

itive strains. Gram-positive bacteria were cultured 34% (n=25) and fungal strains 3% (n=2).

Conclusions: The predominance of Gram-negative rods was caused by ESBL positive and use of broad spectrum antimicrobial prophylaxis. The increased proportion of isolation Multi-Drug-Resistant (MDR) bacteria to antimicrobial agents may be due to the frequent use of these agents for prophylaxis of bacterial infections in liver transplant patients. These (MDR) bacterial strains caused severe UTI's in patients after LT.

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Fungal Infections in Patients after Solid Organ Transplants

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Introduction: Fungal infection is a severe complication in patients undergoing solid organ transplantations.

Objective: The objective of the study was to evaluate species distribution and antifungal susceptibilities of fungal isolates taken from patients after solid organ transplantation (SOT).

Material and Methods: The study included samples of urine, blood, systemic fluids and the swabs of the post-operative wounds, haematomas, drains taken from patients after kidney, liver or simultaneous pancreas-kidney transplantation hospitalized in Institute of Transplantation Medicine, Medical University of Warsaw and Department of General and Transplantation Surgery, Medical University of Warsaw in 2005- 2007. All cultured specimens were isolated by using Sabouraud medium with antibacterial protection using chloramphenicol and gentamicin (bioMerieux, France or Oxoid, England). Yeast-like fungi was incubated on CHROMagar Candida Medium (Becton Dickinson R) and identified by using biochemical, automatic test ID32C (bioMerieux). Susceptibility of the strains to the antifungal agents: amfotericin B, itraconazol, fluconazol, voriconazol was tested using E-test (AB Biodisk) on RPMI agar plates (BiolifeR).

Results: The positive cultures were obtained in 662 samples from 402 patients receiving immunosuppressive therapy. There were cultured 267 isolates of *Candida albicans*, 223 *Candida glabrata*, 39 strains of *C. parapsilosis*, 37 *C. krusei* and 35 of *Trichosporon asahii*, 20 *Saccharomyces cerevisiae*, 9 strains of *Cryptococcus neoformans* 5 *C. lipolytica*, 6 strains of *C. lusitaniae* and 4 of *C. sake*, 5 strain of *C. quilliermondii*, 3 *C. rugosa* and 9 moulds from *Aspergillus fumigatus* species. From cultured strains 37.3% was naturally fluconazole resistant and 0.5% amphotericin B resistant. The acquired resistant to fluconazole was presented by 10 isolates, to itraconazole 26, to voriconazole 2 and to amphotericin B by 3 isolates.

Conclusions: The most numerable groups of isolated fungal species in patients after SOT were *C. albicans* 40% of all isolates and *C. glabrata* 33.7%. Moulds were cultured less often and they were found only in 1.4% of positive cultures. More than one third of isolated fungi were resistant to fluconazole.

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Selection of Resistant Fungi in Liver Transplant Recipients During use of New Anti-fungal Agents

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Background: The echinocandins and the new-generation azoles have been a boon to physicians managing fungal infections in transplant recipients. But Nature abhors a vacuum. In this report, we describe 2 liver transplant recipients on the new broad-spectrum anti-fungals who became colonized and infected with fungi resistant to these new agents.

Methods: Retrospective chart review. MIC of *Candida spp.* to azole anti-fungals measured by E-test method.

Results: Case 1, a 36-year-old man, received a right-lobe graft for fulminant hepatic failure, likely drug-induced. The transplant surgery was complicated by the need for a colostomy. While under standard immunosuppression, he was given caspofungin (CAS) for *Candida lusitaniae* in the bloodstream. Three weeks later blood cultures grew *Trichosporon asahii*, which responded to voriconazole (VCZ). Case 2 was a 56-year-old man with chronic Hepatitis B infection and hepatocellular carcinoma, who underwent living donor liver transplant with a left-lobe graft. He received standard immunosuppression. In the early post-transplant period, hypodensities developed segment 3, suggestive of infarction. In the 3rd post-transplant month, the fully-evolved infarcted areas were documented to be in communication with the biliary tree.

