findings.	Weak
Use D-mannose to reduce recurrent UTI episodes, but patients should be informed of the overall weak and contradictory evidence regarding its effectiveness.	Weak
Use methenamine hippurate to reduce recurrent UTI episodes in women without abnormalities of the urinary tract.	Strong
Use endovesical instillations of hyaluronic acid or a combination of hyaluronic acid and chondroitin sulfate to prevent recurrent UTIs in patients for whom less invasive preventive approaches have been unsuccessful. Patients should be informed that further studies are needed to confirm the results of initial trials.	Weak
Use continuous or postcoital antimicrobial prophylaxis to prevent recurrent UTI when non-antimicrobial interventions have failed. Counsel patients regarding possible side effects.	Strong
For patients with good compliance, self-administered short-term antimicrobial therapy should be considered.	Strong

UTI = urinary tract infection.

Table 3 – Age-related factors associated with recurrent UTI in women

Young and pre-menopausal women	Post-menopausal and elderly women
Sexual intercourse	History of UTI before menopause
Use of spermicide	Urinary incontinence
A new sexual partner	Atrophic vaginitis due to oestrogen deficiency
A mother with a history of UTI	Cystocele
History of UTI during childhood	High postvoid residual urine volume
Blood group antigen secretory status	Blood group antigen secretory status
	Urine catheterisation and functional status
	deterioration in elderly institutionalised women

UTI = urinary tract infection.

rUTI prevention includes counselling regarding avoidance of risk factors, non-antimicrobial measures, and antimicrobial prophylaxis [28,30] (Table 4). These interventions should be attempted in the order listed.

3.5. *Uncomplicated pyelonephritis*

Uncomplicated pyelonephritis is defined as pyelonephritis limited to nonpregnant, premenopausal women with no

known relevant urological abnormalities or comorbidities. It typically presents with fever (>38 °C), chills, flank pain, nausea, vomiting, or tenderness at the costovertebral angle, with or without symptoms of cystitis [31].

Urinalysis, including assessment of white and red blood cells and nitrite, is recommended for routine diagnosis [32]. In addition, a urine culture and antimicrobial susceptibility testing should be performed in all cases of pyelonephritis. Evaluation of the upper urinary tract via ultrasound should be performed to rule out urinary tract obstruction or renal stone disease in patients with a history of urolithiasis, renal function disturbances or a high urine pH [33]. Additional investigations, such as a contrast-enhanced computed tomography scan, or excretory urography, should be considered if the patient remains febrile after 72 h of treatment, or immediately if there is a deterioration in clinical status [33]. For diagnosis of complicating factors in pregnant women, ultrasound or magnetic resonance imaging (MRI) should be used preferentially to avoid radiation risk to the foetus [33].

Prompt differentiation between uncomplicated and potentially obstructive pyelonephritis is crucial, as the latter can swiftly progress to urosepsis. This delineation should be established promptly using appropriate imaging techniques.

Fluoroquinolones and cephalosporins are the only antimicrobial agents that can be recommended for oral empiric treatment of uncomplicated pyelonephritis [34]; oral cephalosporins achieve significantly lower blood and urinary concentrations than the intravenous route (Table 5). Other agents such as nitrofurantoin, oral fosfomycin, and pivmecillinam should be avoided as there are insufficient data regarding their efficacy [35]. It has been shown that a short outpatient course of antibiotic treatment for acute pyelonephritis is equivalent to longer therapy durations in terms of clinical and microbiological success. However, this approach is associated with a higher recurrence rate within 4–6 wk and needs to be tailored to local policies and resistance patterns [36].

Patients with uncomplicated pyelonephritis requiring hospitalisation should be treated initially with an intravenous antimicrobial regimen, such as a fluoroquinolone, an aminoglycoside (with or without ampicillin), or an extended-spectrum cephalosporin or penicillin [37] (Table 6). Carbapenems and novel broad-spectrum antimicrobial agents should only be considered in patients with early culture results indicating the presence of multidrug-resistant organisms. The choice between these agents should be based on local resistance patterns and optimised on the basis of drug susceptibility results. For patients presenting with signs of urosepsis, empiric antimicrobial coverage for extended-spectrum β-lactamase–producing organisms is warranted [38]. Patients initially treated with parenteral therapy who improve clinically and can tolerate oral fluids may transition to oral antimicrobial therapy [39].

For pregnant women with pyelonephritis, outpatient management with appropriate parenteral antimicrobials

Table 6 – Suggested regimens for empirical parenteral antimicrobial therapy for uncomplicated pyelonephritis

Antimicrobial	Daily dose	Comments
First-line treatment		
Ciprofloxacin	400 mg b.i.d.	
Levofloxacin	750 mg q.d.	
Cefotaxime	2 g t.i.d.	Not studied as monotherapy in acute uncomplicated pyelonephritis.
Ceftriaxone	1–2 g q.d.	Lower dose studied, but higher dose recommended.
Second-line treatment		
Cefepime	1–2 g b.i.d.	Lower dose studied, but higher dose recommended.
Piperacillin/tazobactam	2.5–4.5 g t.i.d.	
Gentamicin	5 mg/kg q.d.	Not studied as monotherapy in acute uncomplicated pyelonephritis.
Amikacin	15 mg/kg q.d.	
Last-line alternatives		
Imipenem/cilastatin	0.5 g t.i.d.	Consider only in patients with early culture results indicating the presence of multidrug-resistant organisms.
Meropenem	1 g t.i.d.	
Ceftolozane/tazobactam	1.5 g t.i.d.	
Ceftazidime/avibactam	2.5 g t.i.d.	
Cefiderocol	2g t.i.d.	
Meropenem-vaborbactam	2g t.i.d.	
Plazomicin	15mg/kg o.d.	

b.i.d. = twice daily; t.i.d. = three times daily; q.d. = every day; o.d. = once daily.

Table 5 – Suggested regimens for empirical oral antimicrobial therapy for uncomplicated pyelonephritis

Antimicrobial	Daily dose	Therapyduration	Comments
Ciprofloxacin	500–750 mg b.i.d	7 d	Fluoroquinolone resistance should be <10%.
Levofloxacin	750 mg q.d.	5 d	
Trimethoprim sulfamethoxazole	160/800 mg b.i.d	14 d	If such agents are used empirically, an initial intravenous dose of a long-acting parenteral antimicrobial (eg, ceftriaxone) should be administered.
Cefpodoxime	200 mg b.i.d	10 d	
Ceftibuten	400 mg q.d	10 d	

b.i.d. = twice daily; q.d. = every day.

Please cite this article as: J. Kranz, R. Bartoletti, F. Bruyère et al., European Association of Urology Guidelines on Urological Infections: Summary of the 2024 Guidelines, Eur Urol (2024), https://doi.org/10.1016/j.eururo.2024.03.035

Table 7 – Common factors associated with complicated urinary tract infections [46–49]

Obstruction at any site in the urinary tract	Urinary tract infection in males
Foreign body	Pregnancy
Incomplete voiding	Diabetes mellitus
Vesicoureteral reflux	Immunosuppression
Recent history of instrumentation	Health care–associated infections
ESBL-producing organisms isolated	Multidrug-resistant organisms isolated

ESBL = extended-spectrum β-lactamase.

may also be considered, provided symptoms are mild and close follow-up is feasible [40,41]. For more severe cases of pyelonephritis, hospitalisation and supportive care are usually required. After clinical improvement, parenteral therapy can also be switched to oral therapy for a total treatment duration of 7–10 d. For men with febrile UTI, pyelonephritis, or recurrent infection, or whenever a complicating factor is suspected, a minimum treatment duration of 2 wk is recommended, preferably with a fluoroquinolone, as prostatic involvement is frequent [42].

3.6. Complicated UTIs

A complicated UTI (cUTI) occurs when an individual has host-related factors or specific anatomic or functional abnormalities in the urinary tract that are believed to make the infection more challenging to eradicate in comparison to an uncomplicated infection [43–45]. Recent insights into cUTI management highlight the importance of considering infections caused by multidrug-resistant uropathogens [46]. Table 7 outlines underlying factors commonly associated with cUTIs. A cUTI designation encompasses a wide range of underlying conditions, resulting in a diverse patient population. Therefore, it is evident that a one-size-fits-all approach to cUTI evaluation and treatment is inadequate. However, there are general management principles that can be applied to most patients with cUTIs. The following recommendations are based on the Stichting Werkgroep Antibioticabeleid (SWAB) guidelines from the Dutch Working Party on Antibiotic Policy [47] (Table 8).

