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Research Paper

Infection in Kidney Transplantation: Mind the Gap

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ABSTRACT
Kidney transplant is the best renal replacement option and patients with End Stage Kidney Disease(ESKD) can assess this treatment not only in developed but also in developing countries like Nigeria. However, there is a need to appraise one of the commonest medical complications of thistreatment option, infection.
This review focuses on the common infections that are found in kidney transplant recipients.

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I. INTRODUCTION

Infections are not uncommon following Kidney transplantation (KT) and they are a major cause of morbidity and mortality(1). In Nigeria, infection related mortality was reported to be as high as 30.3% in kidney transplant recipients(KTRs) and the median duration of post KT infection was 270 days (range 2-2190days). (2).The major gaps to consider is that KTRs seldom have typical symptom and signs of the infection and the possibilities of drug-drug interactions while treating these infections.

These infections are preventable to some extent or their impact may be reduced through careful pretransplant screening of donors and recipients.Vaccination and diligent post KT disease surveillance as well as use of chemoprophylaxis must be ensured..

Dynamic assessments of risk of infection.

Infection in kidney transplantation can be generally divided into Donor Derived Infections and Recipient Derived Infections. Also, just like in any solid organ, it is convenient to classify this infection into early, intermediate, and late when they occur within the first month, first to six months and after six months post transplantation, respectively.

Early infections (<1 Month)

These aremostly nosocomial in origin but may also arise from technical problems with donor or recipients.Although, in a recent report by Korean Organ Transplantation Registry Study Group Kim et al reported that these post-transplant infections have adverse effect on graft and patient outcomes and that these outcomes are worse in older KTRs(3), our group had earlier reported the significance of donor-recipient age difference on total duration of graft survival(4) suggesting that early post transplantation infection should not be considered in isolation.

Antimicrobial resistant species like Methicillin-resistant Staphylococcus aureus (MRSA), Vancomycin-resistant enterococcus (VRE) as well as non-albicans candida species have been reported in KTRs within the first month post transplantation(5-7). However, Adeyemi, Qi, Zembower and colleagues reported cases of MRSA presenting in KTRs after 20 months post transplants(8)

Other infections in this category are those resulting from aspiration, catheterization, wound sites leak, ischemia of points of anastomosis as well as *clostridium*defficile colitis.Uncommonly, some of the infections that will occur early in KTRs are donor derived(9-11). These donor derived infections include those resulting from Herpes Simples Virus, Trypanosoma cruzi, West Nile virus, rhabdovirus (rabies) and lymphocytic choriomeningitis viral infection.Also, recipient- derived infection occurring this early in KTRs may be because of colonization with aspergillus and pseudomonas.

Intermediate infection (1-6 months)

There are reports of infections inKTRs which commonly occur at about one to six months following KT. These include Pneumocystis jirovecii(12), Hepatitis B virus (HBV)(13, 14) and Cytomegalovirus (CMV)

infections(15, 16). Others are infections from hepatitis C,Adenovirus, and influenza viral infections(16). Furthermore, cryptococcal neoformans and mycobacterium tuberculosis infections are in this category(17, 18).Some of these are preventable with the use ofchemoprophylaxis(19-21).

Late infection (>6 months)

Most infections in this category are community-acquired and these include community acquired pneumonia(22) and urinary tract infection(23). Others are infection with aspergillus, atypical molds and Mucor species (24, 25). In one of the studies from Nigeria, UTI was reportedas the most common infection in KTRs(2).

There are also late onset viral infections from CMV (colitis and retinitis), HBV and HCV. Skin cancer and post-transplant lymphomatous disease (PTLD) will most often occur in this late stage as well(26).

Pretransplant Screening

Guidelines are available for pretransplant screening such as Kidney Disease Improving Global Outcome (KDIGO)guideline (27). The screenings are either classified as recommended or augmented. The recommended ones are those as documented by societies while the augmented screening is according to regional endemic or epidemic infections.

Vaccination

Assessment of vaccine status of an intending recipient is a compulsory. This allows for administration of required important vaccines. Intending KTRs should be screened and vaccinated at least four weeks before allograft transplantation (28). These vaccines are pneumococcal, HBV and influenza vaccines.
Vaccines can either be inactivated or live. The rule of the thumb is avoidance of live vaccines in KTRs. Table 1 shows types of infection, the appropriate immunization types, and the recommended period for Re-immunization.

