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Urosepsis

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Introduction

Sepsis is the body's inflammatory response to an infection that can lead to multi-organ dysfunction, failure, and even death. Urosepsis is sepsis caused by an infection of the urinary tract (UTI). These infections include cystitis (lower urinary tract/bladder) and pyelonephritis (upper urinary tract/kidney). Nearly 25% of sepsis cases originate from the urogenital tract.[1][2][3]

Etiology

The most common pathogen causing UTIs (and in turn urosepsis) is *Escherichia coli* (50%) followed *Proteus* (15%), *Enterobacter* (15%), *Klebsiella* (15%), *Pseudomonas aeruginosa* (5%), and gram-positive bacteria (15%).[4][4][5]

Epidemiology

In the United States, the number of sepsis cases appears to be increasing over the last 30 to 40 years, but the overall mortality of patients from sepsis has declined. It is estimated that the mortality rate from urosepsis is 30% to 40%. The severity of the disease also appears to be increasing, with the most common manifestations of organ dysfunction being acute respiratory distress syndrome (ARDS), acute kidney injury, and disseminated intravascular coagulopathy (DIC).[6][7]

Pathophysiology

A UTI begins with colonization of the urethral meatus or vaginal introitus by either uropathogens or fecal flora that ascends via the urethra to the bladder. It may also ascend further up to the kidneys via the ureters and cause pyelonephritis. Pyelonephritis also can be caused by bacterial seeding of the kidneys via the lymphatics.[8][9]

While the bacterial pathogen causes the initiation of the disease process, the host response drives and defines sepsis and its severity. The immune response of the host is triggered when the proteins of the microbial pathogen interact with those of the host's cell membrane. The severe pro-inflammatory response can lead to cell necrosis, an increase in neutrophil production that produces bactericidal substances, and increased permeability of endothelial cells that lead to edema formation. After this initial phase, there is an anti-inflammatory response that leads to immunosuppression in the body where neutrophils become dysfunctional and cause further damage to nearby cells. Other systems in the body affected by this inflammatory response include coagulation, autonomic, and endocrine systems.

History and Physical

A practitioner who suspects sepsis in a patient should complete a full physical exam to identify the source of the infection. For urosepsis, one should focus the physical exam on findings signs of UTI or pyelonephritis. This includes costovertebral angle tenderness, evidence of urinary retention and, in men, scrotal pain or fluctuance or tenderness on the digital rectal exam (suggesting prostatitis or abscess). One should also note an indwelling catheter during this exam as that will help guide antibiotic treatment.

Evaluation

The diagnosis of urosepsis requires both suspicion of sepsis as well as evidence of a UTI. SIRS (systemic inflammatory response syndrome) signs and symptoms are indicators of sepsis but are no longer required for the formal diagnosis. This includes abnormalities in body temperature (warm skin), heart rate (bounding pulses, tachycardia), respiratory rate, and white blood cell count (elevation or depression). In more severe forms sepsis is diagnosed when there is evidence of organ dysfunction, including but not limited to kidney dysfunction evidenced by decreased urine output, encephalopathy with an abrupt change in mental status, or a low platelet count. The diagnosis of a complicated UTI in the setting of SIRS includes signs and symptoms (dysuria, flank pain), physical exam findings (costovertebral angle tenderness, evidence of urinary retention), radiologic features, and laboratory results showing bacteriuria and leucocyturia.[10][11]

If urosepsis is suspected, the laboratory workup should include a urinalysis with a urine culture. Providers should also order a complete blood cell count, blood cultures, and a lactate test to look for evidence of end-organ dysfunction. Post-renal obstruction is one of the most common causes of urosepsis. Ultrasound can identify 93% of common contributing causes of urosepsis such as hydronephrosis or prostate abscess. However, the dependence on an experienced ultrasonographer and their availability often limit the utilization of ultrasound for such patients. CT imaging is rapidly available at all hours and is easily reproducible without a need for technologist expertise. This often makes CT scan the diagnostic modality of choice when urosepsis is suspected. It can also identify subtle radiographic findings not easily visualized with ultrasound.

Treatment / Management

Over the last few decades, there has been an increase in multi-drug resistant organisms causing UTIs, making treatment more complicated. This is thought to stem from the overuse of antibiotics to treat suspected UTIs as confirmation is not available for 2 to 3 days until a urine culture with antimicrobial susceptibility has resulted.[12][13]

A lot of research aimed at reducing mortality from sepsis via aggressive treatment modalities, including the original Rivers trial, has shown that early goal-directed therapy (EGDT) reduces mortality from sepsis. While some of the original recommendations of EGDT have been challenged recently, these overall treatment guidelines remain (1) rapid initiation of empiric antibiotics that are chosen to eliminate the suspected source of infection, (2) supportive care, including hemodynamic and pulmonary stabilization, and (3) adjunctive therapies. When choosing antibiotics, the goal is to have these infused into the patient within one hour of a confirmed diagnosis, but they should be given only after urine and blood cultures have been collected. Each hour antibiotics are delayed after the initial six hours is associated with an 8% decrease in survival. Local susceptibility patterns guide antibiotic choice but often includes a third-generation cephalosporin, piperacillin in combination with beta-lactamase inhibitor or a fluoroquinolone. It is important to remember in dosing these antibiotics that many patients with severe sepsis have end-organ dysfunction, including renal and liver impairment, which might affect clearance of antibiotics.

