QMJ
A peer reviewed journal
QATAR MEDICAL JOURNAL

RESEARCH ARTICLE

Community Acquired Urosepsis: A surgical intensive care Experience

Nissar Shaikh[1], Umais Momin[2], Ahmed Atef Shible[3], Muna Al-Musalmani[4], Abdulla Ansari[5]

Address for Correspondence:
Nissar Shaikh
[1]Surgical Intensive Care Unit, Hamad Medical Corporation, Doha, Qatar
[2]Department of Radiology, Hamad Medical Corporation, Doha, Qatar
[3]Clinical Pharmacist, Hamad Medical Corporation, Doha-Qatar Qatar
[4]Medical Director CDC, Hamad Medical Corporation, Doha, Qatar
[5]Chief Medical Officer, Hamad Medical Corporation, Doha, Qatar
Email: nissatfirdous@gmail.com

http://dx.doi.org/10.5339/qmj.2020.8
Submitted: 21 August 2019
Accepted: 24 October 2019

Cite this article as: Shaikh N, Momin U, Atef Shible A, Al- Musalmani M, Ansari A. Community Acquired Urosepsis: A surgical intensive care Experience, Qatar Medical Journal 2020:8 http://dx.doi.org/10.5339/qmj.2020.8

كيوساينس
QSCIENCE
دار جامعة حمد بن خليفة للنشر
HAMAD BIN KHALIFA UNIVERSITY PRESS

ABSTRACT

Urosepsis contributes significantly to the epidemiology of sepsis. Urosepsis can be classified as community acquired or hospital acquired, depending upon the origin of infection acquisition: either from the community or from a healthcare facility.
A great deal of literature is available about nosocomial urosepsis, but the literature regarding community-acquired urosepsis (CAUs) is limited, and studies are underpowered. The aim of our study was to determine the epidemiology, bacteriology, severity, and outcome of CAUs.
Methods and Patients: All patients admitted from the emergency department to the surgical intensive care unit (SICU) with urosepsis over a period of 10 years were identified and included retrospectively from the SICU registry. The study was retrospective. Data were entered into the SPSS program version 23, and groups were compared by using chi-square and t-tests. Results were considered statistically significant at $p \leq 0.05$.
Results: During the study period, 302 patients with CAUs were admitted to the SICU. The common etiology was obstructive uropathy (60%). The Local Arab population outnumbered the non-Arab population (164/54.3%), and there were equal numbers of patients of both genders. Diabetes mellitus and hypertension together were the common comorbidities. Seventy-five percent of patients had acute kidney injury (AKI). Thirty-eight percent of patients had percutaneous nephrostomy, and 24.8% of patients underwent endoscopic stent insertion to relieve the obstruction. Ninety-three percent of patients were admitted with septic shock, and 71.5% had bacteremia. The common bacteria (36.1%) was extended-spectrum beta-lactamase-(ESBL)-producing bacteria, with a predominance of *Escherichia coli* (31.5%). Fifty-four percent of

patients required a change of antibiotics to carbapenem. Eighty-two percent of patients had acute respiratory distress syndrome (ARDS). Patients with bacteremia had a statistically significant AKI, ARDS, and septic shock ($p < 0.001$). Male patients had a significantly higher incidence of oliguria, intubation, and ARDS ($p < 0.05$). Eight patients died of urosepsis during the study period, giving a mortality rate of 2.6%.
Conclusion: In our patients, obstruction of urine flow was the most common cause of CAUs. Our urosepsis patients had a higher bacteremia rate, which led to higher incidences of organ dysfunction and septic shock. ESBL bacteria were a frequent cause of urosepsis, requiring a change of the initial antibiotic to carbapenem. Male patients had a significantly higher rate of organ dysfunction. Mortality in our urosepsis patients was lower than mentioned in the literature.