Repeated fevers led to the placement of a biliary drain. Bile aspirated on the 79th post-transplant day (PTD) grew *Candida albicans*, and the patient was given fluconazole (FCZ). A cholangiogram on the 139th PTD led to bile aspiration that cultured out *C. albicans* (FCZ MIC 1.5ug/ml) and *C. krusei*. A septic episode on the 150th PTD led to the use of VCZ and broad-spectrum antibiotics. Bile aspirated on the 160th PTD grew *C. glabrata* that was resistant to FCZ, VCZ, itraconazole and posaconazole.

Conclusions: Although CAS and VCZ have been a boon to physicians managing fungal infections, their use can be associated with the selection of fungi resistant to them. Care with the use of antibiotics should extend to the anti-fungals as well.

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Urinary Tract Infections in Renal Transplant Recipients

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Objectives: To determine the causative agents of urinary tract infections (UTIs) among renal transplant recipients and to compare the antibiotic susceptibilities of *Escherichia coli* strains isolated from complicated community-acquired UTIs and renal transplant recipients.

Methods: We evaluated 108 episodes of 82 recipients (46 women) with confirmed UTI who were transplanted during the period 1981 to December 2007 at our center. Medical records of the patients were reviewed retrospectively. To compare the susceptibility rates of *E. coli*, 226 isolates from non-transplant patients with complicated community-acquired UTIs were also evaluated.

Results: The mean age of the patients was 35.9 years in the range from 16 to 58 years. Sixty-three patients had single episodes. Seventeen episodes (15.7%) occurred in the first month following the transplantation, 32 (29.6%) in the period of the second month to sixth month and 59 (54.6%) occurred six months after transplantation. Sixty-six (61.1%) of the 108 isolates were *Escherichia coli*, 24 (22.2%) *Klebsiella spp.*, 5 (4.6%) *Enterococcus spp.*, 5 (4.6%) *Staphylococcus aureus*, 3 (2.8%) *Acinetobacter baumannii*, 1 (0.9%)

coagulase-negative *staphylococcus*, 1 (0.9%) *Proteus mirabilis*, 1 (0.9%) *Pseudomonas aeruginosa*, 1 (0.9%) *Salmonella* spp, 1 (0.9%) *Enterobacter aerogenes*. The resistance rates of *E. coli* strains are given in the table. Among the *E. coli* strains isolated transplant recipients, ciprofloxacin resistance rates were 37.5% (3/8) in the first month after transplantation, 71.4% (15/21) in the period of the second month to sixth month, and 35.1% (13/37) 6 months after transplantation. The resistance rates of TMP-SMX in the same time periods were 100% (8/8), 90.5% (19/21), and 73% (27/37), respectively. See Table 1.

Table 1. The resistance rates of *E. coli* strains among renal recipients and the complicated community-acquired UTIs isolates from non-transplant patients

Antimicrobial agent	Renal recipients, n=66 (%)	Community-acquired, n=226 (%)	P value
Ampicillin	61 (92.4)	136 (60.2)	<0.05
Amoxicillin/clavulanic acid	29 (43.9)	88 (38.9)	0.47
Cefazolin	28 (42.4)	74 (32.7)	0.15
Cefuroxime	21 (31.8)	51 (22.6)	0.12
Ceftriaxone	13 (19.7)	38 (16.8)	0.59
Gentamicin	24 (36.4)	33 (14.6)	<0.05
Ciprofloxacin	31 (47.0)	85 (37.6)	0.17
TMP-SMX	54 (81.8)	95 (42.0)	<0.05

Conclusion: *E. coli* was the most frequently isolated organism from UTIs in renal transplant recipients. The rates of resistance to TMP-SMX, ampicillin, gentamicin among *E. coli* isolated from renal recipients were significantly higher than those in community-acquired complicated UTIs. We have observed that the majority of the UTIs occurred beyond the sixth month after transplantation, probably due to the fact that prophylactic TMP-SMX treatment in renal transplant recipients provides some protection against UTIs.

