

## Bacteriological Urinalysis in Patients after Renal Transplantation

BOŻENA ŁAZIŃSKA<sup>1\*</sup>, MICHAŁ CISZEK<sup>2</sup>, ALICJA ROKOSZ<sup>1</sup>, ANNA SAWICKA-GRZELAK<sup>1</sup>,  
LESZEK PAĆZEK<sup>2</sup> and MIROSLAW ŁUCZAK<sup>1</sup>

<sup>1</sup> Chair and Department of Medical Microbiology, Medical University of Warsaw  
T. Chałubińskiego 5, 02-004 Warsaw, Poland

<sup>2</sup> Transplantation Institute, Medical University of Warsaw, Nowogrodzka 59, 02-006 Warsaw, Poland

Received 23 November 2004, received in revised form 3 October 2005, accepted 10 October 2005

### Abstract

The study consisted of microbiological urinalysis performed in 269 patients after renal transplantation who remained under medical care at the Outpatient Service of the Transplantation Institute in Warsaw. The patients enrolled into the study had undergone renal transplantation 6 to 72 months before urine samples were collected. 304 urinalysis were performed. In the group of 269 patients, 42 individuals had bacteria in their urine what was confirmed in 47 urine cultures. Cases of bacteriuria were divided into 5 groups: 5 cases of symptomatic urinary tract infection (5 individuals – 2% of all studied patients), 27 cases of asymptomatic bacteriuria in 22 individuals (8% of all studied patients), 5 cases of insignificant bacteriuria in 5 patients (2%), 10 cases of involuntary urine contamination in 10 cases (4%). Eventually, urinary tract infection (UTI) was established in 27 patients (5 cases of symptomatic UTI and 22 cases of asymptomatic UTI) what makes out for 10% of all studied patients. In cases where urinalysis showed significant bacteriuria, following pathogens were detected in urine cultures: *Escherichia coli*: 22 strains, *Enterococcus faecalis* – 4 strains, *Enterobacter cloacae* – 2 strains and 1 strains of *Ralstonia pickettii*, *Streptococcus uberis*, *Pseudomonas aeruginosa* and *Proteus mirabilis*. Over 90% of Gram-negative bacteria were susceptible to ceftriaxone and ceftazidime, as well as to amikacin and aztreonam which are the drugs usually administered intravenously in hospitalized patients. The only drug of similar efficacy, which could be administered orally in outpatients was fosfomycin.

**Key words:** patients after renal transplantation, urinary tract infections, identification and drug susceptibility of uropathogens

### Introduction

The urinary tract in healthy individual is well protected against infections by both immunologic and nonimmunologic mechanisms. It has a capacity of self-sterilization due to mechanical washing out of bacteria with the urine stream, urine acidification and secretion of Tamm-Horsfall protein by the tubular cells.

Renal allograft recipients represent a group of patients which is particularly susceptible to UTI. The renal transplantation as a surgical procedure, carries a higher risk of any infection, which is linked with hospitalization, the surgical techniques, anesthesia or specific procedures performed at the Intensive Care Unit. Anastomosis of transplant ureter to urinary bladder is challenging surgical procedure often leading to ureter structure or vesicoureteral reflux. Urinary stasis and the presence of vesicoureteral reflux predispose individuals to the multiplication of bacteria in the urinary tract which may be additionally facilitated by body temperature and chemical components of the urine. The 3-month period after renal transplantation makes transplant recipients even more susceptible to the development of UTI due to intensive immunosuppressive therapy. During that time patients often develop various viral, fungal, parasite as well as bacterial infections. Bacterial infections are most likely to occur in the first month after transplantation. In solid organ transplantation (kidney, liver, pancreas or lungs) infections are mainly detected in the respiratory tract, urinary tract, abdominal cavity and gastrointestinal tract. Thus, solid organ allograft recipients should be closely monitored especially during perioperative period. Microbiological monitoring should include

\* Correspondence to: Bożena Łazińska, Chair and Department of Medical Microbiology, Medical University of Warsaw, T. Chałubińskiego 5, 02-004 Warsaw, Poland, tel./fax + 48 (22)6282739, e-mail: b.lazinska@op.pl

regular control measurements of sterility of drains, catheters (upon removal), analysis of sputum, urine, stool, blood and wound swabs (Dzierżanowska and Jeljaszewicz, 1998).