Keywords: Acute respiratory distress syndrome, acute kidney injury, Arabs, bacteremia, diabetes mellitus, *Escherichia coli*, extended-spectrum beta-lactamase, fever, leukocytosis, oliguria, septic shock, urosepsis

Sepsis is one of the leading causes of global mortality, overtaking carcinomas of the colon, breast, and lung.[1] In 31% of sepsis cases, the source is the urogenital tract; accordingly, it is called urosepsis.[2] Broadly, urosepsis is classified into community-acquired urosepsis (CAUs) and hospital-acquired (nosocomial) urosepsis.[3] Community-acquired urosepsis is sepsis acquired in the community, and patients present to the emergency department, whereas in hospital-acquired urosepsis, sepsis occurs during the hospital stay when the patients are admitted for the management of other etiologies.[2,3] Each type of urosepsis differs in its mode of infection, bacteriology, treatment, and outcome. Hospital-acquired urosepsis is caused by multidrug-resistant bacteria, and the mortality rate is high.[1,2,3] The epidemiology and management of nosocomial or hospital-acquired urosepsis is well described in the literature,[2] whereas the literature regarding CAUs and its intensive care management is sparse and consists of smaller studies that do not describe organ dysfunction, therapeutic interventions, and intensive care therapy.[4,5,6]

The aim of our study was to determine the epidemiology, bacteriology, antibiotic therapy, and intensive care management of CAUs.

PATIENTS AND METHODS

After permission was obtained from the research department of our institution (Permission Number: 13429/13), all patients admitted to the surgical intensive care unit of our hospital (the only tertiary care hospital in the country) with urosepsis were identified from the admission registry for a duration of 10 years (2008 – 18). Urosepsis patients' demographic data, presenting complaints, laboratory data on admission, diagnosis, bacteriology, antibiotic therapy (initial antibiotic and subsequent antibiotic prescribed after culture and sensitivity results were available), sequential organ failure assessment (SOFA) score, ICU length of stay, image-guided interventions, endoscopic/surgical interventions performed, severity of sepsis or septic shock status, and outcome were recorded retrospectively. The medical research department waived the need for informed consent as this was a retrospective study.

Urosepsis is defined as systemic inflammatory response syndrome with a suspected or diagnosed source in the urogenital tract.[2] Urosepsis is severe if sepsis leads to organ dysfunction in these patients. If these patients with severe sepsis develop hypotension and do not respond to fluid resuscitation, they are in septic shock.[7]

Tachycardia was considered when patients had a heart rate exceeding 90 beats per minute, tachypnoea when the respiratory rate exceeded 26 inhalations per minute, leukocytosis when the white blood cell count exceeded 11×10^3 cells/μL, and thrombocytopenia if platelet count was less than 100×10^3 platelets/μL. A patient was considered febrile if their core body temperature exceeded 38.3 °C. The severity of the disease and organ dysfunction were evaluated using the SOFA score. Urosepsis patients were identified by clinical presentation and a positive urinary culture from the emergency department and confirmed by ultrasonography or computed tomography.

Patients in septic shock were managed as per the surviving sepsis campaign guidelines.[8] Patients admitted with obstructive uropathy or hydro/pyonephrosis had either image-guided percutaneous nephrostomy or endoscopic stent insertion. Patients with severe coagulopathy or hemodynamic instability or those unable to tolerate positioning for nephrostomy had endoscopic surgical interventions.

Obstructive uropathy included patients with renal calculi, benign enlargement of the prostate, carcinoma

Table 1. Descriptive parameters in urosepsis patients at admission.

