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REVIEW ARTICLE

# Urinary tract infection in renal transplant recipients

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## ABSTRACT

Urinary tract infection (UTI) is common in renal transplant recipients. Frequency of UTIs depend on many factors such as age, female gender, kidney function, co-morbidity, type and amount of immunosuppression, urological instrumentation and/or the follow-up period (short term or long term) after kidney transplantation. UTI may worsen graft and patient survival. A significant proportion of renal transplant recipients with UTIs may develop acute pyelonephritis (APN), which is an independent risk factor for deterioration of graft function. Renal transplant recipients with UTIs are often clinically asymptomatic as a consequence of immunosuppression. UTI, however, may progress to APN (particularly in the early post-transplant period), bacteraemia and the full blown picture of urosepsis. Strategies for long term prophylaxis and antimicrobial treatment of UTI in renal transplant recipients are discussed.

**Keywords** Immunosuppression, renal transplantation, urinary tract infection, uropathogens.

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## Introduction

While kidneys are the most frequently transplanted organs and renal transplantation is the preferred method for treating patients with end-stage renal disease, post-transplantation urinary tract infection (UTI) is still a source of morbidity and graft failure. The importance of this issue is further underscored by the fact that UTI is the most common infection in renal transplant recipients [1–3], ranging from 6% to 86% [4–7] and accounting for approximately 40–50% of all infectious complications. Renal transplant recipients develop UTIs more frequently than the general population [8–10]. The frequency of UTIs depends on many factors such as age and female gender of renal transplant recipients, kidney function and co-morbidities, immunosuppressive protocol or the follow-up period (short term or long term after kidney transplantation). For example, Takai *et al*. [2] reported that 26% of 363 renal transplant patients developed at least one UTI over a mean period of 4 years, while Martinez-Marcos *et al*. [11] found that 63% of 50 consecutive renal transplant recipients followed over a period of 1 year developed UTIs. In the study of Pellé *et al*. [12], most of the patients (75·1%) had at least one episode of UTI during the 5 years of follow-up. The considerable variation in the reported incidences of post-transplant UTI might be due to local outbreaks, differing resistance rates, centre-specific antibiotic strategies, but also due to various definitions and diagnostic criteria. UTIs occur more often in female than in male renal transplant recipients. Most of the UTIs (74%) occurred during the first year after kidney transplantation (81·9% within the first 3 months after surgery). During the second year the proportion of UTIs decreased to 35·7% and further to 21·5% during the fourth year following kidney transplantation [12]. Transplantation of kidneys from deceased donors increases the incidence of postoperative UTI. The use of organs from living donors is likely to lead to lower rates of UTI, probably due to shorter periods of cold ischaemia, less severe ischaemic-reperfusion injury and a lower rate of delayed graft function [13].

## Causative microorganisms

Gram-negative bacterial infections account for more than 70% of UTI and *Escherichia coli* (*E. coli*) is the most common clinical isolate in patients with UTIs, not only in the general population but also after kidney transplantation [14]. *Pseudomonas aeruginosa*, *coagulase-negative Staphylococci* or *Enterobacter* cloacae were frequently detected in the urine within the first 3–5 weeks following kidney transplantation, while *Enterococcus* species and *E. coli* were predominantly detected during the first 6 to 12 weeks, respectively, after surgery. These microorganisms are frequently found to be resistant to trimethoprim-sulfamethoxazole (TMP-SMZ): *E. coli* in 84%, *Enterobacter cloacae* in 67%, coagulase-negative *Staphylococcus* in 86% and *Enterococcus* species in 46% [12], explaining why TMP-SMZ prophylaxis for *Pneumocystis jirovecii* infection does not prevent UTIs in the first 6 months after kidney transplantation. Chuang *et al*. [7] analysed the causative uropathogens in 213 of 500 (43%) renal transplant recipients who developed UTIs over a mean follow-up period of 42 months. In this study, the most common pathogens isolated in urine culture