Table 8 – Recommendations for the treatment of complicated UTIs

Recommendation	Strength rating
Use a combination of: • Amoxicillin plus an aminoglycoside • A second-generation cephalosporin plus an aminoglycoside • An intravenous third-generation cephalosporin as empirical treatment for complicated UTI with systemic symptoms	Strong
Only use ciprofloxacin if the local resistance rate is <10% when: • The entire treatment is given orally • The patient does not require hospitalisation • The patient has anaphylaxis to β-lactam antimicrobials	Strong
Do not use ciprofloxacin and other fluoroquinolones for empirical treatment of complicated UTI in patients from urology departments or when patients have used fluoroquinolones in the last 6 mo.	Strong
Manage any urological abnormality and/or underlying complicating factors.	Strong

UTI = urinary tract infection.

cUTI is associated with typical clinical symptoms (eg, dysuria, urgency, frequency, flank pain, costovertebral angle tenderness, suprapubic pain, and fever), although the symptoms may be atypical in some clinical situations, such as in neuropathic bladder disturbances, catheter-associated UTI (CA-UTI), and previous radical cystectomy with urinary diversion. Clinical presentation can vary from severe obstructive acute pyelonephritis with imminent urosepsis to a postoperative CA-UTI. A urine culture is recommended. The microbial spectrum is greater than for uncomplicated UTIs, and antimicrobial resistance is more likely [48,49]. *E. coli*, *Proteus* spp., *Klebsiella* spp., *Pseudomonas* spp., *Serratia* spp., and *Enterococcus* spp. are the most common species found in cultures.

Appropriate management of the urological abnormality or the underlying complicating factor is mandatory. Optimal antimicrobial therapy for cUTI depends on the severity of the illness at presentation, as well as local resistance patterns and specific host factors (such as allergies). In addition, a urine culture and susceptibility testing should be performed, and initial empiric therapy should be tailored and followed by (oral) administration of an appropriate antimicrobial agent for the uropathogen isolated.

Treatment for 7 d [50] to 14 d (14 d for men when prostatitis cannot be excluded) [51], is generally recommended, but the duration should be closely related to the treatment of the underlying abnormality. When the patient is haemodynamically stable and has been afebrile for at least 48 h, a shorter treatment duration (eg, 7 d) may be considered in cases for which short-course treatment is desirable owing to relative contraindications to the antibiotic administered [52].

3.7. Catheter-associated UTIs

Catheter-associated (CA)-UTI refers to UTI occurring in an individual whose urinary tract is currently catheterised or has been catheterised within the past 48 h. CA-UTIs are the leading cause of secondary health care–associated bacteraemia. Approximately 20% of hospital-acquired bacteraemias arise from the urinary tract, and the mortality associated with this condition is approximately 10% [53]. The incidence of bacteriuria associated with indwelling catheterisation is 3–8% per day [54–58]. Catheterisation duration is the most important risk factor for CA-UTI development [59,60]. A systematic review and meta-analysis demonstrated that patients at high risk of CA-UTI were female, had prolonged catheterisation duration, had diabetes, and had longer hospital and intensive care unit stays [61]. Signs and systemic symptoms compatible with CA-UTI include new onset or worsening of fever, rigor, altered mental status, malaise, or lethargy with no other identified cause, flank pain, costovertebral angle tenderness, acute haematuria, and pelvic discomfort, as well as dysuria, urgent or frequent urination, and suprapubic pain or tenderness in those whose catheter has been removed [47]. For catheterised patients, the presence or absence of odorous or cloudy urine alone should not be used to differentiate

Please cite this article as: J. Kranz, R. Bartoletti, F. Bruyère et al., European Association of Urology Guidelines on Urological Infections: Summary of the 2024 Guidelines, Eur Urol (2024), https://doi.org/10.1016/j.eururo.2024.03.035

Table 9 – Recommendations for CA-UTI management and prevention

Recommendation	Strength rating
Treat symptomatic CA-UTI according to the recommendations for complicated UTI (Table 8).	Strong
Take a urine culture before initiating antimicrobial therapy in catheterised patients whose catheter has been removed.	Strong
Do not treat catheter-associated asymptomatic bacteriuria in general.	Strong
Treat catheter-associated asymptomatic bacteriuria before traumatic urinary tract interventions (eg, transurethral resection of the prostate).	Strong
Replace or remove the indwelling catheter before starting antimicrobial therapy.	Strong
Do not apply topical antiseptics or antimicrobials to the catheter, urethra, or meatus.	Strong
Do not use prophylactic antimicrobials to prevent CA-UTI.	Strong
Do not routinely use antibiotic prophylaxis to prevent clinical UTI after urethral catheter removal.	Weak
The duration of catheterisation should be minimal.	Strong
Use hydrophilic coated catheters to reduce CA-UTI.	Strong
Do not routinely use antibiotic prophylaxis to prevent clinical UTI after urethral catheter removal or in patients performing intermittent self-catheterisation.	Weak

CA-UTI = catheter-associated urinary tract infection.

CA-ABU from CA-UTI [47,48]. Microbiologically, CA-UTI is defined by microbial growth of $>10^3$ cfu/ml of one or more bacterial species in a single catheter urine specimen or in a midstream voided urine specimen from a patient whose urethral, suprapubic, or condom catheter has been removed within the previous 48 h [48]. For catheterised patients, pyuria is not diagnostic for CA-UTI. The presence, absence, or degree of pyuria should not be used to differentiate CA-ABU from CA-UTI. Pyuria accompanying CA-ABU should not be interpreted as an indication for antimicrobial treatment. The absence of pyuria in a symptomatic patient suggests a diagnosis other than CA-UTI [48].

Recommendations for CA-UTI management and prevention are outlined in Table 9.

3.8. *Urosepsis*

Sepsis is defined as life-threatening organ dysfunction resulting from a dysregulated host response to infection. Clinically, organ dysfunction can be indicated by an increase in Sequential (Sepsis-related) Organ Failure Assessment (SOFA) score of $\geq$2 points. A quick SOFA (qSOFA) score has been developed for rapid identification: a respiratory rate of $\geq$22 breaths/min, altered mental status, or systolic blood pressure of $\leq$100 mm Hg.

Table 10 – Recommendations for the diagnosis and treatment of urosepsis

Recommendation	Strength rating
Assess the quick SOFA score to identify patients with potential sepsis.	Strong
Take a urine culture and two sets of blood cultures before starting antimicrobial treatment.	Strong
Administer parenteral high-dose broad-spectrum antimicrobials within the first hour after clinical assumption of sepsis.	Strong
Adapt the initial empiric antimicrobial therapy on the basis of culture results.	Strong
Initiate source control, including removal of foreign bodies, decompression of obstruction, and drainage of abscesses in the urinary tract.	Strong
Provide immediate adequate life-support measures.	Strong

SOFA = Sequential (Sepsis-related) Organ Failure Assessment.

Table 11 – Suggested antimicrobial therapy regimens for urosepsis

Antimicrobial	Daily dose	Therapy duration
Cefotaxime	2 g t.i.d.	7–10 d
Ceftazidime	1–2 g t.i.d.	Longer courses are appropriate in patients with a slow clinical response
Ceftriaxone	1–2 g q.d.	
Cefepime	2 g b.i.d.	
Piperacillin/tazobactam	4.5 g t.i.d.	
Ceftolozane/tazobactam	1.5 g t.i.d.	
Ceftazidime/avibactam	2.5 g t.i.d.	
Gentamicin[a]	5 mg/kg q.d.	
Amikacin[a]	15 mg/kg q.d.	
Ertapenem	1 g q.d.	
Imipenem/cilastatin	0.5 g t.i.d.	
Meropenem	1 g t.i.d.	

b.i.d. = twice daily; t.i.d. = three times daily; q.d. = every day.
[a] Not studied as monotherapy in urosepsis.

For diagnosis of systemic symptoms in sepsis, either the full SOFA or qSOFA score should be assessed.

Microbiological sampling should encompass urine, two sets of blood cultures [62], and, when applicable, drainage fluids. Early imaging investigations, including sonography and computed tomography scans, should be conducted [63]. Management of urosepsis necessitates comprehensive life-supportive measures, timely and suitable antimicrobial therapy, adjunctive interventions, and effective management of urinary tract abnormalities [64] (Tables 10 and 11). It is crucial to establish source control by alleviating any obstruction and draining significant abscesses within the urinary tract [64]. Collaborative treatment involving urologists, intensive care, and infectious disease specialists is recommended for optimal patient care.

3.9. *Urethritis*

Urethritis can arise from infectious or noninfectious causes. It is important to differentiate urethral inflammation from other lower urinary tract infections. Urethral infections are commonly transmitted via sexual contact. It is crucial to differentiate between gonococcal urethritis (GU) and non-gonococcal urethritis (NGU). NGU is a nonspecific diagnosis with various infectious aetiologies, including *Chlamydia trachomatis*, *Mycoplasma genitalium*, *Ureaplasma urealyticum*, and *Trichomonas vaginalis*. The role of *Ureaplasma* spp. in causing urethritis is debated, with recent data suggesting that *U. urealyticum*, but not *U. parvum*, is an aetiological agent in NGU [65]. Symptoms of urethritis include mucopurulent or purulent discharge, dysuria, and urethral pruritus, although many urethral infections are asymptomatic.