Urinary tract infection is the most common complication in patients after renal transplantation recipients and also is the most frequent cause of hospitalization in that group (Krieger *et al.*, 1977). Urinary tract infections may have various clinical manifestations ranging from asymptomatic bacteriuria (positive urine culture without typical symptoms such as: fever, urgency, frequency or suprapubic tenderness) to symptomatic infection (urethritis, cystitis, acute and chronic pyelonephritis). Acute UTI could be complicated by sepsis, acute renal insufficiency, hydronephrosis, pyelonephrosis, renal or perirenal abscess. In many cases the primary renal disease may be a triggering factor for UTI like it is observed in reflux nephropathy, chronic pyelonephritis and polycystic kidney disease with episodes of infection localized in the upper urinary tract (Wetzel *et al.*, 1993). Regardless of the primary cause of renal end-stage disease, patient's own cirrhotic kidneys with regressive cysts may be a source of infection that descends down the urinary tract to reach renal allograft. That is why a renal transplant recipient should be closely monitored to establish as quickly as possible the occult infection. The frequency of UTI in early period after renal transplantation (3–6 months after transplantation) is relatively high and according to various reports from the literature exceeds 50% (Renoult *et al.*, 1994; Castelano *et al.*, 1995; Maraha *et al.*, 2001). There are, however, significant differences in assessment of infection risk in later periods after transplantation (Douglas *et al.*, 1974; Kurijama *et al.*, 1991; Goya *et al.*, 1997).

The aim of this study was the assessment of the rate of bacteriuria in patients after renal transplantation, identification of bacterial strains isolated from those patients and bacterial susceptibility to antibiotics.

## Experimental

### Material and Methods

269 patients (108 women, 161 men) after kidney transplantation with stable graft function and serum creatinine concentration below 2.0 mg/dl were part of the study. Patients, who were enrolled into the study, had been transplanted in the Department of General and Transplantation Surgery, Medical University of Warsaw and in the Department of General, Vascular and Transplantation Surgery, Medical University of Warsaw in the years of 1995–2001. The mean age of studied patients was  $43.2 \pm 9.6$  years (median 47,2):  $44.1 \pm 11.1$  in males (median 44,9) and  $42.9 \pm 12.3$  in females (median 43,2). The mean time since the day of transplantation till the day when the urine sample was collected was  $34.1 \pm 18.9$  months (median 32,6) in the whole studied group.

**Urine samples.** Urine specimens for a colony count were obtained from patients on regular check-up visits at the Outpatient Service of the Transplantation Institute. Urine samples were collected 4 hours after previous urination. First morning urine samples could not be collected because most patients lived a long distance away from the Outpatient Service. Patients were provided with information forms where they could find instruction on how a urine specimen should be correctly collected. They were also asked to indicate the time of urine collecting and last micturition. All urine samples were obtained from a midstream into standardized, sterile containers and delivered to the laboratory at the Chair and Department of Medical Microbiology, Medical University of Warsaw within 2 hours after being collected.

**Microbiological examination of urine samples.** In the first stage quantitative urine culture was performed where all urine samples were plated onto blood and MacConkey agar plates. Urine cultures that contained less or equal than 100 000 ( $\leq 10^5$ ) CFU/ml of bacteria or less or equal than 10 000 ( $\leq 10^4$ ) CFU/ml of fungi (one strain of pathogens in each case) were considered insignificant bacteriuria or insignificant funguria. Urine growth with two or more uropathogens was interpreted as contamination and was not further worked up. Patients in such cases were asked to provide another urine sample for correct assessment. In cases where the number of growing colonies of bacteria exceeded  $10^5$  CFU/ml (significant bacteriuria) or the number of growing fungal colonies exceeded  $10^4$  CFU/ml (significant funguria) the samples were further worked up.

**Identification of pathogenic strains.** The biochemical identification of uropathogenic strains was performed in the automatic ATB Expression system (bioMerieux) with the use of specific test cards: ID 32 STAPH, API 20 STREP, ID 32 E, ID 32 GN, and ID 32 C.