Variables	Mean (± SD)
Age (years)	53.62 (± 17.29)
Duration of ICU stay (days)	6.47 (± 6.69)
Duration of intubation (days)	1.69 (± 4.17)
Worst SOFA score	8.61 (± 3.66)
Temperature (°C)	38.34 (± 1.33)
Heart rate (beats per minute)	111.12 (± 19.72)
WBC (× $10^3/\mu L$)	18.48 (± 9.06)
Platelets (× $10^3/\mu L$)	152.45 (± 111.29)
Serum lactate (mmol/L)	4.44 (± 2.77)
Procalcitonin (ng/mL)	38.87 (± 60.79)
Prothrombin time (PT) (seconds)	12.90 (± 4.86)
INR (international normalizing ratio)	1.32 (± 0.47)
Activated partial thromboplastin time (apTT) (seconds)	35.98 (± 13.90)
Bilirubin (μmol/L)	22.43 (± 23.47)

urinary bladder, and uterus causing obstruction to the urinary flow. Nonobstructive uropathy included patients with pyelonephritis, emphysematous pyelonephritis, and chronic urinary tract infections including cystitis, renal, prostatic abscess, prostatic biopsy, and ureteric stents. Trauma included trauma to the urinary tract requiring suprapubic catheter insertion.

Whenever the variable count was less than 10, it marked as other bacteria or other antibiotics. Data were entered and analyzed using SPSS version 23. Categorical variables were reported using numbers (n) and percentages (%). Continuous variables are reported as mean± SD, and categorical variables were represented as frequency and percentage. The Kolmogorov-Smirnov test proved these variables to be normally distributed. Between-group comparisons were performed by using the chi-square test for categorical variables and the t-test for continuous variables. Differences were considered statistically significant at $p \leq 0.05$.

RESULTS

During the study period, 302 patients were admitted to the surgical intensive care unit (SICU) of our hospital, accounting for 26.31% of total

Table 2. Urosepsis and acute kidney injury, interventions, complications, and outcomes.

Variable		Frequency (n)	Percent (%)
Oliguria	Yes	152	50.3
Acute kidney injury	Yes	227	75.2
Acute renal failure stage 3	Yes	86	28.5
Renal ureteric stone	Yes	184	60.9
Complications of PCN	Septic shock	38	12.6
	Not functioning	5	1.7
Urological surgery	Endoscopic urological surgery	75	24.8
	Lithotripsy	2	0.7
	Nephrectomy	2	0.7
Severe sepsis septic shock	Severe sepsis	19	6.3
	Septic shock	283	93.7
Transfer or death	Died	8	2.6
	Survived	294	97.4

*PCN: percutaneous nephrostomy

Table 3. Microbiology and use of antibiotics in urosepsis patients.

Variables	Organism	Frequency	Percent
Blood culture	None	86	28.5
	E. coli	45	14.9
	E. coli (ESBL)	99	32.8
	K. pneumoniae	18	6
	P. aeruginosa	15	5
	E. faecalis	11	3.6
	Other bacteria*	28	9.3
Blood culture	Negative	86	28.5
	Positive	216	71.5
Urine culture	*E. coli*	78	25.8
	E. coli (ESBL)	109	36.1
	K. pneumoniae	31	10.3
	P. aeruginosa	26	8.6
	Candida species	21	7
	E. faecalis	15	5
	Other bacteria*	22	7.3
Sputum culture	None	299	99
	K. pneumoniae	1	0.3
	P. aeruginosa	1	0.3
	Candida species	1	0.3
Initial antibiotics	Meropenem	106	35.1
	Piperacillin+ Tazobactum	124	41.1
	Ceftriaxone	28	9.3
	Ciprofloxacin	15	5
	Ertapenem	12	4
	Other antibiotics*	17	5.6
Antibiotic change	Meropenem	106	35.1
	Piperacillin+ Tazobactum	68	22.5
	Ceftriaxone	25	8.3
	Ertapenem	24	7.9
	Other Antibiotics*	44	14.6

* In blood culture: "other bacteria" include *Proteus, Candida, E. coli* (MDRO), and methicillin sensitive *Staphylococcus* (MSSA)
* In urine culture: "other bacteria" include *Proteus, Enterococcus cloacae, E. coli, K. pneumoniae* (MDRO), and methicillin sensitive *Staphylococcus* (MSSA).
* In initial antibiotics: "other antibiotics" include Ciproflaxacin, Piperacillin+ Tazobactum, Meropenam+ vancomycin, and Ciprofloxacin+ Meropenem.
* In antibiotic change: "other antibiotics" include Colistin, Caspofungin, Amphotericin, Fluconazole, Ciprofloxacin, Piperacillin+ Tazobactum, Meropenam+ vancomycin, Ciprofloxacin+ meropenem, Cloxacillin, Ciprofloxacin, and Anidulafungin.