Table 1. Immunizations in Kidney Transplantation Recipients

Name	Immunization type	Reimmunization	Contraindication
Varicella	Live attenuated	Give 2nd dose after 1-2 months	Immunosuppressed. Give only prior to transplant. Don't give to those who have received blood products in the last 6 month
Influenza	Inactivated	Yearly	Intranasal formulation because it is live attenuated
Pneumococcal	Component	2nd dose 5 years after	-
Tetanus-diphtheria	Toxoid	Every 10 years. Give booster at 5 years if deep puncture wound	History of neurological reaction or anaphylaxis from pervious dose
Meningococcal	Component	3-5 years in antibody titre declines	-
Haemophilus Influenza type B	Conjugated	Give 2nd dose after 2 months	-
Hepatitis A	Formalin-inactivated	Give booster at 6-12 month	-
Hepatitis B	Recombinant	Repeat 1 and 2 months. Check titre after 1 moth after last shot and if <10, give booster	-
Measles, Mumps, Rubella (MMR)	Live attenuated	Give 2nd dose 1-6 years after	Pregnancy, immunosuppression and anaphylaxis to first dose

Cytomegalovirus (CMV) infection

The incidence of cytomegalovirus infection in KTRs ranges from 16-67%, without prevention(29, 30). In the largest Brazilian cohort of patients so far, Felipe CR et al reported no death from CMV infection but concluded that it was associated with high incidence of acute rejection(AR) and changes in immunosuppression(31). Incidence of CMV disease in KTRs in donor CMV-seronegative/recipient CMV seronegative (D-/R-) is less than 5%. When it is donor derived, it can occur as primary infection or reinfection and these are the commonest type of CMV infection in KTRs(31). It can also occur as a reactivation of latent recipient infection. It is important to differentiate between CMV infection and CMV disease as they are not synonymous. Also, not all individuals with CMV infection will eventually have CMV disease. CMV infection

refers to isolation or detection of viral protein (antigen) or nucleic acid in any body fluid or tissue specimen regardless of symptoms or signs.CMV disease consists of "end-organ disease" and CMV syndrome. However, the definition of "proven CMV end-organ disease," requires the presence of appropriate clinical symptoms and/or signs together with documentation of CMV in tissue from the relevant organ by histopathology, virusisolation, rapid culture, immunohistochemistry, or DNA hybridization(32). Elevated viral DNA levels detected with quantitative Nucleic Acid Test (NAT), such as polymerase chain reaction (PCR), in tissue from the relevant organ likely represent CMV disease and could therefore be accepted as "possible CMV end-organ disease," especially when blood sampled at the same time does not contain CMV DNA. CMV Infection
In defining CMV infection, it is recommended that both the source of the specimens tested (e.g., plasma, serum, whole blood, peripheral blood leukocytes [PBLs], cerebrospinal fluid [CSF], bronchoalveolar lavage [BAL] fluid, urine, or tissue) and the diagnostic method used be described clearly. Antibody testing and culture are less sensitive(32)

More importantly, CMV infection can decrease patient and graft survival and if the infection occurs within the first 100 days post-transplant, there is a strong association with increase patient mortality(31).It is also an independent risk factor for acute rejection, PTLD, posttransplant DM and recurrent TMA post KT(33). It is also linked with Post KT glomerulopathy(34) .Treatment of established CMV disease is different from that of CMV infection.(34-38).Administration of chemoprophylaxis in CMV infection reduces disease incidence by 60% and it is recommended for high risk patients. This is iindicated where donor is positive but recipient is negative (D+/R-), both donor and recipients are positive (D+/R+), donor is negative but recipient is positive (D-/R+). The recommended antiviral medications are Valganciclovir or oral ganciclovir and the recommended duration of treatment is for at least 3 months post KT. However, longer duration of treatment is recommended when donor is positive and recipient is negative (6month in D+/R). Established CMV disease requires reduction of immunosuppression, commencement of antiviral medication with or without adjunct therapy.The preferred choice of medication in life threatening CMV disease is intravenous (IV) ganciclovir at a dose 5mg/kg twice daily. Valcyte in CMV disease Treatment of Solid organ Recipient (VICTOR) trial recommends the use of valganciclovir at a dose of 900mg twice daily with or without adjuvant. CMV resistance is considered if there is no response after 2 weeks of treatment and this can be identified by genotype testing to identify (39, 40).

Epstein Barr Viral infection

This is not as common as CMV infection but it is important because of its association with PTLD(41). This usually occurs within the first-year post KT and 62%-79% of cases have been associated with EBV. The risk for early PTLD include not just EBV infection but also young age recipient, CMV infection,treatment with OKT3 or polyclonal antilymphocyte antibody and the type of organ transplantation(16, 41-43).