were *E. coli* (29%), *Enterococcus* (24%), *Staphylococcus* (12%) and *Klebsiella* (10%). In a study from Iran, *E. coli* was the isolated uropathogenic microorganism in 53·3% of renal transplant recipients with UTIs [15]. In a study from Turkey, *E. coli* was the uropathogen in 61·3% of the isolates obtained from post-transplant UTI patients [16]. In contrast, *E. coli* is reported to be the cause of 80–90% of UTIs in the general population [17,18]. In the study of Dantas *et al*. [19], *Enterobacter cloacae* was responsible for 30·4% of post-transplant UTIs with multiple resistance to antibiotics. Also, low virulence bacteria otherwise non-pathogenic in healthy hosts, have been implicated in post-transplant UTI. Interestingly, by assessing a novel culture-independent technique Domann *et al*. found the existence of a variety of microbes emerge during UTI in renal transplant recipients such as *Anaerococcus lactolyticus*, *Dialister invisus* and *Fusobacterium nucleatum* indicating that UTI could be a polymicrobial infection dominated by a specific well known and thereby highly virulent bacterial strain [20].

Rice and co-workers [14] confirmed the association between upper UTI by virulent *E. coli* and acute allograft injury. Virulent *E. coli* isolated from the urine of renal transplant patients frequently expressed P fimbriae. Acute pyelonephritis (APN) occurred in 40% of renal transplant patients with UTIs, 82% of which had acute allograft injury defined as an increase in serum creatinine ≥ 20%. In addition, 62% of *E. coli* isolates that expressed P fimbriae were associated with acute allograft injury. *E. coli* that express P fimbriae decrease IgA transport into the urine [21] resulting in a reduction of local host defence. Finally, fimbriated *E. coli* may invade the uroepithelium enabling the development of pathogenicity islands within the urinary tract [22].

## Acute pyelonephritis versus asymptomatic bacteriuria

Considerable debate exists about the impact of UTI on long term graft survival. While several studies did not validate an association between lower urinary tract infection and graft and patient survival, a major retrospective analysis indicated that UTIs that occur at least 6 months after kidney transplantation were associated with worse long term patient survival [23]. In the study of Pellé *et al*. [12], 18·7% of the renal transplant recipients with UTIs went on to develop APN, which was calculated to be an independent risk factor for deterioration of renal graft function. Risk factors for APN in the graft were female gender (64% of the patients), recurrent UTIs ($P < 0{\cdot}0001$) and acute rejection episodes ($P < 0{\cdot}003$). Mean serum creatinine values were significantly greater ($2{\cdot}01 \pm 0{\cdot}42$ mg $dL^{-1}$; $P < 0{\cdot}01$) one year after renal transplantation in patients with APN than in patients without UTI ($1{\cdot}59 \pm 0{\cdot}51$ mg $dL^{-1}$) or patients with UTI but without APN ($1{\cdot}60 \pm 0{\cdot}63$ mg $dL^{-1}$). One year after kidney transplantation creatinine clearance of these three groups of patients were $39{\cdot}5 \pm 15{\cdot}5$, $56{\cdot}4 \pm 20{\cdot}5$ and $54{\cdot}6 \pm 21{\cdot}7$ mL $min^{-1}$ $1{\cdot}73$ $m^2$, respectively. Four years after renal transplantation the mean creatinine clearance value for patients with APN was about 50% lower than that found for patients who did not develop UTI or patients who exhibited uncomplicated UTIs [12]. Giral *et al*. [6] also demonstrated that early APN occurring during the first 3 months following renal transplantation is detrimental to graft outcome. APN may be a consequence of more intensive immunosuppression in patients with acute rejection [12]. Also transplant patients experiencing APN may *a priori* be more susceptible to invading uropathogenic microbes due to several co-morbidities including diabetes mellitus and advanced cardiovascular disease and may be more vulnerable after the additional burden of immunosuppression. On the other hand, acute rejection episodes following APN suggest that APN may trigger an immunostimulatory response [12,24], which was also experimentally demonstrated in mice that were challenged with diverse microbial ligands [25].