**Antibiotic susceptibility testing of urine isolates.** Antibiotic susceptibility of isolated Gram-negative bacilli was evaluated in the ATB expression system with the use of ATB UR test strips. Antibiotic susceptibility of isolated staphylococci, streptococci and enterococci was analyzed with the use of the disk diffusion method as recommended by the National Committee for Clinical Laboratory Standards (NCCLS). Specific disk diffusion tests were used to detect Gram-negative bacilli producing the extended spectrum beta-lactamases (ESBL), methicillin-resistant staphylococci (MRS) and high-level aminoglycoside-resistant enterococci (HLAR). Reference strains such as: *S. aureus* ATCC 25923, *S. aureus* ATCC 29213, *S. aureus* MR3, *E. faecalis* ATCC 29212, *E. coli* ATCC 25922, *K. pneumoniae* ATCC 700603 and *P. aeruginosa* ATCC 27853 were used as controls in verification of antibiotic susceptibility.

## Results

Out of the group of 269 patients the presence of bacteria in urine was detected in 42 individuals (in 47 cultures). Cases of bacteriuria were classified into one of these groups: 5 cases of symptomatic urinary tract infection (UTI) in 5 patients (2% of patients), 27 cases of asymptomatic bacteriuria in 22 patients (8%),

5 cases of non-significant bacteriuria in 5 patients (2%), 10 cases of urine contamination in 10 patients (10%). In total, urinary tract infection was detected in 27 (10%) patients (5 of them had symptomatic infection and 22 – asymptomatic). Gram-negative bacterial strains were isolated in most cases of urinary tract

Table I  
Susceptibility to antibiotics of isolated Gram-positive bacterial strains

Bacterial species	Ampicillin	Amox\Clav	Piperacillin	Nitrofurantoin	Vancocycin	Teicoplanin	Ciprofloxacin	Tetracycline
1. <i>Enterococcus faecalis</i>	S	S	S	S	S	S	I	S
2. <i>Enterococcus faecalis</i>	I	I	I	S	S	S	R	R
3. <i>Enterococcus faecalis</i>	S	S	I	S	S	S	R	I
4. <i>Enterococcus faecalis</i>	S	S	S	S	S	S	I	R
5. <i>Streptococcus uberis</i>	S	S	S	S	S	S	S	S
Total (%)	80	80	60	100	100	100	20	40

Table II  
Susceptibility to antibiotics of isolated Gram-negative bacterial strains

Bacterial species	Amoxycillin	Amox\Clav	Piperacillin	Cephalothin	Ceftriaxone	Ceftazidime	Aztreonam	Tobramycin	Amikacin	Gentamicin	Netilmicin	Nitrofurantoin	Cotrimoxazole	Quinolones 1G	Quinolones 2G	Ciprofloxacin	Fosfomycin
1. <i>E. coli</i>	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S
2. <i>E. coli</i>	R	S	I	S	S	S	S	S	S	S	S	S	S	S	S	S	S
3. <i>E. coli</i>	R	S	I	S	S	S	S	S	S	S	S	S	S	S	S	S	S
4. <i>E. coli</i>	R	R	R	R	S	S	S	S	S	S	S	S	R	R	S	S	S
5. <i>E. coli</i>	R	I	S	R	S	I	S	S	S	S	S	S	R	R	S	S	S
6. <i>E. coli</i>	R	S	R	S	S	S	S	S	S	S	S	S	R	S	S	S	S
7. <i>E. coli</i>	R	R	S	R	S	S	S	S	S	S	S	R	S	R	S	S	R
8. <i>E. coli</i>	R	S	S	R	S	S	S	R	S	R	S	R	S	R	R	R	S
9. <i>E. coli</i>	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S
10. <i>E. coli</i>	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S
11. <i>E. coli</i>	I	S	I	I	S	I	I	I	S	S	S	S	I	I	S	S	S
12. <i>E. coli</i>	R	S	R	S	S	S	S	S	S	S	S	S	S	R	R	R	S
13. <i>E. coli</i>	I	S	I	I	S	I	I	I	S	S	S	S	S	I	I	S	I
14. <i>E. coli</i>	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S
15. <i>E. coli</i>	I	S	I	I	S	I	I	I	I	S	S	S	I	I	I	S	I
16. <i>E. coli</i>	I	I	I	I	I	I	I	I	S	S	S	S	I	I	S	I	I
17. <i>E. coli</i>	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S
18. <i>E. coli</i>	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S
19. <i>E. coli</i>	S	S	S	S	S	S	I	S	S	S	S	S	S	S	S	S	S
20. <i>E. coli</i>	R	I	R	I	S	S	I	S	S	S	S	S	R	R	S	S	S
21. <i>E. coli</i>	R	I	R	R	S	S	I	S	S	S	S	S	S	R	S	S	S
22. <i>E. coli</i>	R	S	I	I	S	S	I	S	S	S	S	S	S	S	S	S	S
23. <i>E. cloacae</i>	R	R	R	R	R	S	R	R	R	R	R	R	R	R	R	R	R
24. <i>E. cloacae</i>	R	R	I	R	I	I	I	I	I	R	R	R	R	I	S	S	I
25. <i>Proteus mirabilis</i>	S	S	S	S	S	S	S	S	S	S	S	R	S	S	S	S	S
26. <i>P. aeruginosa</i>	S	S	I	S	S	S	S	S	S	S	S	S	S	S	S	S	S
27. <i>Ralstonia pickettii</i>	I	R	I	I	I	I	I	I	S	S	S	I	S	I	S	I	II
Total (%)	47	73	59	71	90	94	94	88	95	87	91	77	77	65	88	84	90