In addition to early antibiotics, there are other important parts of the management of sepsis. Initial fluid resuscitation with crystalloid is still recommended at a minimum of 30 mL/kg. Consider early administration of vasopressor support to maintain a mean arterial pressure greater than 65 mm Hg. The first choice for vasopressor support in sepsis is norepinephrine (with epinephrine and vasopressin 2 and 3). Tight glucose control is also recommended, with corticosteroids and blood products being more controversial in the literature.

If the patient has a complicating factor in the urinary tract that is identified and warrants treatment, it should be performed as soon as possible (e.g., Foley catheter placement to relieve urinary retention or stent placement to bypass an obstructing ureteral calculus causing urosepsis).

Looking to the future, there is hope for new technologies for early identification of pathogens and rapid anti-microbial susceptibility including biosensors, microfluidics, and other integrated platforms. There is also work on treatments that are directed toward the massive secretion of inflammatory cytokines that are present in severe sepsis.

Differential Diagnosis

Sepsis is the body's response to an infection. Urosepsis is, of course, only one possible cause, and when a patient arrives and the clinician suspect sepsis as the problem, they must look for other sources of infection besides the urinary tract and consider broad-spectrum antibiotics early on if no clear source is identified.

Enhancing Healthcare Team Outcomes

Urosepsis is best managed with an interprofessional team of health professionals that include a nephrologist, infectious disease expert, urologist, intensivist, a nurse and a pharmacist. The key to preventing high morbidity is to prevent the condition that promotes infection. Any urological abnormality that is present needs to be corrected. The pharmacist should ensure that there is no delay in the administration of the antibiotics. The nurse should closely monitor the patient for signs of sepsis and communicate with the healthcare provider. Many of these patients require ICU care with close monitoring and frequent radiological studies. [14][15](Level V).

Outcomes

The outcomes after urosepsis depend on the cause and severity of the infection. The prognosis also depends on the type of bacteria, antimicrobial resistance, and patient comorbidity. This is where the input of a board-certified infectious disease pharmacist can be very beneficial, helping select appropriate antimicrobial coverage, check for drug interactions, and verify all dosing, and report any concerns to the clinical team. If left untreated, the condition has a very high mortality. Even those who survive have a prolonged recovery period. Residual disturbance in renal function is not uncommon.[16][17] [Level 5]

Questions

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References

1. Areda MA, Bailey CR, O'Mara D, Weiss CR. Transplant uretero-inguinal hernia resulting in urosepsis. Radiol Case Rep. 2019 Jan;14(1):14-17. [PMC free article: PMC6172483] [PubMed: 30305858]
2. Thornton HV, Hammond A, Hay AD. Urosepsis: a growing and preventable problem? Br J Gen Pract. 2018 Oct;68(675):493-494. [PMC free article: PMC6145996] [PubMed: 30262628]

3. Fadel MG, Louis C, Tay A, Bolgeri M. Obstructive urosepsis secondary to ureteric herniation into the sciatic foramen. BMJ Case Rep. 2018 Sep 27;2018 [PMC free article: PMC6169623] [PubMed: 30262527]
4. Vikrant S, Gupta D, Singh M. Epidemiology and outcome of acute kidney injury from a tertiary care hospital in India. Saudi J Kidney Dis Transpl. 2018 Jul-Aug;29(4):956-966. [PubMed: 30152435]
5. Scotland KB, Lange D. Prevention and management of urosepsis triggered by ureteroscopy. Res Rep Urol. 2018;10:43-49. [PMC free article: PMC6038880] [PubMed: 30013956]
6. 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. 2018 Jun 18;50(3):507-513. [PubMed: 29930421]
7. 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? Urol. Int. 2018;100(2):203-208. [PubMed: 29421810]
8. Sarhan M, von Mässenhausen A, Hugo C, Oberbauer R, Linkermann A. Immunological consequences of kidney cell death. Cell Death Dis. 2018 Jan 25;9(2):114. [PMC free article: PMC5833784] [PubMed: 29371597]
9. Kodner CM, Thomas Gupton EK. Recurrent urinary tract infections in women: diagnosis and management. Am Fam Physician. 2010 Sep 15;82(6):638-43. [PubMed: 20842992]
10. Vigil HR, Hickling DR. Urinary tract infection in the neurogenic bladder. Transl Androl Urol. 2016 Feb;5(1):72-87. [PMC free article: PMC4739987] [PubMed: 26904414]
11. Dreger NM, Degener S, Ahmad-Nejad P, Wöbker G, Roth S. Urosepsis--Etiology, Diagnosis, and Treatment. Dtsch Arztebl Int. 2015 Dec 04;112(49):837-47; quiz 848. [PMC free article: PMC4711296] [PubMed: 26754121]
12. 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. Med Mal Infect. 2018 Aug;48(5):327-358. [PubMed: 29759852]
13. Wagenlehner FM, Tandogdu Z, Bjerklund Johansen TE. An update on classification and management of urosepsis. Curr Opin Urol. 2017 Mar;27(2):133-137. [PubMed: 27846034]
14. Heppner HJ, Yapan F, Wiedemann A. [Urosepsis in Geriatric Patients]. Aktuelle Urol. 2016 Feb;47(1):54-9. [PubMed: 26916045]
15. Goveas B. Urosepsis: A simple infection turns toxic. Nurse Pract. 2017 Jul 15;42(7):53-54. [PubMed: 28622259]
16. 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. Eur Urol Focus. 2020 Jan 15;6(1):151-156. [PubMed: 30219711]
17. 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 J Urol. 2019 May;37(5):921-929. [PubMed: 30120500]

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