In contrast to the general population, no guidelines for the treatment of asymptomatic bacteriuria exist, especially since no effect of asymptomatic bacteriuria on the risk of a potential subsequent UTI nor a detrimental effect on graft or patient survival has been found. However, observational evidence indicates that asymptomatic bacteriuria may be associated with increased creatinine and impaired renal function [26,27]. One explanation for this seemingly contradictory findings are recent findings that *E. coli* strains displaying distinct virulence factors such as fimbriae and O serotype may be more pathogenic and hence dangerous for the transplant [14]. Such bacteria may elicit subclinical inflammatory responses finally resulting in progressive allograft damage. Such a mechanism was also proposed by Ciszek *et al*. who found that interleukin-8, as a critical inflammatory mediator of genitourinary defence, was increased in the urine of renal transplant patients with asymptomatic bacteriuria compared to non-transplant recipients suggesting an inflammatory response potentially affecting the transplant [26]. Conversely, data were recently presented that indicate that in a collective of renal allograft recipients declining graft function was not ameliorated in patients with prophylaxis during asymptomatic bacteriuria indicating that other critical factors are more important for determining the fate of the allograft [27]. Whether diabetic transplant recipients should be treated for asymptomatic bacteriuria in contrast to the general population has to be determined. The emergence of asymptomatic candiduria should evoke aggressive treatment since fungal UTI may cause serious complications including graft loss and patient death [27].

## Risk factors for the development of urinary tract infections

Generally, many risk factors for UTI in renal allograft recipients are similar to those in the general population, especially the

increased risk for developing a UTI in females. Similarly anatomical factors predisposing to UTI like urinary stasis, reflux and stones are more prominent in renal transplant recipients [28]. Potential risk factors involved in the development of UTIs after kidney transplantation include:

- female gender
- advanced age
- pre-transplant UTIs
- prolonged period of haemodialysis before transplantation
- immunosuppression
- acute rejection episodes
- impaired graft function
- bladder catheter postoperatively
- technical complications associated with ureteral anastomosis
- intraperative ureteral stents
- surgical manipulation of the graft (allograft trauma)
- contaminated graft perfusion solution
- diabetes mellitus
- history of vesicoureteral reflux
- history of polycystic kidney disease
- cadaveric donor
- schistosomiasis

Chuang *et al*. [7] identified several patient characteristics such as female gender, advanced age, history of vesicoureteral reflux, azathioprine use and cadaveric donor (as compared to related donor) to be independently associated with an increased risk for the development of post-transplant UTIs. In the study of Dantas *et al*. [19], risk factors for UTIs after kidney transplantation in multivariate analysis included: deceased donor, duration of urinary bladder catheterization, length of hospitalization before infection and changes in the initial immunosuppressive regimen due to acute rejection. While the risk of developing bacteriuria is increased by approximately 5% with each day that a bladder catheter is *in situ* thereby significantly increasing the risk of urinary tract infection in the general population [29], this issue has not been studied in renal transplant patients who routinely have a bladder catheter during transplantation. Nevertheless, the earliest possible removal of the bladder catheter is generally advocated and it has been suggested that early catheter removal may lead to a drop in UTI rates [30].

As mentioned above, female renal transplant patients have a statistically significant higher incidence of UTIs compared to male transplant recipients [7,15,31–33]. For example, in the study of Chuang *et al*. [7], 68% of the female transplant patients but only 30% of male transplant patients had at least one UTI post-transplant. It would have been interesting to know the incidence of UTI before the transplant procedure in each population. On the other hand, Dantas *et al*. [19] did not find any sex-related difference in the frequency of post-transplant UTI during hospitalization in the early post-transplant period.

