International Journal of Current Advanced Research
ISSN: O: 2319-6475, ISSN: P: 2319-6505, Impact Factor: SJIF: 5.995
Available Online at www.journalijcar.org
Volume 7; Issue 3(D); March 2018; Page No. 10671-10675
DOI: http://dx.doi.org/10.24327/ijcar.2018.10675.1820

Review Article

URINARY TRACT INFECTION: A REVIEW

Rachana L. Patnayak., Dnyanesh B. Amle* and P. K. Khodiar

Department of Biochemistry, Pt. J. N. M. Medical College, Raipur, C.G

A R T I C L E I N F O

Article History:

Received 11th December, 2017
Received in revised form 21st January, 2018 Accepted 06th February, 2018
Published online 28th March, 2018

Key words:

Urinary tract infections, UPEC, Innate immunity, biomarkers, antimicrobial resistance

A B S T R A C T

Urinary tract infections have become serious health issue globally, adding economic burden to the society. The infection is observed in outpatients as well as in hospitalized patients and is more common in women than in men. Causative organisms cover a range of microbiota where, Uropathogenic *Escherichia coli* (UPEC), *Klebsiella* species and *Proteus mirabilis* are the most common isolates. In diabetic patients, the case is even more serious. In pregnant women UTI comes with lot of complications. Urinary tract infection is guarded by innate immunity. Toll- like receptors (TLRs) recognizes and mobilizes immune responses of the uroepithelial cells. TLR4 on activation undergoes a pathway that results in production of Antimicrobial peptides (AMPs). These mechanisms help to suppress UTI. The incidence of UTI goes proportionally with their age. In order to distinguish between cystitis and pyelonephritis biomarkers are employed. Increasing antimicrobial resistance to pathogenic species have become problem in carefully diagnosing the infection. This article overviews the epidemiology, common etiologies, novel diagnosis, evolution of antibiotic resistance patterns and preventive measures of the infection. The current management practises highlights to reduce the use of unnecessary indwelling catheters and deciphering proper awareness programs in the community relating to the infection.

INTRODUCTION

Urinary tract infections (UTIs) are one of the most common infections observed in outpatients as well as hospitalised patients. It is estimated that around 150 million people are affected each year globally (Stamm and Norrby, 2001). Out of hospital acquired infections, UTI accounts for 35% of nosocomial infections, and it is the second most common cause of bacteraemia in hospitalised patients (Akhtar, 2000). UTIs are a significant cause of morbidity in infant boys, old men and females of all ages (Flores-Mireles *et al*., 2015).The infection is more frequently caused by Gram- negative bacteria (GNB), especially *Escherichia coli* and Klebsiella species, as compared to Gram- positive bacteria (GPB) and fungi.

Urinary tract infection may affect lower urinary tract, characterised by dysuria, micturition frequency and urinary retention; and is called cystitis or bladder infection. When upper urinary tract is affected, it is called kidney infection or pyelonephritis, and it describes clinical syndromes such as fever and flank pain often associated with dysuria, urgency and frequency (Nielubowicz and Mobley, 2010). Clinically, UTIs are categorised as complicated or uncomplicated. Uncomplicated infections affect otherwise healthy individuals and are most commonly caused by uropathogenic *E.coli*, whereas complicated UTIs are defined as infections with underlying difficulties such as urinary tract abnormality and catheterization and are commonly caused by species such as *Proteus mirabilis* (Nielubowicz and Mobley, 2010).

Corresponding author:* **Dnyanesh B. Amle
Department of Biochemistry, Pt. J. N. M. Medical College, Raipur, C.G

Epidemiology of Urinary tract infection

Incidence of UTI is higher in women than in men. Pathogenesis of UTI in women is mainly due to their anatomical structure. Fairly short and straight urethra and its close association with anus provides platform for ascendance of bacteria in the urinary tract. The fecal- perineal- urethral hypothesis explains the cause of infections by enteric bacteria, indicating that *E. coli* strains residing in the rectal flora serve as a reservoir of UTIs (Yamamoto *et al*., 1997). In women, incidence range among age grouped 20- 40 years is 25%- 30%, while in older women, above 60 years of age, it ranges from 4- 43% (Mittal *et al*., 2017). The infection is more frequent among women in age group of 18- 29 years, when women are more likely to initiate sexual activity. Vaginal intercourse frequency is subjected to be the major risk factor in this group. Uncomplicated UTI has been nicknamed as 'honeymoon cystitis'.