In cases of severe urethritis, empiric treatment should commence on diagnosis (Tables 12 and 13). However, if the patient's symptoms are mild, it is advisable to delay

Please cite this article as: J. Kranz, R. Bartoletti, F. Bruyère et al., European Association of Urology Guidelines on Urological Infections: Summary of the 2024 Guidelines, Eur Urol (2024), https://doi.org/10.1016/j.eururo.2024.03.035

Table 12 – Recommendations for diagnostic evaluation and antimicrobial treatment of urethritis

Recommendation	Strength rating
Perform a Gram stain of urethral discharge or a urethral smear for a preliminary diagnosis of gonococcal urethritis.	Strong
Perform a validated NAAT on a first-void urine sample or urethral smear before empirical treatment to diagnose chlamydial and gonococcal infections.	Strong
Delay treatment until NAAT results are available to guide treatment choice in patients with mild symptoms.	Strong
Perform a urethral swab culture before initiation of treatment in patients with a positive NAAT for gonorrhoea to assess the antimicrobial resistance profile of the infective strain.	Strong
Use a pathogen-directed treatment based on local resistance data.	Strong
Sexual partners should be treated while maintaining patient confidentiality.	Strong

NAAT = nucleic acid amplification test.

treatment until guided by the results of the nucleic acid amplification tests. It is crucial to evaluate and treat all at-risk sexual partners while upholding patient confidentiality [66,67].

3.10. *Bacterial prostatitis*

Prostatitis is a frequent diagnosis, yet fewer than 10% of cases are confirmed to have bacterial infection . Enterobacterales are the primary pathogens in acute bacterial prostatitis (ABP) [69]. Chronic bacterial prostatitis (CBP) encompasses a broader spectrum of species, which may include atypical microorganisms [70]. Urologists are advised to use the classification proposed by the National Institute of Diabetes, Digestive, and Kidney Diseases, which distinguishes bacterial prostatitis, with confirmed or suspected infection, from chronic pelvic pain syndrome [71–73]. Acute bacterial prostatitis usually presents abruptly with voiding symptoms and distressing but poorly localised pain. It is often associated with malaise and fever. Chronic bacterial prostatitis is defined by symptoms that persist for at least 3 mo [74–76]. The predominant symptoms are pain at various locations including the perineum, scrotum, penis, and inner part of the leg, as well as lower urinary tract symptoms [71–73]. Table 14 outlines recommendations for the diagnosis of bacterial prostatitis.

In ABP, parenteral administration of high doses of bactericidal antimicrobials, such as broad-spectrum penicillins, a third-generation cephalosporin, or a fluoroquinolone, is recommended [77]. For initial therapy, any of these antimicrobials may be combined with an aminoglycoside [69,74–87]. After normalisation of infection parameters, oral therapy can be substituted and continued for a total of 2–4 wk [88].

Fluoroquinolones are recommended as first-line agents in the empiric treatment of CBP because of their favourable pharmacokinetic properties [89], generally good safety profile, and antibacterial activity against Gram-negative pathogens including *Pseudomonas aeruginosa* and *C. trachomatis* [90,91] (Table 15).

Approximately 10% of men with ABP will experience urinary retention [92], which can be managed by urethral or suprapubic catheterisation. However, recent evidence suggests that suprapubic catheterisation can reduce the risk

Table 13 – Suggested antimicrobial therapy regimens for urethritis

Pathogen	Antimicrobial	Dosage and therapy duration	Alternative regimens
Gonococcal infection	Ceftriaxone Azithromycin	1 g i.m. or i.v.[a] SD 1 g p.o. SD	• Cefixime 400 mg p.o. SD plus azithromycin 1 g p.o. SD • In cases of cephalosporin allergy: • Gentamicin 240 mg i.m. SD plus azithromycin 2 g p.o. SD • Gemifloxacin 320 mg p.o. SD plus azithromycin 2 g p.o. SD • Spectinomycin 2 g i.m. SD • Fosfomycin trometamol 3 g p.o. on days 1, 3, and 5In cases of azithromycin allergy, in combination with ceftriaxone or cefixime: • Doxycycline 100 mg b.i.d, p.o. 7 d
Non-gonococcal infection (unidentified pathogen)	Doxycycline	100 mg b.i.d, p.o. 7 d	Azithromycin 500 mg p.o. on day 1 250 mg p.o. for 4 d
Chlamydia trachomatis	Azithromycin or Doxycycline	1.0–1.5 g p.o. SD 100 mg b.i.d. p.o. for 7 d	• Levofloxacin 500 mg p.o. q.d. 7 d • Ofloxacin 200 mg p.o. b.i.d. 7 d
Mycoplasma genitalium	Azithromycin	500 mg p.o. on day 1 250 mg p.o. for 4 d	In cases of macrolide resistance: • Moxifloxacin 400 mg q.d. 7–14 d
Ureaplasma urealyticum	Doxycycline	100 mg b.i.d, p.o. 7 d	Azithromycin 1.0–1.5 g p.o. SD
Trichomonas vaginalis	MetronidazoleTinidazole	2 g p.o. SD 2 g p.o. SD	Metronidazole 500 mg p.o. b.i.d. 7 d
Persistent non-gonococcal urethritis			
After first-line doxycycline	Azithromycin plus Metronidazole	500 mg p.o. on day 1 250 mg p.o. for 4 d 400 mg b.i.d. p.o. 5 d	If macrolide-resistant *M. genitalium* is detected, moxifloxacin should be substituted for azithromycin
After first-line azithromycin	Moxifloxacin plus Metronidazole	400 mg p.o. q.d. 7–14 d 400 mg b.i.d. p.o. 5 d	

SD = single dose; b.i.d. = twice daily; q.d. = every day; p.o. = oral; i.m. = intramuscular; i.v. = intravenous.
[a] Despite a lack of randomised controlled trials there is increasing evidence that i.v. ceftriaxone is safe and effective for the treatment of gonorrhoeal infections and avoids the discomfort of an i.m. injection for patients [68].

Table 14 – Recommendations for the diagnosis of bacterial prostatitis

Recommendation	Strength rating
Do not perform prostatic massage in ABP.	Strong
Take a midstream urine dipstick to check nitrite and leukocytes in patients with clinical suspicion of ABP.	Weak
Take a midstream urine culture in patients with ABP symptoms to guide diagnosis and tailor antibiotic treatment.	Weak
Take a blood culture and a total blood count in patients presenting with ABP.	Weak
Perform accurate microbiological evaluation for atypical pathogens such as *Chlamydia trachomatis* and *Mycoplasma* species in patients with CBP.	Weak
Perform the Meares and Stamey 2- or 4-glass test in patients with CBP.	Strong
Perform transrectal ultrasound in selected cases to rule out the presence of prostatic abscess.	Weak
Do not routinely perform microbiological analysis of the ejaculate alone to diagnose CBP.	Weak

ABP = acute bacterial prostatitis; CBP = chronic bacterial prostatitis.

Table 15 – Suggested antimicrobial therapy regimens for chronic bacterial prostatitis

Antimicrobial	Daily dose	Therapy duration	Comments
Fluoroquinolone	Optimal oral daily dose	4–6 wk	
Doxycycline	100 mg b.i.d	10 d	Only for *C. trachomatis* or *Mycoplasma* infections
Azithromycin	500 mg once daily	3 wk	Only for *C. trachomatis* infections
Metronidazole	500 mg t.i.d.	14 d	Only for *T. vaginalis* infections

b.i.d. = twice daily; t.i.d. = three times daily.

of development of CBP [93]. In cases of prostatic abscess, both drainage and conservative treatment strategies appear feasible [94]; if the abscess cavity is <1 cm in diameter, conservative treatment might be effective, while larger abscesses are better treated by single aspiration or continuous drainage [95].

3.11. Acute infective epididymitis

Epididymitis is a prevalent condition that can manifest as acute, chronic, or recurrent episodes [96]. Acute epididymitis is clinically characterised by pain, swelling, and elevated temperature of the epididymis, potentially involving the testis and scrotal skin. In up to 90% of cases, the condition is caused by migration of pathogens from the urethra or bladder, which can be identified via appropriate diagnostics [97]. The predominant pathogens isolated are Enterobacterales, *C. trachomatis*, and *Neisseria gonorrhoeae* [98]. Figure 2 outlines the diagnostic and treatment algorithm for men with acute epididymitis.

3.12. Fournier's gangrene

Fournier's gangrene is an aggressive and frequently fatal polymicrobial soft-tissue infection of the perineum, perianal region, and external genitalia [99]. Typically, there is painful swelling of the scrotum or perineum with sepsis [99]. Patient risk factors for occurrence and mortality include immunocompromised status, most commonly because of diabetes or malnutrition, recent urethral or perineal surgery, and high body mass index. In up to 40% of cases, the onset is more insidious, with undiagnosed pain often resulting in delayed treatment [100]. A high index of suspicion and careful examination, particularly of obese patients, is required. Computed tomography or MRI can help in defining pararectal involvement, suggesting the need for bowel diversion [99]. A broad-spectrum antibiotic on presentation (Table 16 [101]), with subsequent refinement according to culture results and the clinical response, is recommended. The degree of internal necrosis is usually vastly greater than suggested by external signs, and consequently adequate repeated surgical debridement with urinary diversion via a suprapubic catheter is necessary to reduce mortality [99]. Adjunctive treatments for Fournier's gangrene should not be used except in the context of clinical trials.