admissions to the SICU. Of these, 151 (50%) were male, and Arab patients (164/54.3) outnumbered non-Arab patients (138/45.7%), which included the Indian, Pakistani, Bengali, and East Asian population. The common etiology was obstructive uropathy (184/60.9%). A nonobstructive etiology was seen in 113 (37.4%) patients, and 5 (1.7%) patients had urosepsis in posttraumatic period.

The majority of patients (91/30.1%) did not have comorbidities. Among the comorbidities present, the majority of patients had both diabetes mellitus (DM) and hypertension (HTN) (56/18.5%). DM and HTN were also present in other patient groups. DM, HTN, and coronary artery disease were present in 41/13.6% patients, 27/8.9% patients had other comorbidities. "Other" included pregnancy, hyperparathyroidism, and trauma (including spinal injury, urinary bladder carcinoma, and uterine carcinoma). Twenty-five patients (8.3%) were bedridden with DM, HTN, or CVA (cerebrovascular accidents), 24 (7.9%) patients had only DM, 19 (6.3%) had only HTN, and 19 patients

(6.3%) had chronic urinary infections (UTI) due to renal calculi.

Table 1 shows the descriptive parameters. The mean age of our patients was 53 years, all of them had tachycardia (mean heart rate 111 beats per minute), leukocytosis (mean WBC 18 × 10^3 cells/μL), mean platelet count (152 × 10^3 platelets/μL), normal coagulation parameters (mean INR = 1.32, PT = 12.9 seconds and aPTT = 35.98 seconds), and mild elevation in serum bilirubin (mean 22.43 μmol/L).

Table 2 shows involvement of the renal system in urosepsis patients, interventions and its complications, urosepsis complicated by severe sepsis, and septic shock and its outcome. Acute kidney injury (AKI) occurred in 227 (75%) urosepsis patients and 86 (28%) patients progressed to chronic renal failure. The majority of patients (184/60.9%) had renal stones, and percutaneous nephrostomy (PCN) was performed in 116 (38.4%) patients. PCN progressed to septic shock in 38 (12.6%) patients, and in 5 (1.7%), PCN was nonfunctional. Endoscopic ureteric stenting was performed in 75 (24.8%) patients, lithotripsy and nephrectomy in 2 patients (1.7%) in each category.

The majority of our urosepsis patients had septic shock (223/93.7%). Eight patients died, giving a mortality rate of 2.6% in our urosepsis population (Table 2).

The most common urosepsis-causing bacteria were ESBL-(Extended-Spectrum Beta-Lactamase)-producing bacteria (109/36.1%), with a predominance of *E. coli* (ESBL) (95/31.5%) followed by *E. coli* 78/25.8% and "other bacteria" in urine cultures, including *Proteus mirabilis*, *Enterobacter cloacae*, multidrug-resistant (MDRO) *Klebsiella pneumoniae,* and methicillin-sensitive *Staphylococcus aureus* (Table 3).

Two hundred sixteen (71.5%) patients had bacteremia; the most common organism causing bacteremia was *E. coli* (ESBL) in 99 (32.8%) patients. "Other bacteria" in blood culture included *Proteus mirabilis*, *E. coli,* and multidrug-resistant (MDRO) *K. pneumoniae, Candida* species, and methicillin-sensitive *Staphylococcus aureus*. In blood and urine culture, ESBL-producing bacteria were represented by

Table 4. Urosepsis, bacteraemia, and significant variables.