Microbiology of the infection

A range of population of bacteria can cause urinary tract infection. However, Gram negative species and uropathogenic *E. coli* (UPEC) remains the soul causative organism. 75 - 95% of uncomplicated UTI are caused by Gram negative pathogens,

mainly UPEC, *Klebsiella pneumonia*, *Staphylococcus saprophyticus*, *Enterococcus species* and group B streptococcus. For complicated UTI, the order of prevalence of pathogens is almost common, that is UPEC is followed by *Enterococcus spp.*, *K. pneumonia* and *Candida spp*. (Flores-Mireles *et al*., 2015; Kline and Lewis, 2016) In an observational study of community acquired urinary Tract Infection (CA- UTI), conducted in south India, *E. coli* (66.9%) was the most common organism causing CA-UTI with extended spectrum beta lactamase (ESBL) resistance seen in nearly two- thirds of the cases (42.2%) (Eshwarappa *et al*., 2011).

Uropathogenic *E. coli* are certain pathotypes of extraintestinal pathogenic *E. coli*. UPEC strains are different from the commensal *E. coli*. The former possess an extragenetic material, and often has pathogenecity- associated islands (PAI), which encodes genes that contributes to bacterial pathogenesis (Mobley *et al*., 2009). UPEC is transmitted by sexual contact suggesting increased risk of recurrence in the infection. Type 1 fimbriae are proven virulence factors for *E. coli* in the urinary tract. UPEC express many types of fimbriae with distinguished receptor affinity. *P. fimbriae* is associated with pyelonephritis incidence with UPEC isolates. *P. fimbriae* mediates binding to the glob series of αGal(1-4) βGal-containing glycolipids and has a specific role as a colonization factor in the urinary tract (Buckles *et al*., 2004). According to a review study of patients who presented community acquired UTI due to CtXm-m-15-producing *E.coli* in New Zealand between 2004 and 2006, 10 out of 11 patients reported recent travel to India. The prevalence of *E. coli* strains with CtXm-m-mediated resistance is high in India (Freeman *et al*., 2008). This suggests possible transmission of UPEC via food or water or through person to person contact during their travel. There have been studies which provide strong support for the role of food reservoirs or food-borne transmission in disseminating UPEC, which further causes UTI (Vincent *et al*., 2010). UPEC forms a bio-film which protects them from various stresses.

Proteus mirabilis is most often isolated from human intestinal tract as part of normal human intestinal flora. *P. mirabilis* is capable of causing symptomatic infections of the urinary tract and is found in asymptomatic bacteriuria cases, particularly in elderly and patients with type 2 diabetes (Schaffer and Pearson, 2015). *P. mirabilis* produces urease as an inducible virulence factor, which hydrolyses urea into ammonia and carbon- dioxide, which results in an increase in local pH. The alkaline pH guides the precipitation of calcium and magnesium ions and the formation of urinary stones composed of struvite and apatite. In *P. mirabilis* urease is coded by ureDABCEFG operon (Island and Mobley, 1995). These stones can block the flow of urine and cause tissue damage. The precipitated minerals may induce the formation of crystalline bio-films and eventually block urinary flow through the catheter. The stones protect bacteria against antibiotics and acts as a focal point for other species of bacteria to establish. Although, fimbriae of *P. mirabilis* contributes as virulent factor in UTI, their characteristic expression is yet unknown. *P. Mirabilis* is highly resistant towards range of antibiotics, particularly tetracycline (Schaffer and Pearson, 2015).

Urinary Tract infections by fungus is usually caused by Candida spp., *Cryptococcus neoformans*, Aspergillus species and the endemic mycoses are also believed to cause UTI. A colony count of 10,000- 15,000 cfu/ml is the suggested value for the diagnosis of fungal UTI.

Etiology

The bacterial etiology for different groups of UTI patients is well defined. The etiology is also affected by the latent factors in host such as age, sex, diabetes, catheterisation or spinal cord injury.