3.13. Management of human papillomavirus in men

Human papillomavirus is one of the most frequently sexually transmitted viruses and encompasses both oncogenic (low- and high-risk variants) and non-oncogenic viruses. The most important information regarding diagnosis and therapy can be found in Figure 3. For further details, please refer to the full version of the guideline online (https://uroweb.org/guidelines/urological-infections).

3.14. Genitourinary tuberculosis

Although tuberculosis (TB) is one of the most common infectious diseases worldwide, its extrapulmonary manifestation, genitourinary TB (GUTB), is often underestimated by urologists, especially in non-endemic regions such as Europe. GUTB represented 4.6% of extrapulmonary TB cases in the EU between 1997 and 2017 [102]. The disease can affect all the genitourinary organs and is almost always secondary to the haematogenous spread of chronic latent TB infection (LTBI) [103]. Risk factors include primary TB and LTBI, diabetes, advanced age, low body mass index, oncological comorbidities, immunosuppression (including human immunodeficiency virus), renal failure, and poor socioeconomic conditions. The risk of reactivation is estimated to be up to 15% during an individual's lifetime [104]. Diagnosis of GUTB is challenging owing to the lack of a single diagnostic test (Table 17). Diagnosis relies on a high index of suspicion according to patient history, along with microbiological, molecular, and histological testing, as well as imaging findings . Patients typically present with nonspecific urological symptoms such as haematuria, an increase in urinary frequency, difficulty in voiding, and abdominal, lumbar, and suprapubic pain. Among female patients, symptoms may include menstrual irregularities and pelvic pain. Patients may also present for infertility issues; however, infertility and TB are not addressed in detail here.

Combination drug therapy is the first-line treatment for GUTB (Table 18). The World Health Organisation advises a daily regimen for 6 mo for newly diagnosed extrapulmonary TB, involving an initial intensive phase of 2 mo with

Please cite this article as: J. Kranz, R. Bartoletti, F. Bruyère et al., European Association of Urology Guidelines on Urological Infections: Summary of the 2024 Guidelines, Eur Urol (2024), https://doi.org/10.1016/j.eururo.2024.03.035

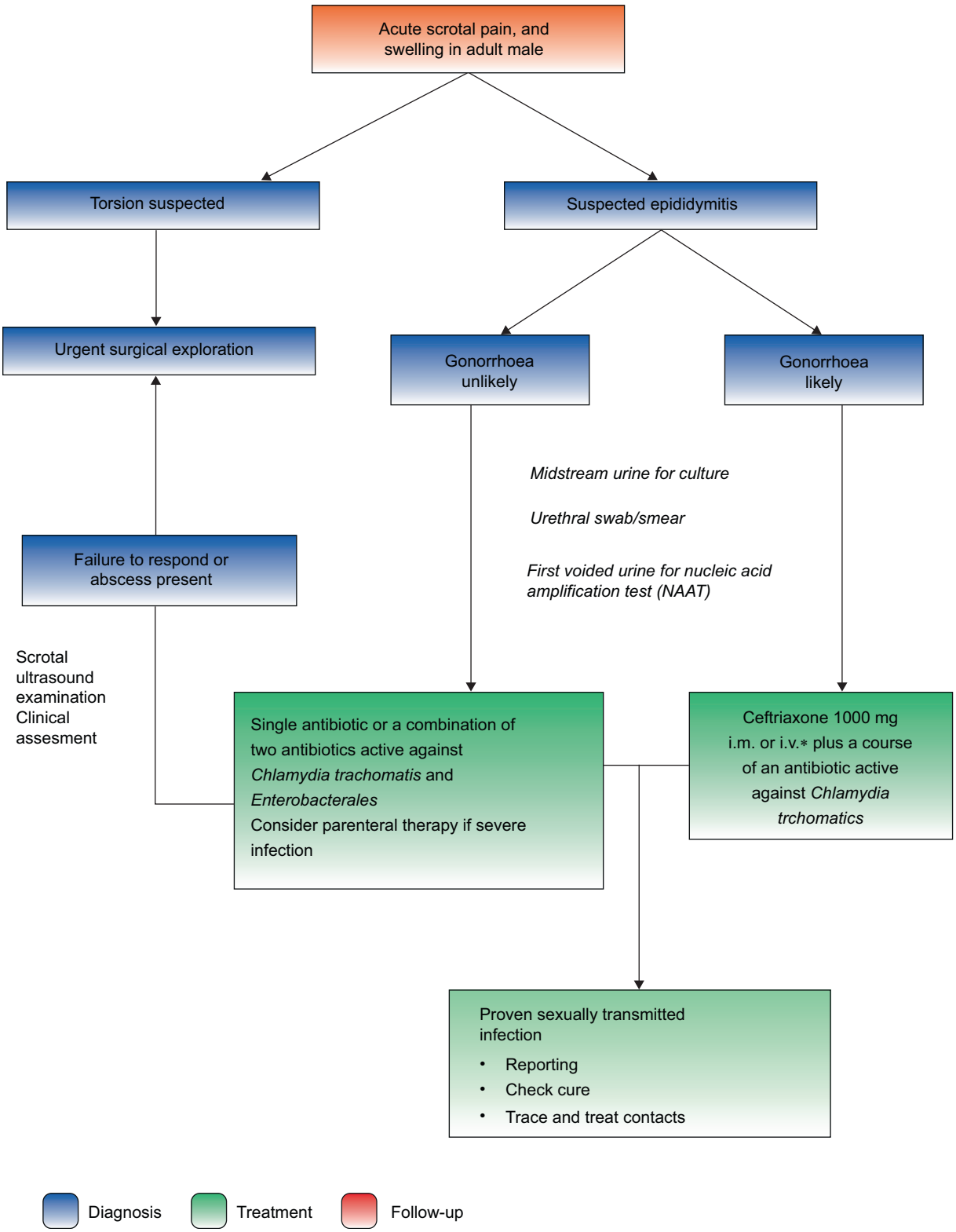

Fig. 2 – Diagnosis and treatment algorithm for epididymitis. i.m. = intramuscular; i.v. = intravenous. * Despite a lack of randomised controlled trials, there is increasing evidence that intravenous ceftriaxone is safe and effective for the treatment of gonorrhoeal infections and avoids the discomfort of an intramuscular injection for patients [68].

isoniazid, rifampicin, pyrazinamide, and ethambutol, followed by a 4-mo continuation phase with isoniazid and rifampicin [105]. In cases of multidrug-resistant TB (resistance to rifampicin and isoniazid), a personalised treatment approach is recommended, which should include at least five effective TB medications during the intensive phase,

Table 16 – Suggested antimicrobial therapy regimens for Fournier's gangrene of mixed microbiological aetiology (adapted from [101])

Antimicrobial	Dosage
Piperacillin-tazobactam plus	4.5 g every 6–8 h i.v.
Vancomycin	15 mg/kg every 12 h
Imipenem-cilastatin	1 g every 6–8 h i.v.
Meropenem	1 g every 8 h i.v.
Ertapenem	1 g once daily
Gentamicin	5 mg/kg daily
Cefotaxime plus	2 g every 6 h i.v.
Metronidazole or	500 mg every 6 h i.v.
Clindamycin	600–900 mg every 8 h i.v.
Cefotaxime plus	2 g every 6 h i.v.
Fosfomycin plus	5 g every 8 h i.v.
Metronidazole	500 mg every 6 h i.v.

i.v. = intravenous.

including pyrazinamide and four core second-line TB medicines [106].

Nevertheless, more than 50% of patients may require ablative, endoscopic, or reconstructive surgery owing to the destructive nature of TB infection, which is often compounded by delayed initial diagnosis [107–109]. Some 26.9% of GUTB cases have a nonfunctioning unilateral kidney and 7.4% have renal failure [109]. Owing to the scarcity of high-quality evidence regarding surgical management of GUTB, the panel was unable to make a definitive recommendation on surgical intervention. Patients with GUTB should be assessed on an individualised basis and the decision to operate taken depending on the location, extent of disease progression and damage to the genitourinary system.