Quinolones 1G – nalidixic acid, Quinolones 2G – norfloxacin

infection: *Escherichia coli* in 22 cases, *Enterobacter cloacae* in 2 cases, *Proteus mirabilis*, *Pseudomonas aeruginosa* and *Ralstonia pickettii* each in 1 case. Gram-positive strains were isolated in 5 cases: *Enterococcus faecalis* in 4 and *Streptococcus uberis* in 1 case. Susceptibility of cultured bacterial strains is shown in Tables I and II. There was no HLAR strains detected among enterococci. In the group of Gram-negative bacilli there was no ESBL-positive strains. Multidrug resistant strains of Gram-negative rods can produce  $\beta$ -lactamases of AmpC type. These strains are resistant to all  $\beta$ -lactams, except carbapenems.

In the group of patients with urinary tract infection 5 patients had diabetes mellitus (19% of the group): 2 patients were on insulin therapy and 3 on oral hypoglycemic agents. Diseases of native kidneys leading to end stage renal failure in the group of patients with urinary tract infection were: glomerulonephritis in 8 cases (30% of patients), chronic pyelonephritis in 7 (26%), reflux nephropathy in 4 (15%), diabetic nephropathy in 2 (7%), polycystic kidney disease in 3 (11%) and unknown in 3 (11%) cases.

## Discussion

In the present study UTI was found in 10% of kidney transplant recipients during routine outpatient visits. The data of the frequency of UTI in kidney graft recipients differs among laboratories from 4.2 to 73.7%, depending on group of patients (hospitalized vs. ambulatory), time of observation (early period after transplantation vs. years of observation), chemoprophylaxis used and the definition of UTI (Hamshere *et al.*, 1974; Belitsky *et al.*, 1982; Cuvelier *et al.*, 1985; Maddux *et al.*, 1989). UTI after kidney transplantation is most common during hospitalization period, directly after transplantation procedure. The mean time from the procedure to clinical features of UTI lasts 4–7 days (Midtvedt *et al.*, 1998), the mean period of hospitalization of patients with UTI after transplantation procedure lasts 36 days in comparison to 27 days in patients without UTI (Kentouni-Noly *et al.*, 1994). In the early period, 1–3 months after kidney transplantation, frequency of bacteriuria in patients is high – 39.5 to 73.7% which is confirmed by many laboratories (Renoult *et al.*, 1994; Castelano *et al.*, 1995; Maraha *et al.*, 2001) and it can be even as high as 85% (Mroz *et al.*, 1993). The lowest percentage of UTI was described in one of Japanese transplant center: 10% in perioperative period and 4.2% in ambulatory follow up (Goya *et al.*, 1997). In this study the 5-day perioperative therapy with III generation cephalosporin intravenously and 4-month prophylaxis with trimethoprim-sulphamethoxazole in relatively high dose ( $3 \times 480$  mg every second day) were administered to all patients.

During later period after transplantation the frequency of UTI decreases but identification and management of this complication is a challenge in ambulatory medical care. Almost 80% of UTI cases in this group of patients are lower urinary tract infections, usually asymptomatic (Schmaldienst *et al.*, 2002). In our study asymptomatic bacteriuria were diagnosed in relatively high rate (8% of study population) and symptomatic UTI in only 2% of patients. There is no evidence about the influence of asymptomatic bacteriuria on the function of transplanted kidney. In some reports, in which the aim was the assessment of the influence of symptomatic UTI on the function of transplanted kidney, it was showed that this influence exists, but it can be observed after long – over 3-year observation (Muller *et al.*, 1998). Opinions about treatment of asymptomatic bacteriuria in patients after transplantation differ among authors. Some of them believe that this treatment is always necessary (Cormio *et al.*, 2002; Raz, 2001), some – that the therapy is necessary only in early period after the procedure (Duława *et al.*, 2001; Korzeniowski, 1991). The third group of authors suggests that the treatment is not required (Nicolle, 2000; Goya *et al.*, 1991).