Diabetes

Diabetes mellitus has long term effect on genitourinary system. These effects act as predisposing factors for the growth of bacterial UTI among diabetes mellitus patients (Patterson and Andriole, 1997). The spectrum of UTIs in these patients ranges from asymptomatic bacteriuria (ASB) to lower UTI (cystitis), pyelonephritis and severe urosepsis. High renal parenchymal glucose levels create favourable environment for the growth and multiplication of microorganisms, which may further lead to pyelonephritis and renal complications (Nitzan *et al*., 2015). In a case study of 155 consecutive Type 1 (n=102) and type2 (n=53) diabetic individuals and 128 healthy controls, it was observed that patients with diabetic mellitus had 10 and 3 fold increased risk of UTI and renal scarring respectively (Goswami *et al*., 2001). The incidence of UTI in diabetic patients increases with increase in age. In a prospective single centre study conducted in Guntur, India, a total of 100 patients with type 2 diabetes mellitus aged over 60 years with suggestive symptoms of UTI were examined. 43% of type 2 diabetic patients had bacteriuria and female patients with diabetes history of >15 years and diabetes complications had increased predominance for developing UTI (Sharma *et al*., 2017).

Management of recurrent episodes of UTI is similar to non-diabetic patients (Nitzan *et al*., 2015). In diabetic patients UTI are more grievous, caused by more resistant pathogens and are associated with worse outcomes than in patients with no diabetes.

Pregnancy

Urinary tract infection is often associated with pregnancy. Physiological, hormonal and anatomical change during pregnancy leads to expression of different types of receptors in the urinary tract which enhances the specificity of the infection. Prevalence of ASB is high in pregnancy. In a study conducted on pregnant women with suspected UTIs, 417 urine samples were collected from women who were at different stages of their pregnancy. Overall incidence of UTI in this study was found to be 49.4%. If not treated, 20%- 30% of ASB will lead to acute pyelonephritis (Manjula *et al*., 2013).

Catheter associated UTI

The centre for disease control and prevention (CDC) defines catheter associated UTI for those patients who have an indwelling catheter in place for 48 hours or more and symptoms such as fever or chills, new onset of burning pain, urgency or frequency if not catheterised at that point of time, change in urine character, flank or suprapubic pain or tenderness or change or decrease in mental or functional status in patients (Sangamithra *et al*., 2017). Catheter associated urinary tract infections are the most common nosocomial infections which constitutes 30% - 40% of all hospital acquired infections (Vinoth *et al*., 2017). Nosocomial infections and hospital- acquired infections are principal cause

of morbidity and mortality in healthcare settings especially in intensive care units (ICU) and therefore increasing patient's length of stay in acute care facilities (Hanumantha and Pilli, 2016). Biofilm formation along the catheter surface is the supreme cause of bacteriuria. The risk factors associated with catheter associated UTIs includes female sex, old age, prolonged catheterisation, impaired immunity, diabetes, renal dysfunction, severity of illness, insertion of the catheter outside of the operating room, inadequate professional training of the person who inserts the catheter, incontinence and the inpatients in the orthopaedic and the neurology departments (Trautner and Darouiche, 2004). The organisms colonizing intravascular device, access the device through four different routes of infection- (1) invasion of the skin insertion site, (2) contamination of the catheter hub, (3) hematogenous spread from a distant site of infection, or (4) infusion of contaminated fluid through the device (Trautner and Darouiche, 2004). Multiple studies have shown that 21% to 55.7% of urinary catheters are placed in patients who do not have an appropriate indication, and therefore may not even need a catheter. Therefore avoiding unnecessary urinary catheter use is the most important strategy in prevention of catheter associated UTI (Meddings *et al*., 2014).

Immune responses during Urinary Tract Infection

The infection develops when bacteria overcomes the barriers like urine flow, mucus production, and uroepithelial coating with Tamm- Horsfall protein (THP) or antimicrobial peptides (AMPs). The antibacterial defence of the urinary tract relies almost entirely on innate immunity. In urinary tract, alteration in the immune mechanisms leads to uroepithelial tissue destruction, parenchymal scarring or overwhelming infection. If uropathogens rupture the uroepithelium toll- like receptors (TLR) recognise and mobilize the immune responses of the bladder and kidney epithelial cells. TLRs are trans-membrane proteins with multiple leucine rich repeats that recognise highly conserved structural motifs known as pathogen-associated microbial patterns (PAMPs), exclusively released by microbial pathogens. In the urinary tract, the common TLRs includes TLR2 (recognises bacterial lipoteichoic acid or lipoprotein), TLR3 (recognises dsRNA), TLR9 recognises unmethylated DNA of bacteria and viruses) and TLR11 (recognises parasites).among the TLRs expressed, TLR4 is the most studied one (Spencer *et al*., 2014).