3.15. *Periprocedural antibiotic prophylaxis*

Urological surgeons should prioritise and vigilantly maintain an aseptic environment to minimise the risk of infections originating from both endogenous (patient microbiome) and exogenous (nosocomial/health care–associated) sources. This entails the use of proper methods for instrument cleaning and sterilisation, implementation of

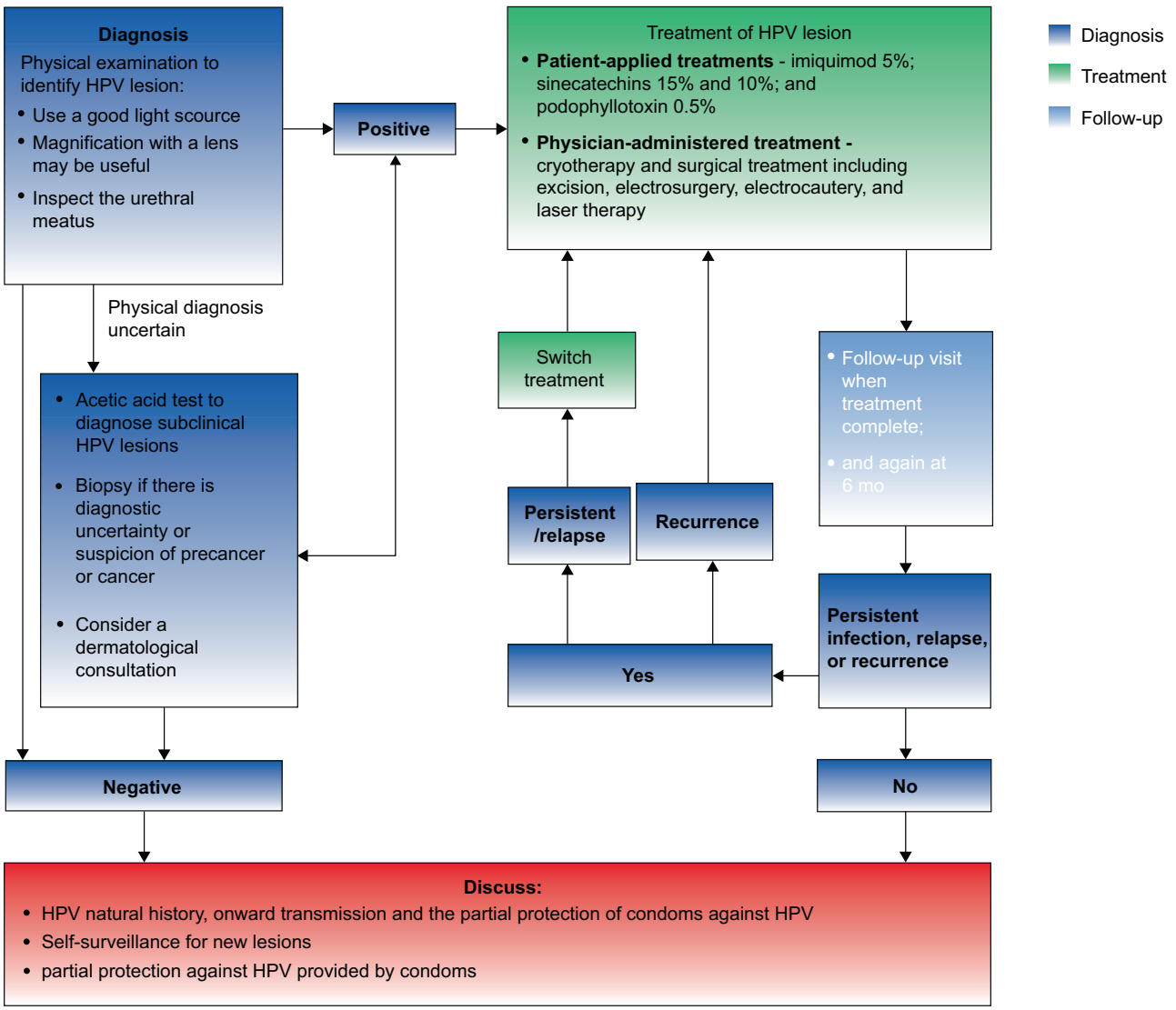

Fig. 3 – Diagnosis and treatment algorithm for management of human papillomavirus (HPV) in men.

Table 17 – Recommendations for the diagnosis of GUTB

Recommendation	Strength rating
Take a full medical history including history of previous tuberculosis infection (pulmonary and extrapulmonary) form all patients presenting with persistent nonspecific genitourinary symptoms and no identifiable cause.	Strong
Perform smear microscopy on urine, semen, tissue specimens, discharged or prostatic massage fluid using Ziehl-Neelsen or auramine staining in patients with suspected GUTB.	Weak
Perform an acid-fast bacillus culture on three midstream first-void urine samples on three consecutive days for *Mycobacterium tuberculosis* isolation in patients with suspected GUTB.	Strong
Use a recommended PCR test system in addition to a microbiological reference standard for urine specimens as a diagnostic test in patients with signs and symptoms of GUTB.	Weak
Use imaging modalities in combination with culture and/or PCR to aid in the diagnosis of GUTB and to assess the location and extent of damage to the genitourinary system.	Weak

GUTB = genitourinary tuberculosis; PCR = polymerase chain reaction.

Table 18 – Treatment regimens for newly diagnosed GUTB and MDR-TB [106]

Antimicrobial	Dosage
6-mo regimen for newly diagnosed GUTB	
Intensive 2-mo phase	
Isoniazid	5 mg/kg every 24 h; maximum daily dose 300 mg
Rifampicin	10 mg/kg every 24 h; maximum daily dose 600 mg
Pyrazinamide	25 mg/kg every 24 h; max daily dosage 2000 mg
Ethambutol	15–20 mg/kg every 24 h; maximum daily dose 800–1600 mg, depending on body weight
Continuation 4-mo phase	
Isoniazid	5 mg/kg every 24 h; maximum daily dose 300 mg
Rifampicin	10 mg/kg every 24 h; maximum daily dose 600 mg
Treatment regimen for MDR-TB	
Treat MDR-TB with an individualised treatment regimen including at least 5 effective TB medicines during the intensive phase, including pyrazinamide and 4 core second-line TB medicines.[a]	
Group A: fluoroquinolones	Levofloxacin, moxifloxacin, and gatifloxacin
Group B: second-line injectables	Amikacin, capreomycin, kanamycin, and streptomycin[b]
Group C: other second-line agents	Ethionamide/prothionamide, cycloserine/terizidone, linezolid, and clofazimine
Group D: add-on agents (not part of the core MDR-TB regimen)	D1: Pyrazinamide, ethambutol, and high-dose isoniazid D2: Bedaquiline and delamamid D3: *p*-Aminosalicylic acid, imipenem-cilastatin, meropenem, amoxicillin-clavulanate, and thioacetazone[c]

GUTB = genitourinary tuberculosis; MDR-TB = multidrug-resistant tuberculosis.
[a] Drugs should be chosen as follows: 1 from group A, 1 from group B, and at least 2 from group C. If the minimum number of five TB medicines cannot be composed of drugs included in groups A–C, an agent from group D2 and other agents from group D3 may be added to bring the total to five [106].
[b] Streptomycin can substitute for other injectable drugs if none of these agents can be used and if the strain is not resistant [106].
[c] Thioacetazone should not be used if the patient is seropositive for human immunodeficiency virus [106].

Table 19 – Suggested antimicrobial prophylaxis regimens before urological procedures

Procedure	Prophylaxis recommended	Antimicrobial
Urodynamics	No	N/A
Cystoscopy	No	
Extracorporeal shockwave lithotripsy	No	
Ureteroscopy	Yes	Trimethoprim
Percutaneous nephrolithotomy	Yes (single dose)	Trimethoprim-sulfamethoxazole
TUR of the prostate	Yes	Cephalosporin group 2 or 3
TUR of the bladder	Yes, in patients with high risk of postoperative sepsis.	Aminopenicillin plus a β-lactamase inhibitor
Transrectal prostate biopsy	Yes	1. Targeted prophylaxis on the basis of a rectal swab or stool culture. 2. Augmented prophylaxis with two or more different antibiotic classes.[a] 3. Alternative antibiotics • Fosfomycin trometamol[b] (eg, 3 g before and 3 g 24–48 h after biopsy) • Cephalosporin (eg, ceftriaxone 1 g i.m.; cefixime 400 mg p.o. for 3 d starting 24 h before biopsy) • Aminoglycoside (eg, gentamicin 3mg/kg i.v.; amikacin 15mg/kg i.m.)

TUR = transurethral resection; i.m. = intramuscular; i.v. = intravenous; p.o. = oral.
[a] This option contravenes antibiotic stewardship principles.
[b] The indication for fosfomycin trometamol for prostate biopsy has been withdrawn in Germany as the manufacturers did not submit the necessary pharmacokinetic data in support of this indication. Urologists are advised to check their local guidance in relation to the use of fosfomycin trometamol for prostate biopsy.

Please cite this article as: J. Kranz, R. Bartoletti, F. Bruyère et al., European Association of Urology Guidelines on Urological Infections: Summary of the 2024 Guidelines, Eur Urol (2024), https://doi.org/10.1016/j.eururo.2024.03.035

regular and thorough cleaning protocols for operating rooms and recovery areas, and meticulous disinfection of any potential sources of contamination. They should also have knowledge of the local pathogen prevalence for each type of procedure, their antibiotic susceptibility profiles, and their virulence to establish written local guidelines.

The available evidence enabled the panel to make recommendations concerning urodynamics, cystoscopy, stone procedures (extracorporeal shockwave lithotripsy, ureteroscopy, and percutaneous nephrolithotomy), transurethral resection of the prostate, and transurethral resection of the bladder. For nephrectomy and prostatectomy, the scientific evidence was too weak to allow the panel to make recommendations either for or against antibiotic prophylaxis (Table 19).