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Chlamydia pneumoniae Infection and Spiramycin Therapy in Kidney Transplant Recipients

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Background: The importance role of *C. pneumoniae* latent infection in pathogenesis of atherosclerosis is been well known and clinical efficacy of antibiotic therapy has been described. One of the most important problems in the transplantation field is slow deterioration of transplanted kidney function. Previous research by our centre pointed to a significant role of *C. pneumoniae* infection in the development of chronic renal allograft dysfunction. There are trials that confirmed *C. pneumoniae* infection role in liver chronic rejection and vasculopathy of the transplanted heart. Pathogenesis of these changes were similar with atherosclerosis.

Objectives: The aim of the study was to evaluate the *C. pneumoniae* presence prior to and after kidney transplantation and to determine the role of spiramycin therapy in patients after kidney transplantation.

Material and Methods: The study group consisted of 50 patients (25 pairs) who received kidney transplants from cadaveric donors. One of the two kidneys from one donor was transplanted to a patient randomised to spiramycin (dose of 2x3million U/day orally for 3 months) (group S) and the other was transplanted to a patient as-

signed as control (group C). Markers of infection were assessed on day 1 post transplantation and 3 months later (average=94 days). All (n=50) patients were examined for bacterial DNA presence in peripheral blood leukocytes using real-time PCR and titers of serum anti *C. pneumoniae* IgG and IgA antibodies using microimmunofluorescence (MIF). *C. pneumoniae* infection was diagnosed as *C. pneumoniae* DNA presence in peripheral blood leukocytes or positive antibodies titres in both classes IgA (titer>1:16) and IgG (titer >1:64).

Results: *C. pneumoniae* infection was initially diagnosed in 14 patients from group S and in 8 patients from group C (p=ns) after 3 months in 12 and 9 patients respectively (p=ns). Conversion from positive to negative *C. pneumoniae* status occurred in 7 patients from group S and one patient from group C (p=0.04). Conversion from negative to positive *C. pneumoniae* status occurred in 5 patients from group S and 2 patients from group C (p=ns). Negative *C. pneumoniae* status during research occurred in 6 patients from group S and in 15 patients from group C (p= 0.02).

Conclusions: The results suggest a possible role for spiramycin treatment of in *C. pneumoniae* infection in kidney allograft recipients. Spiramycin is safe and has no significant interactions with immunosuppression treatment in kidney allograft recipients.

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Molecular Diagnosis, Follow-up and Identification of Natural *Trypanosoma cruzi* Populations in Chagas Heart Disease Patients Undergoing Clinical Reactivation after Heart Transplantation

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Background: End-stage Chronic Chagas heart disease (ChD) caused by *T. cruzi* involves dilated cardiomyopathy, complex arrhythmias, heart failure and sudden death. Heart transplantation (HT) is a valid treatment for ChD but reactivation of *T. cruzi* (RA) is the main complication of concomitant immunosuppression. Natural *T. cruzi* populations display different tissue tropisms, growth rates and drug susceptibility. Their genetic polymorphism supports distinction of two major lineages: *T. cruzi* I and II. They have been identified from cultured stocks, underestimating diversity and precluding direct association studies of the parasite role with pathogenesis.

Objectives: To follow-up *T. cruzi* infection and genotype populations associated to heart tropism and RA directly in clinical samples from ChD undergoing HT, using molecular methods.

Methods: 9 ChD patients with a mean follow-up of 907 days (37–2279 d); routine parasitologic tests and PCR of minicircle DNA (kDNA) in blood, cardiac explants, endomyocardial biopsies of the allograft heart and skin lesions. Lineage identification was done by genomic PCR (Lg-PCR) and intra-lineage typing by RFLP-PCR of kDNA.

Results: Sections from 7/8 heart explants of ChD were kDNA-PCR+ and 6/7 Lg-PCR+ (2 Tc I, 3 Tc II and 1 both). RFLP-PCR profiles were polymorphic in different patient's hearts and also among sections of a same explant. Between 1-3 and 1-6 weeks after HT, kDNA-PCR and Lg-PCR turned into positive in the 9 patients, showing recrudescence of infection leading in 6 to patent parasitemia concomitant with RA within a mean period of 71.6 days. 4/9 ChD had Tc I and 5/9 Tc II in blood. Lg-PCR was positive earlier in Tc II than in Tc I infections. 5/6 RA patients developed lower limbs skin lesions and 1 chagasic myocarditis; in 5 patients identical lineages and RFLP-