Patients with asymptomatic bacteriuria exhibited decrease in TLR4 expression. On activation TLR4 stimulates changes in renal cyclooxygenase-2 which is responsible for the formation of inflammatory prostanoids that participate in the regulation of bladder mucous barrier and cytoprotection. This, further results in pro- inflammatory pathways. As a result chemokines are released and leucocytes are recruited to the site of infection. There is an increase in production of AMPs by uroepithelium and circulating leucocytes. Together, all these mechanisms help to suppress the UTI (Spencer *et al*., 2014).

Biomarkers for UTI

To diagnose UTI we rely on clinical presentations. In elderly and immunocompromised individuals, clinical presentation often poses challenge to the diagnosis procedure. Multiple studies have shown that clinical characteristics alone prove to be scanty in localising the site of infection in the urinary tract. To differentiate between the lower urinary tract infection and pyelonephritis, biomarkers such as erythrocyte sedimentation rate (ESR), C reactive protein (CPR) and leucocyte counting is used (Nanda and Mehta, 2009).

Procalcitonin (PCT), a propeptide of calcitonin has been used in the diagnostics and assessment of severity of UTI. In healthy people after the third day of life serum procalcitonin level is below 0.5 ng/ml and is produced in the C cells of thyroid gland. A severe bacterial infection leads to abundant secretion of procalcitonin by the monocyte macrophage system. PCT has proved to be good marker to diagnose renal parenchymal damage in the paediatric group (Nanda and Mehta, 2009; Zagajewska and Nowicki, 2017).

Interleukin 6 (IL-6) and Interleukin 8 (IL-8) have been studied to diagnose UTI in adults and to differentiate between pyelonephritis and lower UTI in children. Urinary IL-8 has been suggested as a biomarker for UTI in catheterised postoperative patients (Nanda and Mehta, 2009).

Antibiotic resistance pattern

Urinary tract infections are becoming difficult to treat owing to the antimicrobial resistance shown by UTI pathogens. Studies clearly reveal the increasing patterns of antibiotic resistance in uropathogens of both community- associated UTI and nosocomially acquired UTIs. Multidrug resistant uropathogenic organisms have become threat to public health. Since antibiotic resistance mechanisms are achieved via horizontal gene transfer, bacteria can acquire resistance to multiple antibiotics.

A study was conducted to determine patterns of resistance amongst community acquired (CA) uropathogens in India to help establish local guidelines on treatment of CA- UTI. According to this study, high levels of Extended- Spectrum Beta-Lactamase (ESBL) producers among gram negative CA-uropathogens were seen in India. It also concluded the alarming rate of resistance to ciprofloxacin, co-trimoxazole, amoxicillin (Kothari and Sagar, 2008). In another study conducted in North India, which comprised of 2941 urine samples, *E.coli* accounted for 56.8% of uropathogens and 91 of 119 (76.5%) *E.coli* isolates were multi drug resistant (MDR). The isolates showed high levels of resistance to ampicillin (88.4%), amoxicillin- clavulanic acid (74.4%), norfloxacin (74.2%), cefuroxime (72.2%), Ceftriaxone (71.4%) and cotrimoxazole (64.2%). The study interpreted the high rate of resistance in the inpatients due to increased usage of cephalosporins in the hospital for empirical therapy (Niranjan and Malini, 2014). *E.coli* and Klebsiella isolates were observed to be highly resistant against nitrofurantoin (80% and 76% resistant respectively) in a study at tertiary care hospital in north India (Mohammed *et al*., 2007).

Antibiotic resistance is becoming a nuisance to public health threatening the lives of hospitalized individuals as well as those with chronic conditions and thereby adding considerably to health care costs. Hence it is the need of the hour to formulate strict antibiotics prescription policy in our country.