4. Conclusions

This summary of the EAU guidelines on urological infections provides current insights into the diagnosis, treatment, and prevention of urological infections, and can facilitate implementation of the recommendations in clinical practice, particularly focusing on antimicrobial stewardship in response to the escalating global threat of antimicrobial resistance. The full version is available online (https://uroweb.org/guidelines/urological-infections).

Author contributions: Jennifer Kranz had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study concept and design: Kranz, Bonkat, Bartoletti, Bruyère, Cai, Geerlings, Köves, Schubert, Pilatz, Veeratterapillay, Wagenlehner, Bausch, Devlies, Horváth, Leitner, Mantica, Mezei.
Acquisition of data: Kranz, Bonkat, Bartoletti, Bruyère, Cai, Geerlings, Köves, Schubert, Pilatz, Veeratterapillay, Wagenlehner, Bausch, Devlies, Horváth, Leitner, Mantica, Mezei, Smith.
Analysis and interpretation of data: Kranz, Bonkat, Bartoletti, Bruyère, Cai, Geerlings, Köves, Schubert, Pilatz, Veeratterapillay, Wagenlehner, Bausch, Devlies, Horváth, Leitner, Mantica, Mezei.
Drafting of the manuscript: Kranz, Bonkat.
Critical revision of the manuscript for important intellectual content: Kranz, Bonkat, Bartoletti, Bruyère, Cai, Geerlings, Köves, Schubert, Pilatz, Veeratterapillay, Wagenlehner, Bausch, Devlies, Horváth, Leitner, Mantica, Mezei.
Statistical analysis: None.
Obtaining funding: None.
Administrative, technical, or material support: Kranz, Bonkat, Smith.
Supervision: Kranz, Bonkat.
Other: None.

Financial disclosures: Jennifer Kranz certifies that all conflicts of interest, including specific financial interests and relationships and affiliations relevant to the subject matter or materials discussed in the manuscript (eg, employment/affiliation, grants or funding, consultancies, honoraria, stock ownership or options, expert testimony, royalties, or patents filed, received, or pending), are the following: Prof. Dr. G. Bonkat received consultation fee from Janssen-Cilag AG, OM Pharma, IBSA, Zambon SpA and Sun Pharma. He also received company speaker honorarium from Zambon SpA, OM Pharma, IBSA, Bionorica SE, Hoechst Marion Roussel and Sun Pharma. Additionally, he received fellowship and travel grants from Sun Pharma, OM Pharma, IBSA and Bionorica SE. Prof. F. Bruyère is a member of the infectious committee and a member of the General Assembly. Prof. Dr. SE Geerlings received consultation fee for an advisory board of Immunotek (2022). PD Dr. Jennifer Kranz received consultation fee from Bionorica, GSK and Shionogi. She also received company speaker honorarium from Bionorica, GSK, Janssen-Cilag GmbH, MSD and Apogepha Arzneimittel GmbH. She also received research grants from DFG. Dr. R. Veeratterapillay received travel grants fellowship from Ipsen Limited. Prof. Dr. F. Wagenlehner holds the position of Director Clinic of Urology, Pediatric Urology and Andrology for Justs Liebig University. He received consultation fee from Achaogen, Bionorica, GSK, Janssen, Klosterfrau, Pfizer, MIP Pharma, Shionogi, Spero and VenatorRX, OM-Pharma. He also received company speaker honorarium from Astellas, AstraZeneca, Bionorica, GSK, Janssen, Klosterfrau, MSD, Pfizer, MIP Pharma and OM-Pharma. Addtionally, he has trial participation with GSK, Klosterfrau, Select Immune, Janssen and VenatoRx. He also received research grants from DFG and DZIF. Nothing to disclose: Riccardo Bartoletti, Katharin Bausch, Tomasso Cai, Wout Devlies, József Horváth, Béla Köves, Lorenz Leitner, Guglielmo Mantica, Tünde Mezei, Sören Schubert, Emma J. Smith, Adrian Pilatz

***Funding/Support and role of the sponsor*:** None.

References

[1] Kranz J, Schmidt S, Lebert C, Schneidewind L, Schmiemann G, Wagenlehner F. Clinical practice guideline: uncomplicated bacterial community-acquired urinary tract infection in adults—epidemiology, diagnosis, treatment, and prevention. Dtsch Arztebl Int 2017;114:866–73. https://doi.org/10.3238/arztebl.2017.0866.
[2] Wagenlehner FME, Bjerklund Johansen TE, Cai T, et al. Epidemiology, definition and treatment of complicated urinary tract infections. Nat Rev Urol 2020;17:586–600. https://doi.org/10.1038/s41585-020-0362-4.
[3] Tandogdu Z, Cai T, Koves B, Wagenlehner F, Bjerklund-Johansen TE. Urinary tract infections in immunocompromised patients with diabetes, chronic kidney disease, and kidney transplant. Eur Urol Focus 2016;2:394–9.
[4] Antimicrobial Resistance Collaborators. Global burden of bacterial antimicrobial resistance in 2019: a systematic analysis. Lancet 2022;399:629–55.
[5] Guyatt GH, Oxman AD, Kunz R, et al. What is "quality of evidence" and why is it important to clinicians? BMJ 2008;336:995–8.
[6] Nicolle LE, Bradley S, Colgan R, et al. Infectious Diseases Society of America guidelines for the diagnosis and treatment of asymptomatic bacteriuria in adults. Clin Infect Dis 2005;40:643–54.
[7] Wagenlehner FM, Tandogdu Z, Bjerklund Johansen TE. An update on classification and management of urosepsis. Curr Opin Urol 2017;27:133–7.
[8] Tenke P, Koves B, Johansen TE. An update on prevention and treatment of catheter-associated urinary tract infections. Curr Opin Infect Dis 2014;27:102–7.
[9] Johansen TE, Botto H, Cek M, et al. Critical review of current definitions of urinary tract infections and proposal of an EAU/ESIU classification system. Int J Antimicrob Agents 2011;38 (Suppl):64–70.
[10] Lutay N, Ambite I, Grönberg Hernandez J, et al. Bacterial control of host gene expression through RNA polymerase II. J Clin Invest 2013;123:2366–79.
[11] Kass EH. Asymptomatic infections of the urinary tract. Trans Assoc Am Phys 1956;69:56–64.
[12] Gleckman R, Esposito A, Crowley M, Natsios GA. Reliability of a single urine culture in establishing diagnosis of asymptomatic bacteriuria in adult males. J Clin Microbiol 1979;9:596–7.
[13] Warren JW, Tenney JH, Hoopes JM, Muncie HL, Anthony WC. A prospective microbiologic study of bacteriuria in patients with chronic indwelling urethral catheters. J Infect Dis 1982;146:719–23.