Advanced age has been recognized as an independent risk factor for the development of post-transplant UTIs. In the study of Chuang *et al*. [7], 55% of the patients who were 65 years of age or older at kidney transplantation developed post-transplant UTIs compared to 30% of patients who were younger than 30 years. Kidney transplantation in elderly patients is often associated with a higher infection rate than that observed in younger patients [34]. Trouillhet *et al*. [35] compared 40 cases (patients older than 65 years) with 40 controls (younger than 65 years) receiving a kidney transplant between January 2000 and August 2002. Infections occurred in 32 cases (80%) but only in 14 controls (32%) during the follow-up. UTIs with *E. coli* (26 vs. 6) or with *Enterococcus faecalis* (9 vs. 3) were more often found in cases than in controls [35]. Impaired cellular immunity and possibly a lower tolerance to immunosuppression along with immunocompromising co-morbidities such as diabetes mellitus may also contribute to the significantly higher percentage of bacterial infections in the elderly patient.

As expected, suppression of the immune system generally increases infection rates including UTIs. Interestingly, it is the treatment with antimetabolites like azathioprine or mycophenolate mofetil-based that has been reported to be associated with a higher incidence of UTI and APN [7,36]. Also therapy with depleting antibodies like antithymocyte globulin for induction therapy increases the UTI risk [13]. Generally, in most studies at least bacteriuria is associated with higher doses of immunosuppressive drugs. Recent insights into the immunobiology of the urinary tract defence demonstrating a highly sophisticated molecular machinery finally evoking an antigen-specific response against urinary microbial antigens make it plausible to propose a direct relationship between infection and immunosuppression.

While the transplant ureter is anastomosed via an extravesical technique and may have a short anti-reflux tunnel, most vesicoureteric anastomoses after transplantation are constantly refluxing. In the study of Chuang *et al*. [7], vesicoureteral reflux disease increased the relative risk for development of a UTI in the renal transplant population. Similarly, Erturk *et al*. [37] also reported a high incidence of UTIs over a mean period of 54 months in renal transplant patients with a history of vesicoureteral reflux. Whether ureteral reimplantation or nephrectomy of the refluxive kidney prevents UTIs in these patients needs to be determined in future studies. Some kidney transplant centres may have a relatively high percentage of urological complications. In those cases, renal transplant patients may benefit from the use of double-J ureteral stents [38–40] by a significant reduction of urinary leakage and ureteral obstruction [40]. However, a significant increase in UTIs was observed when such devices were left more than 30 days after renal transplantation compared to the rate of

 

UTIs in control renal transplant patients suggesting that stent removal within 4 weeks of insertion should be considered [40].

It is unclear whether a history of diabetes mellitus increases [10,40,41] or does not influence [2,7,9] the risk of developing UTIs in renal transplant recipients. Moreover, after renal transplantation a state of post-transplant hyperglycaemia or new-onset diabetes after transplantation develops regularly as a primary consequence of the immunosuppression, thereby complicating the exact potential aetiologic role of abnormal glucose homeostasis. Hence, further studies are warranted to clarify this complex issue. However, diabetes mellitus is strongly associated with a fungally mediated UTI typically by infection with *Candida albicans* [13].