			p value
Oliguria			
Bacteraemia Negative {Number (%)} Positive {Number (%)}	No 60(40) 90(60)	Yes 26(17.1) 126(82.9)	0.001*
Acute Kidney Injury (AKI)			
Bacteraemia Negative {Number (%)} Positive {Number (%)}	No 33(44) 42(56)	Yes 53(23.3) 174(76.7)	0.001*
Renal Stones			
Bacteraemia Negative {Number (%)} Positive {Number (%)}	No 39(33.1) 79(66.9)	Yes 47(25.5) 137(74.5)	0.1
Septic Shock			
Bacteraemia Negative {Number (%)} Positive {Number (%)}	No 16(84.2) 3(15.8)	Yes 70(24.70) 213(75.3)	0.001*
Acute respiratory distress syndrome (ARDS)			
Bacteraemia Negative {Number (%)} Positive {Number (%)}	No 38(44.2) 86(65.8)	Yes 70(22.48) 216(71.52)	0.002*

*Statistically significant

Table 5. Urosepsis patients gender and significant variables.

			p value
Oliguria			
Gender Male {Number (%)} Female {Number (%)}	No 63 (42) 87 (58)	Yes 88 (57.9) 64 (42.1)	0.004*
Acute Kidney injury (AKI)			
Gender Male {Number (%)} Female {Number (%)}	No 28 (37.3%) 47 (62.7)	Yes 123 (54.2) 104 (45.8)	0.08
Renal Stone			
Gender Male {Number (%)} Female {Number (%)}	No 58 (49.2) 60 (50.8)	Yes 93 (50.5) 91 (49.5)	0.45
Septic Shock			
Gender Male {Number (%)} Female {Number (%)}	No 9 (47.4) 10 (52.6)	Yes 142 (50.2) 141 (49.8)	0.5
Intubation			
Gender Male {Number (%)} Female {Number (%)}	No 95 (46.3) 110 (53.7)	Yes 56 (57.7) 41 (42.3)	0.042*
Acute Respiratory Distress syndrome (ARDS)			
Gender Male {Number (%)} Female {Number (%)}	No 35 (23.2) 51 (33.8)	Yes 116 (76.8) 100 (66.2)	0.02*
Bacteraemia			
Gender Male {Number (%)} Female {Number (%)}	No 36 (41.9) 50 (58.1)	Yes 115 (58.1) 101 (41.9)	0.027*

*Statistically significant

ESBL *E. coli*, ESBL *K. pneumoniae,* and ESBL *Enterobacter cloacae* (Table 3).

One patient each had growth of *K. pneumoniae*, *Pseudomonas aeruginosa,* and *Candida* in urine, blood, and sputum. The initial antibiotic of choice in our urosepsis patients was Piperacillin+Tazobactum followed by meropenem; "other first-line antibiotics" included meropenem and vancomycin, meropenem, and ciprofloxacin, Piperacillin+Tazobactum, and ciprofloxacin. Once culture and sensitivity information was available, antibiotics were changed accordingly. "Other antibiotics" used when antibiotics were changed included colistin, amphotericin, fluconazole, caspofungin, anidulafungin, ciprofloxacin, meropenem and vancomycin, meropenem and ciprofloxacin, Piperacillin+Tazobactum, and ciprofloxacin (Table 3). In 203 (67.21%) patients, antibiotics were changed. The common antibiotic changed was meropenem (141/46.7% patients), and in total, 54.6% patients' antibiotics were changed to carbapenems (Table 3).

Acute respiratory distress syndrome (ARDS) occurred in 250 (82.7%) patients, requiring intubation in 97 (32.1%) patients, 83 (27.5%) patients who required frusemide (Lasix) for diuresis. The majority of these patients had mild to moderate ARDS (123/40.73% and 101/33.45%, respectively). Only 26 (8.61%) patients had severe ARDS.

Patients with bacteremia (positive blood culture) had a significantly ($p < 0.001$) higher incidence of oliguria, acute kidney injury (AKI), septic shock, and ARDS (Table 4).