[14] Hansson S, Jodal U, Lincoln K, Svanborg-Edén C. Untreated asymptomatic bacteriuria in girls: II—Effect of phenoxymethylpenicillin and erythromycin given for intercurrent infections. BMJ 1989;298:856–9.
[15] Cai T, Mazzoli S, Mondaini N, et al. The role of asymptomatic bacteriuria in young women with recurrent urinary tract infections: to treat or not to treat? Clin Infect Dis 2012;55:771–7.
[16] Kazemier BM, Koningstein FN, Schneeberger C, et al. Maternal and neonatal consequences of treated and untreated asymptomatic bacteriuria in pregnancy: a prospective cohort study with an embedded randomised controlled trial. Lancet Infect Dis 2015;15:1324–33.
[17] Wagenlehner FM, Hoyme U, Kaase M, Fünfstück R, Naber KG, Schmiemann G. Uncomplicated urinary tract infections. Dtsch Arztebl Int 2011;108:415–23.
[18] Stamm WE, Hooton TM. Management of urinary tract infections in adults. N Engl J Med 1993;329:1328–34.
[19] Foxman B, Somsel P, Tallman P, et al. Urinary tract infection among women aged 40 to 65: behavioral and sexual risk factors. J Clin Epidemiol 2001;54:710–8.
[20] van Buul LW, Vreeken HL, Bradley SF, et al. The development of a decision tool for the empiric treatment of suspected urinary tract infection in frail older adults: a Delphi consensus procedure. J Am Med Dir Assoc 2018;19:757–64.
[21] Bent S, Nallamothu BK, Simel DL, Fihn SD, Saint S. Does this woman have an acute uncomplicated urinary tract infection? JAMA 2002;287:2701–10.
[22] Bradbury SM. Collection of urine specimens in general practice: to clean or not to clean? J R Coll Gen Pract 1988;38:363–5.
[23] Lifshitz E, Kramer L. Outpatient urine culture: does collection technique matter? Arch Intern Med 2000;160:2537–40.
[24] Gágyor I, Bleidorn J, Kochen MM, Schmiemann G, Wegscheider K, Hummers-Pradier E. Ibuprofen versus fosfomycin for uncomplicated urinary tract infection in women: randomised controlled trial. BMJ 2015;351:h6544.
[25] Vik I, Bollestad M, Grude N, et al. Ibuprofen versus pivmecillinam for uncomplicated urinary tract infection in women—a double-blind, randomized non-inferiority trial. PLoS Med 2018;15:e1002569.
[26] Kronenberg A, Bütikofer L, Odutayo A, et al. Symptomatic treatment of uncomplicated lower urinary tract infections in the ambulatory setting: randomised, double blind trial. BMJ 2017;359:j4784.
[27] Wagenlehner FM, Abramov-Sommariva D, Höller M, Steindl H, Naber KG. Non-antibiotic herbal therapy (BNO 1045) versus antibiotic therapy (fosfomycin trometamol) for the treatment of acute lower uncomplicated urinary tract infections in women: a double-blind, parallel-group, randomized, multicentre, non-inferiority phase III trial. Urol Int 2018;101:327–36.
[28] Hooton TM. Recurrent urinary tract infection in women. Int J Antimicrob Agents 2001;17:259–68.
[29] Naber KG, Tirán-Saucedo J, Wagenlehner FM, RECAP group, et al. Psychosocial burden of recurrent uncomplicated urinary tract infections. GMS. Infect Dis 2022;10:Doc01.
[30] Cai T, Tamanini I, Collini L, et al. Management of recurrent cystitis in women: when prompt identification of risk factors might make a difference. Eur Urol Focus 2022;8:1476–82.
[31] Scholes D, Hooton TM, Roberts PL, Gupta K, Stapleton AE, Stamm WE. Risk factors associated with acute pyelonephritis in healthy women. Ann Intern Med 2005;142:20–7.
[32] Fulop T. Acute pyelonephritis workup. Medscape; 2012. https://emedicine.medscape.com/article/245559-workup.
[33] van Nieuwkoop C, Hoppe BP, Bonten TN, et al. Predicting the need for radiologic imaging in adults with febrile urinary tract infection. Clin Infect Dis 2010;51:1266–72.
[34] Cattrall JWS, Robinson AV, Kirby A, et al. A systematic review of randomised clinical trials for oral antibiotic treatment of acute pyelonephritis. Eur J Clin Microbiol Infect Dis 2018;37:2285–91.
[35] Gupta K, Hooton TM, Naber KG, et al. International clinical practice guidelines for the treatment of acute uncomplicated cystitis and pyelonephritis in women: a 2010 update by the Infectious Diseases Society of America and the European Society for Microbiology and Infectious Diseases. Clin Infect Dis 2011;52:e103–20.
[36] Berti F, Attardo TA, Piras S, et al. Short versus long course antibiotic therapy for acute pyelonephritis in adults: a systematic review and meta-analysis. Ital J Med 2018;12:39.
[37] Hooton TM. Clinical practice. Uncomplicated urinary tract infection. N Engl J Med 2012;366:1028–37.
[38] Pitout JD. Infections with extended-spectrum beta-lactamase-producing Enterobacteriaceae: changing epidemiology and drug treatment choices. Drugs 2010;70:313–33.
[39] Mombelli G, Pezzoli R, Pinoja-Lutz G, Monotti R, Marone C, Franciolli M. Oral vs intravenous ciprofloxacin in the initial empirical management of severe pyelonephritis or complicated urinary tract infections: a prospective randomized clinical trial. Arch Intern Med 1999;159:53–8.
[40] Millar LK, Wing DA, Paul RH, Grimes DA. Outpatient treatment of pyelonephritis in pregnancy: a randomized controlled trial. Obstet Gynecol 1995;86:560–4.
[41] Wing DA, Hendershott CM, Debuque L, Millar LK. A randomized trial of three antibiotic regimens for the treatment of pyelonephritis in pregnancy. Obstet Gynecol 1998;92:249–53.
[42] Ulleryd P, Sandberg T. Ciprofloxacin for 2 or 4 weeks in the treatment of febrile urinary tract infection in men: a randomized trial with a 1 year follow-up. Scand J Infect Dis 2003;35:34–9.
[43] Reyner K, Heffner AC, Karvetski CH. Urinary obstruction is an important complicating factor in patients with septic shock due to urinary infection. Am J Emerg Med 2016;34:694–6.
[44] Heyns CF. Urinary tract infection associated with conditions causing urinary tract obstruction and stasis, excluding urolithiasis and neuropathic bladder. World J Urol 2012;30:77–83.
[45] Spoorenberg V, Hulscher ME, Geskus RB, et al. Better antibiotic use in complicated urinary tract infections; multicentre cluster randomised trial of 2 improvement strategies. Ned Tijdschr Geneeskd 2016;160:D460.
[46] Bader MS, Loeb M, Brooks AA. An update on the management of urinary tract infections in the era of antimicrobial resistance. Postgrad Med 2017;129:242–58.
[47] Geerlings SE, van den Broek PJ, van Haarst EP, et al. Optimisation of the antibiotic policy in the Netherlands. X. The SWAB guideline for antimicrobial treatment of complicated urinary tract infections. Ned Tijdschr Geneeskd 2006;150:2370–6.
[48] Hooton TM, Bradley SF, Cardenas DD, et al. Diagnosis, prevention, and treatment of catheter-associated urinary tract infection in adults: 2009 International Clinical Practice Guidelines from the Infectious Diseases Society of America. Clin Infect Dis 2010;50:625–63.
[49] Peterson J, Kaul S, Khashab M, Fisher A, Kahn JB. Identification and pretherapy susceptibility of pathogens in patients with complicated urinary tract infection or acute pyelonephritis enrolled in a clinical study in the United States from November 2004 through April 2006. Clin Ther 2007;29:2215–21.
[50] Wagenlehner FM, Umeh O, Steenbergen J, Yuan G, Darouiche RO. Ceftolozane-tazobactam compared with levofloxacin in the treatment of complicated urinary-tract infections, including pyelonephritis: a randomised, double-blind, phase 3 trial (ASPECT-cUTI). Lancet 2015;385:1949–56.
[51] Rudrabhatla P, Deepanjali S, Mandal J, Swaminathan RP, Kadhiravan T. Stopping the effective non-fluoroquinolone antibiotics at day 7 vs continuing until day 14 in adults with acute pyelonephritis requiring hospitalization: a randomized non-inferiority trial. PLoS One 2018;13:e0197302.
[52] Ren H, Li X, Ni ZH, et al. Treatment of complicated urinary tract infection and acute pyelonephritis by short-course intravenous levofloxacin (750 mg/day) or conventional intravenous/oral levofloxacin (500 mg/day): prospective, open-label, randomized, controlled, multicenter, non-inferiority clinical trial. Int Urol Nephrol 2017;49:499–507.
[53] Gould CV, Umscheid CA, Agarwal RK, et al. Guideline for prevention of catheter-associated urinary tract infections 2009. Infect Control Hosp Epidemiol 2010;31:319–26.
[54] Garibaldi RA, Burke P, Dickman ML, Smith CB. Factors predisposing to bacteriuria during indwelling urethral catheterization. N Engl J Med 1974;291:215–9.
[55] Kunin CM, McCormack RC. Prevention of catheter-induced urinary-tract infections by sterile closed drainage. N Engl J Med 1966;274:1155–61.

Please cite this article as: J. Kranz, R. Bartoletti, F. Bruyère et al., European Association of Urology Guidelines on Urological Infections: Summary of the 2024 Guidelines, Eur Urol (2024), https://doi.org/10.1016/j.eururo.2024.03.035