EBV infection normally presents as non-febrile syndrome, lymphadenopathy, enlarged liver and spleen, atypical lymphocytosis and organ specific symptoms which include pneumonia, hepatitis, and gastroenteritis. It can also present as cytopenia(42). Mortality following EBV-associated PTLD is about 50% and the diagnosis requires histologic confirmation with immunologic cell- typing There is no consensus regarding the treatment of EBV(44).However,Schachtner and Reinke reported a possible efficacy of rituximab in the treatment of EBV infection(45)

BK Polyoma Virus (BKV) infection

This is associated with polyomavirus associated nephropathy (PyVAN) and polyomavirus associated hemorrhagic cystitis. It is not uncommon as it affects up to 10% of KTR with attendant 10%-80% graft loss(46). BKV viremia usually develops within the first 3 monthsfollowing KT and nephropathy usually occur within the first 2 years posttransplant. The risk factor for PyVAN include high level of immunosuppressants and recipient characteristic (old age, male sex, decrease BK virus- specific T cell activation).(47) The risk factor could also be donor characteristics (female, deceased donation, increase cold ischemia time, HLA mismatch and African-American ethnicity.

The diagnosis of BKV infection is made through the identification of viremia, NAT. Pathology and detection of decoy cells in the kidney(48)

BKV can present as non-specific febrile syndrome, lymphadenopathy, enlarged liver and spleen, atypical lymphocytosis and organ specific symptoms (pneumonia, hepatitis and gastroenteritis(46).

Hepatitis B Viral (HBV) infection

This is Increasingly being recognized in ESRD patients. (49-51), Donor derived HBV infection is rare in KTRs as they are readily diagnosed during pre-transplantation screening. Patients with chronic HBV and clear viremia are now considered suitable for KT. However, serial monitoring of HBV DNA every 3-6 month is required in such patients. Also, there is a need to screen for Hepatocellular cancer (HCC) with ∝-fetoprotein and

abdominal ultrasound every 12 months in KTRs with HBV infection. Reported treatment options include interferon alpha(IFN∝), pegylated IFN, lamivudine, entecavir, telbivudine,tenofovir and adefovir(52).

Hepatitis C Infection(HCV)

Recognition of HCV infection pre-transplantation is important. NAT is required as antibody formation is impaired in ESRD patients.KTR infected with HCV have decrease survival. Therefore, it is important to treat before transplantation(53). Reported post KT complications of HCV include glomerulopathy, diabetes mellitus,cirrhosis as well as cholestatic hepatitis .Treatment is with interferon (IFN) with a reported response rate of 20%-90%. Also there is there is a strong association between HCV infection increase risk of allograft dysfunction. Ribavirin is generally contraindicated in Kidney failure(54-56)

Human Immunodeficiency Virus (HIV) Infection.

Selected HIV infected patients can now undergo KT with graft and patient survival rates comparable with non-HIV infected KTR older than 65yr. However, there are criteria for KT in this group of patients(57). This includeundetectable viral load, CD4 count of 200 cells/ µl, absence of untreatable infection and absence of malignancy(57, 58). It is important to coordinate the use of highly active antiretroviral therapy (HAART) with the patient's own HIV caregiver but protease inhibitor (PI) should be avoided where possible as it interacts with immunosuppressants(59)

Antithymocyte globulin (ATG) is not an ideal induction agent in this population of KTR as it decreases CD4 count. Instead, use of monoclonal anti IL2 receptor antibodies such as basiliximab/daclizumab is preferred for induction. Lifelong treatment with trimethoprim-Sulfamethoxazole (TMP-SMX) is recommended for KTRs with HIV infection(59).

Urinary Tract Infection (UTI)

The commonest bacterial infection in KTR. Incidence is about 17% in the 1st 6mo post KT (USRDS). In Nigeria the prevalence of UTI in KTRs is reported as 39.4% (2)The known risk factors include female gender, deceased donor KT, kidney-pancreas transplantation, prolonged catheterization. Uretero-vesical stent and increase immunosuppressants. *E.coli* is the commonest causative organism in bacteria UTI and the should be treated for 7 -14 days. However, fungi UTI-Candiduria is better treated with fluconazole 200mg daily for 7-14 days(60, 61)

MycobacteriumTuberculosis (MTB)

Incidence of MTB infection among KTRs varies according to locality. It is about 5%-15% in India/Pakistan area(62) and about 0.45% in France(63, 64). In Nigeria, it is reported to be about 5.6%(2). It can occur in KTR who had negative tuberculin skin test reaction (mostly because of anergy in ESRD) and it often present as extrapulmonary lesion. Treatment of active disease is same as for non KTR (2months of rifampicin, isoniazid, ethambutol, and pyrazinamide then 4 months ofrifampicin and isoniazid) RIEP then 4mo RI). Avoidance Rifampicin is suggested as it activates cytochrome P3A4 (CYP3A4) pathway thereby markedly reducing the level of calcineurin inhibitors (CNIs)(62, 65, 66)

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