As our study population had an equal number of male and females. We compared the rate of organ

dysfunction between the genders and found that the oliguria, AKI, intubation, bacteremia, and ARDS were significantly higher in males ($p < 0.005$) (Table 5), but there was no significant difference ($p = 0.45$ and 0.5) in renal stones or the occurrence of septic shock between genders (Table 5).

DISCUSSION

The incidence of sepsis is rising, and if it progresses to septic shock, the mortality increases significantly.[1,9] A significant portion of sepsis originates from the genitourinary tract.[4,8] In our study, CAUs accounts for 26.5% of all admissions to the SICU.

Obstructive uropathy due to renal stones is a well-known cause of urosepsis, particularly from stone belts in the world. The southeastern USA is the stone belt, where a high incidence of renal stones is attributed to a high consumption of ice coffee, which contains a high mount of oxalate.[10] India, Pakistan, and southern China form the stone belt of Asia.[10] We found that the local Arabic population had a higher incidence of obstructive uropathy. Robertson described urolithiasis to be common in the Arabian Gulf population due to dietary and environmental factors.[11] The incidence of urosepsis was equal in both genders; in contrast, other authors found a predominance of the condition in females, owing to the shorter female urethra.[12]

The most common risk factor for urosepsis in our patients was the combination of DM and hypertension (HTN). According to Van Nieuwkoop et al., 80% of urosepsis was due to DM,[13] whereas Yamamichi et al. found carcinoma as the most common comorbidity of urosepsis.[12] Apart from DM and HTN, our patients with CVA (bedridden) were also at a higher risk for urosepsis.

In a multicenter study, it was found that patients older than 65 years had a 2.5 times greater risk of urosepsis; comparatively, our patients were younger.[12]

All our patients had a triad of urosepsis, loin pain, fever, and leukocytosis. Procalcitonin and lactate were high, indicating severe sepsis and septic shock. The average SOFA score was 8.6, indicating that at least two organs were dysfunctional in our patients. The average ICU (intensive care unit) length of stay of patients with urosepsis was 6 days. In spite of higher SOFA scores and procalcitonin and lactate levels, the ICU stay was comparable. According to a prospective study by Cardoso et al., the length of ICU stay for urosepsis was 7 days.[6]

The majority of patients had septic shock (93.7%) and were managed in the intensive care unit. In a study by Yamamichi et al., only 42.5% of urosepsis patients progressed to uroseptic shock.[12] Hsiao et al. described that the presence of AKI in urosepsis patients was a significant risk factor for the development of shock.[14] Patients with bacteremia are also at a higher risk of developing septic shock.[15] As the majority of our patients had AKI and bacteremia, which may have contributed to urosepsis progressing to septic shock, the incidence of septic shock was higher in our patient population.

The kidneys are frequently involved in urosepsis and sepsis patients. In urosepsis patients, AKI is due to the combined effects of sepsis and back pressure due to obstruction in the urinary tract. Post obstructive AKI can occur in up to 22% of urosepsis patients.[16] Post obstructive AKI mainly occurs due to renal vasoconstriction, and if obstruction is not relieved immediately, it causes renal scaring or fibrosis leading to chronic renal failure. In our urosepsis patients, 75.2% had AKI and 28.5% of them progressed to chronic renal failure. This incidence is higher than described in the literature, possibly due to double impact, post obstruction AKI, and sepsis. Recent studies showed that 20% of urosepsis patients had AKI.[14,16]

The optimal timing for relieving the obstruction in the urinary tract is not known, but if the patient is septic, it becomes an emergency. The majority (38%) of our patients had PCN. The advantages of PCN are its high success rate and ability to be performed under local anesthesia and the round-the-clock availability of the interventional radiologist. The disadvantages of PCN are that it is invasive and can cause the spread of sepsis and septic shock.[17] Up to 21% of patients can have PCN progress to sepsis and septic shock.[17,18] In our urosepsis population, 12.5% of patients developed post PCN septic shock. Twenty-five percent of our urosepsis patients underwent endoscopic stenting to relieve the obstruction. These were patients with severe coagulopathy who could not be positioned for PCN. The advantage of endoscopic stenting is that it is less invasive and carries a lower risk of severe hemorrhage.[19]