[56] Hartstein AI, Garber SB, Ward TT, Jones SR, Morthland VH. Nosocomial urinary tract infection: a prospective evaluation of 108 catheterized patients. Infect Control 1981;2:380–6.
[57] Warren JW, Damron D, Tenney JH, Hoopes JM, Deforge B, Muncie Jr HL. Fever, bacteremia, and death as complications of bacteriuria in women with long-term urethral catheters. J Infect Dis 1987;155:1151–8.
[58] Classen DC, Larsen RA, Burke JP, Stevens LE. Prevention of catheter-associated bacteriuria: clinical trial of methods to block three known pathways of infection. Am J Infect Control 1991;19:136–42.
[59] Saint S, Lipsky BA. Preventing catheter-related bacteriuria: should we? Can we? How? Arch Intern Med 1999;159:800–8.
[60] Maki DG, Tambyah PA. Engineering out the risk for infection with urinary catheters. Emerg Infect Dis 2001;7:342–7.
[61] Li F, Song M, Xu L, Deng B, Zhu S, Li X. Risk factors for catheter-associated urinary tract infection among hospitalized patients: a systematic review and meta-analysis of observational studies. J Adv Nurs 2019;75:517–27.
[62] Wilson ML, Mitchell M, Morris AJ, et al. Principles and procedures for blood cultures; approved guideline. Clinical and Laboratory Standards Institute; 2007. https://clsi.org/media/1448/m47a_sample.pdf.
[63] Howell MD, Davis AM. Management of sepsis and septic shock. JAMA 2017;317:847–8.
[64] Dellinger RP, Levy MM, Rhodes A, et al. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock, 2012. Intensive Care Med 2013;39:165–228.
[65] Zhang N, Wang R, Li X, Liu X, Tang Z, Liu Y. Are Ureaplasma spp. a cause of nongonococcal urethritis? A systematic review and meta-analysis. PLoS One 2014;9:e113771.
[66] Horner PJ, Blee K, Falk L, van der Meijden W, Moi M. 2016 European guideline on the management of non-gonococcal urethritis. Int J STD AIDS 2016;27:928–37.
[67] Ong JJ, Sarumpaet A, Chow EP, et al. Should female partners of men with non-gonococcal urethritis, negative for Chlamydia trachomatis and Mycoplasma genitalium, be informed and treated? Clinical outcomes from a partner study of heterosexual men with NGU. Sex Trans Dis 2017;44:126–30.
[68] Muratani T, Inatomi H, Ando Y, Kawai S, Akasaka S, Matsumoto T. Single dose 1 g ceftriaxone for urogenital and pharyngeal infection caused by Neisseria gonorrhoeae. Int J Urol 2008;15:837–42.
[69] Schneider H, Ludwig M, Hossain HM, Diemer T, Weidner W. The 2001 Giessen Cohort Study on patients with prostatitis syndrome—an evaluation of inflammatory status and search for microorganisms 10 years after a first analysis. Andrologia 2003;35:258–62.
[70] Lipsky BA, Byren I, Hoey CT. Treatment of bacterial prostatitis. Clin Infect Dis 2010;50:164152.
[71] Alexander RB, Ponniah S, Hasday J, Hebel JR. Elevated levels of proinflammatory cytokines in the semen of patients with chronic prostatitis/chronic pelvic pain syndrome. Urology 1998;52:744–9.
[72] Alexander RB, Trissel D. Chronic prostatitis: results of an Internet survey. Urology 1996;48:568–74.
[73] Zermann DH, Ishigooka M, Doggweiler R, Schmidt RA. Neurourological insights into the etiology of genitourinary pain in men. J Urol 1999;161:903–8.
[74] Schaeffer AJ. Prostatitis: US perspective. Int J Antimicrob Agents 1999;11:205–11.
[75] Krieger JN, Nyberg Jr L, Nickel JC. NIH consensus definition and classification of prostatitis. JAMA 1999;282:236–7.
[76] National Institute of Diabetes and Digestive and Kidney Disease. Chronic prostatitis workshop, Bethesda, MD, 1995.
[77] Wagenlehner FM et al.. Bacterial prostatitis. World J Urol 2013;31:711.
[78] Naber KG, Weidner W. Prostatitis, epididymitis and orchitis. In: Armstrong D, Cohen J, editors. Infectious diseases. London, UK: Mosby; 1999.
[79] Badalyan RR, Fanarjyan SV, Aghajanyan IG. Chlamydial and ureaplasmal infections in patients with nonbacterial chronic prostatitis. Andrologia 2003;35:263–5.
[80] Berger RE. Epididymitis. In: Holmes KK, Mardh PA, Sparling PF, Wiesner PJ, editors. Sexually transmitted diseases. New York, NY: McGraw-Hill; 1984.
[81] Robinson AJ, Grant JB, Spencer RC, Potter C, Kinghorn GR. Acute epididymitis: why patient and consort must be investigated. Br J Urol 1990;66:642–5.
[82] Krieger JN, Egan KJ, Ross SO, Jacobs R, Berger RE. Chronic pelvic pains represent the most prominent urogenital symptoms of "chronic prostatitis". Urology 1996;48:715–21.
[83] Nickel JC. Effective office management of chronic prostatitis. Urol Clin North Am 1998;25:677–84.
[84] Gill BC, Shoskes DA. Bacterial prostatitis. Curr Opin Infect Dis 2016;29:86–91.
[85] Wagenlehner FM, Naber KG. Prostatitis: the role of antibiotic treatment. World J Urol 2003;21:105–8.
[86] Krieger JN. Recurrent lower urinary tract infections in men. J New Rem Clin 1998;47:4–15.
[87] Litwin MS, McNaughton-Collins M, Fowler Jr FH, et al. The National Institutes of Health chronic prostatitis symptom index: development and validation of a new outcome measure. J Urol 1999;162:369–75.
[88] Schaeffer AJ, Weidner W, Barbalias GA, et al. Summary consensus statement: diagnosis and management of chronic prostatitis/chronic pelvic pain syndrome. Eur Urol Suppl 2003;2(2):1–4.
[89] Bjerklund Johansen TE, Grüneberg RN, Guibert J, et al. The role of antibiotics in the treatment of chronic prostatitis: a consensus statement. Eur Urol 1998;34:457–66.
[90] Perletti G, Marras E, Wagenlehner F, Magri V. Antimicrobial therapy for chronic bacterial prostatitis. Cochrane Database Syst Rev 2013;2013:CD009071.
[91] Cai T, Mazzoli S, Addonisio P, Boddi V, Geppetti P, Bartoletti R. Clinical and microbiological efficacy of prulifloxacin for the treatment of chronic bacterial prostatitis due to Chlamydia trachomatis infection: results from a prospective, randomized and open-label study. Methods Find Exp Clin Pharmacol 2010;32:39–45.
[92] Hua LX, Zhang JX, Wu HF, et al. The diagnosis and treatment of acute prostatitis: report of 35 cases. Zhonghua Nan Ke Xue 2005;11:897–9.
[93] Yoon BI, Kim S, Han DS, et al. Acute bacterial prostatitis: how to prevent and manage chronic infection? J Infect Chemother 2012;18:444–50.
[94] Ludwig M, Schroeder-Printzen I, Schiefer HG, Weidner W. Diagnosis and therapeutic management of 18 patients with prostatic abscess. Urology 1999;53:340–5.
[95] Chou YH, Tiu CM, Liu JY, et al. Prostatic abscess: transrectal color Doppler ultrasonic diagnosis and minimally invasive therapeutic management. Ultrasound Med Biol 2004;30:719–24.
[96] Çek M, Sturdza L, Pilatz A. Acute and chronic epididymitis. Eur Urol Suppl 2017;16:124–31.
[97] Pilatz A, Hossain H, Kaiser R, et al. Acute epididymitis revisited: impact of molecular diagnostics on etiology and contemporary guideline recommendations. Eur Urol 2015;68:428–35.
[98] Harnisch JP, Berger RE, Alexander ER, Monda G, Holmes KK. Aetiology of acute epididymitis. Lancet 1977;1:819–21.
[99] Chennamsetty A, Khourdaji I, Burks F, Killinger KA. Contemporary diagnosis and management of Fournier's gangrene. Ther Adv Urol 2015;7:203–15.
[100] Eke N. Fournier's gangrene: a review of 1726 cases. Br J Surg 2000;87:718–28.
[101] Stevens DL, Bisno AL, Chambers HF, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. Clin Infect Dis 2014;59:147–59.
[102] Hayward SE, Rustage K, Nellums LB, et al. Extrapulmonary tuberculosis among migrants in Europe, 1995 to 2017. Clinical Microbiol Infect 2021;27:1347.e1–e7.
[103] Kang W, Liu S, Du J, et al. Epidemiology of concurrent extrapulmonary tuberculosis in inpatients with extrapulmonary tuberculosis lesions in China: a large-scale observational multi-centre investigation. Int J Infect Dis 2022;115:79–85.
[104] Vynnycky E, Fine PE. The natural history of tuberculosis: the implications of age-dependent risks of disease and the role of reinfection. Epidemiol Infect 1997;119:183–201.
[105] World Health Organization. WHO consolidated guidelines on tuberculosis module 4: treatment: drug-susceptible tuberculosis

Please cite this article as: J. Kranz, R. Bartoletti, F. Bruyère et al., European Association of Urology Guidelines on Urological Infections: Summary of the 2024 Guidelines, Eur Urol (2024), https://doi.org/10.1016/j.eururo.2024.03.035

treatment 2022. https://www.who.int/publications/i/item/9789240048126.

[106] European Centre for Disease Prevention and Control. European Union standards for tuberculosis care – 2017 update. https://www.ecdc.europa.eu/en/publications-data/european-union-standards-tuberculosis-care-2017-update.

[107] Huang Y, Chen B, Cao D, et al. Surgical management of tuberculous epididymo-orchitis: a retrospective study of 81 cases with long-term follow-up. BMC Infect Dis 2021;21:1068.

[108] Mittal A, Ranjan S, Narain T, Panwar V. Surgical management of genitourinary tuberculosis: our experience and review of literature. Pol Przegl Chir 2020;92:1–6.

[109] Figueiredo AA, Lucon AM, Falci Jr R, Srougi M. Epidemiology of urogenital tuberculosis worldwide. Int J Urol 2008;15:827–32.

Please cite this article as: J. Kranz, R. Bartoletti, F. Bruyère et al., European Association of Urology Guidelines on Urological Infections: Summary of the 2024 Guidelines, Eur Urol (2024), https://doi.org/10.1016/j.eururo.2024.03.035