In our study the incidence of diabetes mellitus in UTI group was 20% which is comparable to rate observed in whole population of kidney transplant patients. Chronic pyelonephritis and reflux nephropathy account for 30% diseases led to end stage renal failure in the group of patients with UTI. It is well known that patient's own cirrhotic kidneys with regressive cysts may be a source of infection descending the urinary tract to the renal allograft. Our observation could support recommendation for more thorough monitoring for UTI in this groups of patients.

The dominating bacteria in the cultures from tested urine specimens were Gram-negative microorganisms. Over 90% of isolated Gram-negative strains were sensitive to ceftriaxone, ceftazidime, amikacin and aztreonam – the drugs that can be administered intravenously in the hospitals. The only drug with similar effectiveness which can be administered orally in ambulatory medical care was fosfomycin. Ciprofloxacin and norfloxacin showed relatively high effectiveness (84% and 88% susceptible Gram-negative bacterial strains). These agents could be used in empiric treatment of UTI in patients after kidney transplantation. Although it was a small number of isolated strains, analysis of the data of susceptibility to antibiotics of Gram-positive bacteria showed that all of cultured enterococci were susceptible to nitrofurantoin. Urinary tract infection caused by enterococci is especially dangerous because it could lead to urosepsis, especially in patients with immunosuppres-

sion-associated leukopenia (Caballero-Granado *et al.*, 2001). Unfortunately, poor tissue penetration of nitrofurantoin and low urine concentration in patients with decreased glomerular filtration rate make this drug ineffective in most of kidney transplant recipients. In conclusion urine culture should be performed before starting UTI treatment in renal transplant recipients, especially caused by Gram-positive bacterial strains.

The main aim of postoperative prophylaxis in graft recipients is eradication of Gram-negative bacilli of the family *Enterobacteriaceae*. Antibiotics that can be used for this purpose are orally administered fluoroquinolones and co-trimoxazole. In our study Gram-negative bacterial strains susceptibility to these agents was relatively high (88% and 77%, respectively) what makes them reasonable choice for postoperative prophylaxis.

Overall, the antimicrobial therapy should be considered individually, the choice of the drug should be based on pharmacokinetics of the drug, specificity of the disease and interactions with immunosuppressive drugs (Dzierżanowska and Jeljaszewicz, 1998).