The most common bacterial cause of urosepsis in our study was *E. coli* (ESBL), with a predominance of ESBL bacteria in contrast to the literature where *E. coli* are the most common causative agents of urosepsis.[3,4,6] Seventy percent of our urosepsis patients' blood cultures were positive (had bacteremia). The rate of bacteremia was higher (71%) than the one described in the literature (41%).[20] The common initial antibiotics prescribed to our urosepsis patients were Tazocin® and meropenem. After culture and sensitivity results became available, in more than half of the urosepsis patients, antibiotics were changed to carbapenems.

The respiratory system was the second most common organ to be affected in our patients. Seventy percent of patients had acute respiratory distress syndrome (ARDS), the majority had mild to moderate ARDS, and 30% patients required endotracheal intubation. The remaining patients were managed well with non-invasive ventilation and negative fluid balance using frusemide (Lasix) diuresis. In our urosepsis patients, the rate of ARDS is higher (82%) than mentioned in the literature (7%).[21]

Our urosepsis patients with bacteremia had significantly higher AKI, ARDS, and septic shock. A previous report described complication rates of 51% to 64% in patients with septic shock and AKI, respectively.[22]

We found a significant difference between genders in urosepsis patients. Males had significantly higher AKI, ARDS, bacteremia, oliguria, and intubation rates.

There was no significant difference between genders in the occurrence of shock in our urosepsis patients. It is described in the literature that females are protected from the adverse effects of sepsis, organ dysfunction and septic shock by the female sex hormones, whereas males are susceptible due to diminished cell-mediated immunity.[23] Xin-Hua Qiang et al. compared mortality from various sepsis etiologies and concluded that among all sepsis types, urosepsis had a significantly better outcome (6%).[5] Fukunaga et al. also reported mortality of 6% in their urosepsis patients.[24] In our urosepsis population, the mortality was lower which, may be related to early source control, a change in the antibiotic, and supportive intensive care therapy.

The limitations of our study were its retrospective design and single-centered nature.

Future studies should be directed at evaluating the difference in outcome between obstructive urosepsis with image-guided (PCN) and endoscopic urological interventions. A prospective, multicenter study is required to evaluate the initial antibiotics prescribed for urosepsis with septic shock.

CONCLUSION

Our study demonstrates that CAUs frequently due to obstruction of urinary flow. Our patients had a higher incidence of bacteremia, which led to higher organ dysfunction and septic shock. The extended beta-lactamase bacteria were a common cause of urosepsis and hence required a change of antibiotics to carbapenems. Although there was no gender predominance in our patients, males suffered significantly higher rates of organ dysfunction. In spite of the higher incidence of bacteremia, organ dysfunction, and septic shock in our patient population, the outcome was better.

REFERENCES

1. Dreger NM, Degener S, Ahmad-Nejad P, Wöbker G, Roth S. Urosepsis – etiology, diagnosis, and treatment. *Dtsch Arztebl Int*. 2015;112:837 – 847.
2. Taha H, Raji SJ, Khallaf A, Abu HS, Mathew R, Rashed H, et al. Improving catheter associated urinary tract infection rates in the medical units. *BMJ Qual Improv Rep*. 2017;6:1 – 4.
3. Shaikh N, Umm-E-Amara, Hassan J, Qazi Z, Chanda A, Mahemood Z, et al. Urosepsis: flow is life, IntechOpen, DOI: 10.5772/intechopen.82262.
4. Liang X, Huang J, Xing M, He L, Zhu X, Weng Y, et al. Risk factors and outcomes of urosepsis in patients with calculous pyonephrosis receiving surgical intervention: a single center retrospective study. *BMC Anesthesiology*. 2019;61:2 – 8.
5. Qiang XH, Duyu T, Li YN, Zhou LX. Prognosis risk of urosepsis in critical care medicine: A prospective observational study. *Biomed Res Int*. 2016;2016:1 – 5.
6. Cardoso T, Ribeiro O, Costa-Pereira A, Carneiro A. A SACiUCI Study Group. Community-acquired

and healthcare-related urosepsis: a multicenter prospective study. *Crit Care*. 2008;12(Suppl 2):P8.