Sadeghi *et al*. [42] found a gender-related urinary cytokine pattern in renal transplant recipients: Anti-inflammatory soluble interleukin-2 receptor antagonist (sIL-2RA) was significantly higher in females than in males, particularly in bacteriuric females while the pro-inflammatory cytokines IL-6 and IL-8 were significantly higher in male renal transplant patients with bacteriuria. In addition, urinary sIL-2RA and sIL-6R excretions were significantly higher in male renal transplant patients with leukocyturia than in those without leukocyturia. Bacteriuria in males was associated with higher doses of immunosuppressive drugs. It was concluded that male renal transplant recipients have a strong inflammatory cytokine response, while female transplant patients display a strong anti-inflammatory response during UTIs [42]. The clinical relevance of the observed gender-related differences between male and female renal transplant patients remains unclear, also the differences observed between patients with bacteriuria and/or leukocyturia compared to those without. Elevated urinary IL-6 excretion has been reported in immunocompetent adults and children with UTIs [43,44]. Bacteriuria is accompanied by elevated urinary IL-6 levels locally produced [45], probably by renal fibroblasts, macrophages and/or renal epithelial cells [46–49]. It was hypothesized that a differential cytokine pattern may also play a role in the protection of healthy versus susceptible individuals, which is also underscored by recent findings demonstrating a particular chemokine receptor polymorphism as the susceptibility gene for urinary tract infection in females [42]. Levels of IL-8 were higher in the urine of patients with asymptomatic bacteriuria who had undergone transplantation than in controls. It was proposed that an increased concentration of IL-8 reflects an inflammatory process that might eventually lead to marked graft damage [26].

## Complications associated with urinary tract infections

Renal transplant recipients with UTIs are more likely to be clinically asymptomatic compared to non-immunocompromised patients, and do not mount the typical inflammatory response to infection primarily as a consequence of immunosuppression. On the other hand, UTI is often associated with APN and rapidly developing bacteraemia potentially progressing to the full-blown picture of urosepsis, particularly during the early post-transplant period. Therefore, careful surveillance is necessary to identify and eliminate these infections. Importantly, UTIs have been shown to be the most common source of bacteraemia in renal transplant recipients [11,24,50]. Patients are at especially high risk for UTI in the first month post-transplant, where the bacteraemia-associated mortality is around 11% in this period. In the study of Chuang *et al*. [7], nine of the 10 patients who died from sepsis had post-transplant UTIs. Infection must be a primary consideration when transplant recipients present to an emergency department with acute illness. In the study of Trzeciak *et al*. [51] infections were the most common indication for emergency department admission (77/217; 35%) and UTI and pneumonia were the most common infections. Nine of 77 patients (11·7%) with infections developed severe sepsis [51]. The most common source of sepsis in renal transplant recipients is the urinary tract [3].

Interestingly, UTIs did not increase the risk for renal graft loss but were significantly associated with increased mortality. In contrast, post-transplant APN was associated with an impairment of long term allograft function but not with the mortality of the patients [12]. In children, the risk for graft loss after early UTI, defined as occurring < 6 months after kidney transplantation, was elevated [adjusted hazard ratio (AHR) 5·47; $P < 0·001$] but not after late UTI, defined as occurring ≥ 6 months after transplantation. Risk for post-transplant death was not increased significantly after either early UTI or late UTI [52].

There are conflicting data on UTIs occurring late after renal transplantation. Late UTIs after renal transplantation have been reported to be rather 'benign' [53,54]. However, other studies suggest that many patients with late UTI's present with advanced infection [55,56]. Retrospective data obtained from the United States Renal Data System (USRDS) from 28 942 patients in the US demonstrate that UTIs occurring late after renal transplantation were independently associated with an increased risk of subsequent recipient death and graft loss [23]. In this regard, it has been shown in both renal transplant recipients and dialysis patients that those patients who develop infections and septicaemia are at an increased risk of dying from causes including cardiovascular disease than are patients who do not develop post-transplantation infections. Muller *et al*. [57] found that patients who experienced chronic rejection displayed a significantly higher rate of UTI more than 2 years after kidney transplantation. In contrast, Giral *et al*. [6] found that early, but not late, recurrent APN was significantly associated with graft loss possibly by inducing or accelerating graft fibrosis. Finally, Munoz reported that UTI manifesting after 6 months of renal transplantation is associated with a serum creatinine > 2 mg $dL^{-1}$, an excess steroid dose (> 20 mg $day^{-1}$), polydrug immunosuppression and chronic viral illness [27].