### Literature

- Belitsky P., S.G. Lannon, A.S. MacDonald, A.D. Cohen, T.J. Marrie, P. Houlihan and A. Whalen. 1982. Urinary tract infections (UTI) after kidney transplantation. *Transplant Proc.* **14**: 696–699.
- Caballero-Granado F.J., B. Becerril, J.M. Cisneros, L. Cuberos, I. Moreno and J. Pachon. 2001. Case-control study of risk factors for the development of enterococcal bacteremia. *Eur. J. Clin. Microbiol. Infect. Dis.* **20**: 83–90.
- Castelano A.M., K. Soto, J.M. Grinyo, S. Gilvernet, D. Seron, J. Torras, L. Riera, J.M. Cruzado and J. Alsina. 1995. Prophylaxis of urinary tract infection in renal transplantation: comparison of three different protocols using ceftriaxone-cloxacillin, aztreonam-cloxacillin, or aztreonam-amoxicillin-clavulanic acid. *Transplant Proc.* **27**: 2277–2279.
- Cormio L., B. Berardi, A. Callea, N. Fiorentino, D. Sblendorio, V. Zizzi and A. Traficante. 2002. Antimicrobial prophylaxis for transrectal prostatic biopsy: a prospective study of ciprofloxacin vs piperacillin/tazobactam. *BJU Int.* **90**: 700–702.
- Cuvelier R., Y. Pirson, G.P. Alexandre and C. van Ypersele de Strihou. 1985. Late urinary tract infection after transplantation: prevalence, predisposition and morbidity. *Nephron* **40**: 76–78.
- Douglas J.F., S. Clarke, J. Kennedy, J. McEvoy and M.G. McGeown. 1974. Urinary tract infection after renal transplantation. *Lancet* **2**: 1015.
- Duława J., M. Myśliwiec and A. Wielgosz. 2001. Identification and treatment of urinary tract infections (in Polish). In: B. Rutkowski and S. Czekalski (eds), Standards in management and treatment of kidney diseases. MAKmed.
- Dzierżanowska D. and J. Jeljaszewicz. 1998. Hospital infections (in Polish).  $\alpha$ -medica press, Bielsko-Biała.
- Goya N., K. Tanabe, Y. Iguchi, T. Oshima, T. Yagisawa, H. Toma, T. Agishi, K. Ota and K. Takahashi. 1997. Prevalence of urinary tract infection during outpatient follow-up after renal transplantation. *Infection* **25**: 101–105.
- Hamshere R.J., G.D. Chisholm and R. Shackman. 1974. Late urinary-tract infection after renal transplantation. *Lancet* **2**: 793–794.
- Kentouni-Noly J.C., P. Cloix, X. Martin, M. Rabodonirina, N. Lefrancois, M. Sepetjan. 1994. Analysis of nosocomial infections in renal transplant and pancreas transplant recipients. *Transplant Proc.* **26**: 284.
- Korzeniowski O.M. 1991. Urinary tract infection in impaired host. *Med. Clin. North Am.* **75**: 391–401.
- Krieger J.N., L. Tapia, W.T. Stubenbord, K.H. Stenzel and A.L. Rubin. 1977. Urinary infection in kidney transplantation. *Urology* **9**: 130–136.
- Kurijama M., T. Nagai, H. Uno, Y. Nishida, T. Ishihara, K. Kobayashi, Y. Takahashi, A. Saito and K. Kawada. 1991. Urinary tract infections after kidney transplantation. *Acta Urol. Jpn.* **37**: 1173–1179.
- Maddux M.S., S.A. Veremis, W.D. Bauma, R. Pollak and M.F. Mozes. 1989. Effective prophylaxis of early post-transplant urinary tract infections (UTI) in the cyclosporine (CSA) era. *Transplant Proc.* **21**: 2108–2109.
- Maraha B., H. Bonten, H. van Hooff, H. Fiolet, A.G. Buiting and E.E. Stobberingh. 2001. Infectious complications and antibiotic use in renal transplant recipients during a 1-year follow-up. *Clin. Microbiol. Infect.* **7**: 619–625.
- Midtvedt K., A. Hartmann, T. Midtvedt and I.B. Brekke. 1998. Routine perioperative antibiotic prophylactic in renal transplantation. *Nephrol. Dial. Transplant.* **13**: 1637–1641.
- Muller V., G. Becker, M. Delfs, K.H. Albrecht, T. Philipp and U. Heemann. 1998. Do urinary tract infections trigger chronic kidney transplant rejection in man? *J. Urol.* **159**: 1826–1829.
- Mroz E., J. Uzar, T. Szepietowski, J. Starek and J. Bartelmus. 1993. [Urinary tract infections in the first three months following kidney transplantation]. *Pol. Tyg. Lek.* **48**: 448–451.
- Nicolle L.E. 2000. Asymptomatic bacteriuria-important or not? *N. Engl. J. Med.* **304**: 1037–1039.
- Raz R. 2001. Asymptomatic bacteria-clinical significance and management. *Nephrol. Dial. Transplant.* **16**: 135–136.
- Renoult E., F. Aouragh, D. Mayeux, D. Hestin, A. Lataste, J. Hubert, J. L'Hermite, M. Weber and M. Kessler. 1994. Factors influencing early urinary tract infections in kidney transplant recipients. *Transplant. Proc.* **26**: 2056–2058.
- Schmaldienst S., E. Dittrich and W.H. Hörl. 2002. Urinary tract infections after renal transplantation. *Curr. Opin. Urol.* **12**: 125–130.
- Stein G. and R. Funfstuck. 1999. Asymptomatic bacteriuria-what to do. *Nephrol. Dial. Transplant.* **14**: 1618–1621.
- Wetzel O., M. Hormi, L. Le Normand, G. Karam, J. Guenel, J. Auvinne and J.M. Buzelin. 1993. Autosomal dominant polycystic kidney disease: urologic complications and results of kidney transplantation: 217 patients. *Prog. Urol.* **3**: 252–262.