7. Bonkat G, Cai T, Veeratterapillay R, Bruyère F, Bartoletti R, Pilatz A, et al. Management of Urosepsis in 2018. *European Urology Focus*. 2019;4:5 – 9.
8. Rhodes A, Evans LE, Alhazzani W, Levy MM, Antonelli M, Ferrer R, et al. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock: 2016. *Intensive Care Med*. 2017;43:304 – 377.
9. Gitz Holler J, Jensen HK, Henriksen DP, Rasmussen LM, Mikkelsen S, Pedersen C, et al. Etiology of shock in the emergency department. *Shock*. 2019;51:60 – 67.
10. Fontenelle LF, Sarti TD. Kidney stones. *Am Fam Physician*. 2019;99:490 – 496.
11. Robertson WG. Stone formation in Middle Eastern Gulf States: A review. *AJU*. 2012;10:265 – 272.
12. Yamamichi F, Shigemura K, Kitagawa K, Toki Matsu I, Arakawa S, Fujisawa M. Shock due to urosepsis: A multicentre study. *Can Urol Assoc*. 2017;11: e105 – e109.
13. Van Nieuwkoop C, Bonten TN, Van't Wout JW, Kuijper EJ, Groenevld GH, Becker MJ, et al. Procalcitonin reflects bacteraemia and bacterial overload in urosepsis syndrome: A prospective observational study. *Critical Care*. 2010;14:R206.
14. Hsiao CY, Yang HY, Chang CH, Lin HL, Wu CY, Hsiao MC, et al. Risk factors for development of septic shock in patients with urinary tract infection. *Biomed Res Int*. 2015;2015:1 – 7.
15. Jones GR, Lowes JA. The systemic inflammatory response as predictor of bacteraemia and outcome from sepsis. *QJM*. 1996;89:5 – 15.
16. Hamdi A, Hajage D, Glabeke EV, Belenfant X, Vincent F, Gonzalez F, et al. Severe post-renal acute kidney injury, post-obstructive diuresis and renal recovery. *BJUI*. 2012;110:1027 – 1034.
17. Li AC, Regalado SP. Emergent PERCUTANEOUS nephrostomy for diagnosis and management of pyonephrosis. *Semin Intervent Radiol*. 2012;29: 218 – 225.
18. Hsu CS, Wang CJ, Chang CH, Tsai PC, Chen HW, Su YC. Emergency percutaneous nephrostomy versus emergency percutaneous nephrolithotomy in patients with sepsis associated with large uretero-pelvic junction stone impaction: a randomized controlled trial. *Int Braz J Urol*. 2017;43:481 – 488.
19. www.uptodate.com/content/placement-and-management of indwelling-urethral-stents/down loaded on 10th Sep 2019.
20. Chin BS, Kim JM. Risk factors for all causes in hospital mortality among Korean bacteraemia in urinary tract infection patients. *Archives Gerontology and Geriatric*. 2010;52:50 – 55.
21. Kim WY, Hong SB. Sepsis and acute respiratory distress syndrome. Recent Update. *Tuberc Respir Dis (Seoul)*. 2016;79:53 – 57.
22. Bilgili B, Haliloglu M, Cinel I. Sepsis and acute injury kidney injury. *Turk J Anesthesiol Reanim*. 2014;42:294 – 301.
23. Angele MK, Pratschke S, Hubbard WJ, Chaudhary IH. Gender differences in sepsis. *Cardiological and immunological aspects. Virulence*. 2014;5:12 – 19.
24. Fukunaga A, KawakitaIn M. Risk factors for mortality in patients with urosepsis. *European Urology Supplements*. 2017;16:e129.