In the study of Dupont *et al*. [58], 87·5% of the patients with late recurrent UTIs after renal transplantation were female. The first episode of UTI in this study occurred at a medium of 24 months after transplantation. All patients had a minimum of three documented UTIs with a median of six UTIs per patient. Twenty-four patients (75%) were found to have focal renal cortical defects on the 99 mTc 2,3 dimercapto-succinic acid single-photon emission computed tomography (99 mTc-DMSA SPECT) scan, a pattern typical of scarring due to infection [58]. Interestingly, 87% (13/15) of the patients with reflux had focal cortical defects on 99 mTc-DMSA SPECT scan, but also 65% (11/17) of the patients with a history of UTIs but without evidence of vesicoureteric reflux. These data indicate that late recurrent UTIs may indeed be damaging to renal allografts, even in the absence of reflux into the graft. However, levels of proteinuria and serum creatinine at the time of the 99 mTc-DMSA SPECT scan were not different between renal transplant patients with and without scarring. The authors explained the lack of an effect on graft survival by successful intervention with prophylactic antibiotics and surveillance urine cultures in their study [58]. Reflux into the graft due to a loss of innervation is a frequent finding with an incidence of up to 86% [59–62]. It does not necessarily compromise graft function or predispose to recurrent UTIs [59,60,62].

## Prophylaxis and treatment of urinary tract infections in renal transplant recipients

The majority of centres routinely use antimicrobial prophylaxis after renal transplantation within the first 6 months although the individual antibiotic strategies vary. In a randomized controlled trial prophylaxis with high dose (320/1600 mg, daily in two divided doses) TMP-SMZ reduced UTIs during the first months after kidney transplantation to 25% compared to 49·2% in patients on moderate dose (160/800 mg) or low dose (80/400 mg) TMP-SMZ daily [14]. It was not clear, however, why different doses of TMP-SMZ were chosen. This study confirmed earlier results of Fox *et al*. [49] showing that a daily dose of 320/1600 mg TMP/SMZ is an effective prophylactic measure after kidney transplantation. One should, however, consider that a high percentage of uropathogens is TMP-SMZ resistant [14,16,19]. Valera *et al*. [63] evaluated prospectively all UTIs in 161 kidney recipients transplanted between July 2003 and July 2005. All patients received prophylaxis with sulfadoxine-pyrimethamine. Patients with asymptomatic bacteriuria were excluded. In this study, 41 patients (25%) suffered at least one UTI episode. Most common clinical features included uncomplicated acute bacterial cystitis (71 episodes, 77%). Nevertheless, 21 episodes (23%) of APN were observed indicating the low efficacy of such a prophylaxis in renal transplant patients. The causative microorganisms were *E. coli* in 41 cases (71% of UTIs). Long term antibiotic prophylaxis was demonstrated to decrease the incidence of UTI along with hospitalization, suggesting at least cost-effectiveness of this approach. Nevertheless, it remains controversial to support long term antimicrobial therapy in all renal transplant recipients, since the long term efficacy and inherent risk of resistance has not been adequately addressed in these patients. As in the general population, an increasing amount of resistance of *E. coli* isolates is detected against TMP-SMZ (about 60 to 100%) and, e.g. ciprofloxacin (up to 75%) in renal transplant recipients possibly associated with the use of TMP-SMZ as routine prophylaxis against *Pneumocystis jirovecii* and the unselected use of the potent effects of fluorochinolones against many *Enterobacteriae* especially *E. coli*. Furthermore, bacterial strains are emerging that are resistant to multiple antibiotics including broad spectrum antibiotics like cephalosporines and fluorochinolones. Hence it remains doubtful whether long term antibiotic prophylaxis affects graft and patient survival after kidney transplantation [7].