Management and treatment regimens

Urinary tract infections results in economic and public health burdens markedly affect the life quality of afflicted individuals (Flores-Mireles *et al*., 2015). Ideally, treatment with antimicrobial agents, eradicate uropathogens bringing about resolution in clinical signs, having few adverse effects but prevents re-infection. Currently, treatments with antibiotics

such as trimethoprim, sulfamethoxazole, cotrimoxazole, ciprofloxacin and ampicillin are amongst most commonly recommended for UTIs (Flores-Mireles *et al*., 2015). Phenazopyridine, a urinary tract antiseptic and analgesic, provides symptomatic relief from the pain, burning, frequency and urgency associated with UTI during the first 24- 48 hours of therapy and is recommended as safe treatment during pregnancy (Hotchandani and Aggrawal, 2012). However increasing rates in the resistance patterns of antibiotics has gained ground causing the burden in society economically and functionally. The single most effective intervention to prevent catheter associated UTI is by avoiding the use of indwelling catheter (Meddings *et al*., 2014). Ultimate prevention of the infection requires technical advances in catheter materials which prevents biofilm formation (Nicolle, 2014). Incidence of UTI can be reduced in community by featuring proper awareness programmes related to the infection in the society and maintain proper hygiene.

Conflict of interest: Authors declare that they have no conflict of interest.

References

1. Stamm, W.E. and Norrby, S.R. 2001. Urinary tract infections: disease panorama and challenges. *J Infect Dis.,* 183(Suppl 1): S1-S4.
2. Akhtar, N. 2000. Urinary tract bacterial pathogens; their antimicrobial susceptibility patterns at Bhawalpur. The Professional., 7(2): 131-137.
3. Flores-Mireles, A.L., Walker, J.N., Caparon, M. and Hultgren, S.J. 2015. Urinary tract infections: epidemiology, mechanism of infection and and treatment options. *Nat. Rev. Microbiol*., 13(5): 269-284.
4. Nielubowicz, G.R. and Mobley, H.L.T. 2010. Host-pathogen interactions in urinary tract infections. *Nat. Rev. Urol*., 7(8): 430-441.
5. Yamamoto, S., Tsukamoto, T., Terai. A., Kurazono, H., Takeda, Y. and Yoshida, O. 1997. Genetic evidence supporting the fecal-perineal-urethral hypothesis in cystitis caused by Escherichia coli. *J. Urol*., 157(3): 1127-1129.
6. Mittal, S., Kumar, A. and Sayal, P. Recurrent urinary tract infections and management. urinary tract infections & treatment, pp.1-14.
7. Kline, K.A. and Lewis, A.L. 2016. Gram-positive uropathogens, polymicrobial urinary tract infection, and the emerging microbiota of the urinary tract. *Microbiol. Spectr*., 4(2): 1-54.
8. Eshwarappa, M., Dosegowda, R., Vrithmani Aprameya, I., Khan, M.W., Shiva Kumar, P. and Kempegowda, P. 2011. Clinico-microbiological profile of urinary tract infection in south India. *Indian J. Nephrol.,* 21(1): 31-36.
9. Mobley, H.L.T., Donnenberg, M.S. and Hagan, E.C. 2009. Uropathogenic Escherichia coli. EcoSal. Plus., 3(2).
10. Buckles, E.L., Bahrani-Mougeot, F.K., Molina, A., Lockatell, C.V., Johnson, D.E., Drachberg, C.B., Burland, V., Blattner, F.R. and Donnenberg, M. 2004. Identification and characterization of a novel uropathogenic Escherichia coli-associated fimbrial gene cluster. *Infect. Immun*., 72(7): 3890-3901.
11. Freeman, J.T., McBride, S.J., Heffernan. H., Bathqate, T., Pope, C. and Ellis-Peqler, R.B. 2008. Community-onset genitourinary tract infection due to CTX-M-15-producing Escherichia coli among travelers to the Indian subcontinent in New Zealand. *Clin. Infect. Dis*., 47(5): 689-692.
12. Vincent, C., Boerlin, P., Daignault, D., Dazosis, C.M., Dutil, L., Galanakis, C., Reid-Smith, R.J., TelliS, P.P., Ziebell, K. and Manges, A.R. 2010. Food reservoir for Escherichia coli causing urinary tract infections. *Emerg. Infect. Dis.,* 16(1): 88-95.
13. Schaffer, J.N. and Pearson, M.M. 2015. Proteus mirabilis and urinary tract infections. *Microbiol. Spectr.,* 3(5): 1-43.
14. Island, M.D. and Mobley, H.L. 1995. Proteus mirabilis urease: operon fusion and linker insertion analysis of ure gene organization, regulation, and function. *J. Bacteriol*., 19(177): 5653-5660.
15. Patterson, J.E. and Andriole, V.T. 1997. Bacterial urinary tract infections in diabetes. Infect. *Dis. Clin. North Am.,* 11(3): 735-750.
16. Nitzan, O., Elias, M., Chazan, B. and Saliba, W. 2015. Urinary tract infections in patients with type 2 diabetes mellitus: review of prevalence, diagnosis, and management. *Diabetes Metab. Syndr. and Obes*.,8: 129-136.
17. Goswami, R., Bal, C., Tejaswi, S. Punjabi, G. Kapil. A. and Kochupillai, N. 2001. Prevalence of urinary tract infection and renal scars in patients with diabetes mellitus. *Diabetes Res. Clin. Pract.* 53(3): 181-186.
18. Sharma, S., Govind, B. and Naidu, S.K. Kinjarapu S, Rasool M. 2017. Clinical and laboratory profile of urinary tract infections in type 2 diabetics aged over 60 Years. *J. Clin. Diagn. Res*., 11(4): 25-28.
19. Manjula, N., Math, G.C., Patil, S.A., Gaddad, S.M. and Shivannavar, C.T. 2013. Incidence of urinary tract infections and its aetiological agents among pregnant women in Karnataka region. *Adv. Microbiol*., 3(6): 473-478.
20. Spencer, J.D., Schwaderer, A.L., Becknel, B., Watson, J. and Hains, D.S. 2014. The innate immune response during urinary tract infection and pyelonephritis. *Pediatr. Nephrol*., 29(7): 1139-1149.
21. Nanda, N. and Mehta, M.J. 2009. Novel biomarkers for the diagnosis of urinary tract infection-A systematic review. *Biomark. Insights*., 4: 111- 121.
22. Zagajewska, A.M. and Nowicki, M. 2017. New markers of urinary tract infection. *Clin. Chim. Acta*., 471: 286-291.
23. Kothari, A. and Sagar, V. 2008. Antibiotic resistance in pathogens causing community-acquired urinary tract infections in India: a multicenter study. *J. Infect. Dev. Ctries*., 2(5): 354-358.
24. Niranjan, V. and Malini, A. 2014. Antimicrobial resistance pattern in Escherichia coli causing urinary tract infection among inpatients. *Indian J. Med. Res*., 139(6): 945- 948.
25. Mohammed, A., Mohammed, S. and Asad, K.U. 2007. Etiology and antibiotic resistance patterns of community-acquired urinary tract infections in JNMC hospital Aligarh, India. A*nn. Clin. Microbiol. Antimicrob.,* 6(4): 4-7.