Since UTI in renal transplant patients may not be clinically apparent and rapidly evolve to APN, bacteraemia and even urosepsis post-transplant UTI is managed by the initial administration of empirical antibiotics covering both Gram-negative and Gram-positive bacteria. Specific therapy is thereafter initiated when culture results become available and until the pathogen has been eradicated by assessment of midstream urine samples after a certain follow-up. Lower urinary tract infection without signs of significant patient compromise (malaise, generalized discomfort) or sepsis (fever, hypotension) may be managed on an outpatient basis, but clinical suspicion for APN requires hospital admission and intravenous antibiotics along with adequate fluid management. However, as in the general population it has not been demonstrated that intravenous antibiotic therapy is superior to oral administration with regard to hospital length, cost effectiveness or other end points. While it has been proposed to initiate antibiotic therapy in graft pyelonephritis with at least two antibiotics, at our centre a single broad spectrum is administered that accounts for the typical hierarchic prevalence of local microrganisms dominated by gram-negative bacteria. No general recommendations exist concerning the length of antibiotic therapy in post-transplant UTI. It has been advocated that early UTI should be treated for 10–14 days and if a ureteric stent is present, then the catheter should be removed and examined by culture. It has also been recommended that late post-transplant UTI be treated for 5–7 days. Lower UTI presenting with clinical features of APN, however, should be treated for at least 10–14 days and a patient with urosepsis should at least be treated for 14–21 days.

If post-transplant UTI relapses or recurs immediate investigations have to be performed including imaging studies including CT of the kidneys (stones, complex cysts), CT-PET (cyst infection?) and urological investigations like cystoscopy, urodynamics and micturating cystogram (reflux, bladder

 

dysfunction) before a prolonged course of antibiotic therapy (up to 3 months or even longer) may be initiated. An important topic for the treatment of chronic relapsing post-transplant UTI is the use of cranberry juice, which putatively prevents the adhesion of uropathogenic bacteria to the uroepithelium [64]. Like in the general population no solid data exist on this issue. However, in the generalized population the use of topical oestrogen has successfully resulted in reduction of UTI events in postmenopausal women [65]. A low vaginal pH produced by vaginal lactobacillus colonisation may drop recurrences of UTIs markedly [66]. No data exist about these treatment modalities in renal transplant recipients.

Finally, fungal UTI initially require the removal of indwelling catheters and stents and the administration of potent antifungal agents. In this regard fluconazole is given in a dose of 100 mg per day after an initial loading dose of 200 mg for at least one week; amphotericin is no longer used in many centres especially since newer antifungal compounds like voriconazole, posaconazole and caspofungin have recently become available although systematic data on their safety and efficacy in renal transplant patients are still lacking [28].

## Summary

Infection *per se* is still one of the most important problems in renal transplantation and UTI ranks among the most common infections that the transplanted patient encounters after receiving the allograft. UTI can acutely compromise graft function and left uncontrolled may lead to patient death. Various factors determine the incidence and severity of the post-transplant UTI. Microbial invasion of the host can occur as asymptomatic bacteriuria. However, currently no data exist to support antibiotic therapy in this case with the exception of pregnant renal transplant recipients. If UTI is suspected prompt initial empiric antibiotic therapy is recommended, further thorough investigations are required to identify potential underlying causes of chronically recurring infections. Furthermore, as overimmunosuppression unnecessarily poses the patient to an increased risk of infection in general, individualization of the respective therapy by careful reduction/conversion of one or more of the immunosuppressants may help to allow more effective antimicrobial potency of the host immune system. One of the current and naturally future challenges of the treatment of post-transplant UTI is the careful and selective use of antibiotics since current data indicate a rising incidence of multi-resistant uropathogenic strains. Recent data indicate that transplant pyelonephritis in contrast to mere lower urinary tract UTI may be causally linked to inferior allograft function and possibly patient survival. Thus, the uncertainty about the necessity of long term prophylaxis and the unknown impact of asymptomatic bacteria, in conjunction with improvements in UTI prophylaxis and treatment, make further studies of post-transplant UTI a necessary and fruitful area of future research.

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