26. Sangamithra, V., Praveen, S.S. and Manonmoney. 2007. Incidence of catheter associated urinary tract infection in medical ICU in a tertiary care hospital. *Int. J. Curr. Microbiol. Appl. Sci.,* 6(4): 662-669.
27. Vinoth, M., Prabagaravarthanan, R. and Bhaskar, M. 2017. Prevalence of microorganisms causing catheter associated urinary tract infections (CAUTI) among catheterised patients admitted in a tertiary care hospital. *Int. J. Res. Med. Sci*., 5(6): 2367-2372.
28. Hanumantha, S. and Pilli, H.P. 2016. Catheter associated urinary tract infection (CAUTI)– incidence and microbiological profile in a tertiary care hospital in Andhra Pradesh. *Indian J. Microbiol. Res.,*3(4): 454-457.
29. Trautner, B.W. and Darouiche, R.O. 2004. Catheter-associated infections: pathogenesis affects prevention. *Arch. Intern. Med*., 164(8): 842-850.
30. Meddings, J., Rogers, M.A.M., Krein, S.L.K., Fakeih, M.G., Olmsted, R.N. and Saint, S. 2014. Reducing unnecessary urinarycatheter use and other strategies to prevent catheter-associated urinary tract infection: an integrative review. *BMJ Qual. Saf*., 23(4): 277-289.
31. Hotchandani, R. and Aggrawal, K. 2014. Urinary tract infections in women. *Indian J. Clin. Pract*., 23(4): 187-192.
32. Nicolle, L. 2014. Catheter associated urinary tract infections. Antimicrob. Resist. *Infect. Control*., 3(23): 1-8.

How to cite this article:

Rachana L. Patnayak *et al* (2018) 'Urinary Tract Infection: A Review', *International Journal of Current Advanced Research,* 07(3), pp. 10671-10675. DOI: http://dx.doi.org/10.24327/ijcar.2018.10675.1820

****