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Editorial

Recent Advances and Clinical Outcomes of Kidney Transplantation

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Abstract: Recent advances in surgical, immunosuppressive and monitoring protocols have led to the significant improvement of overall one-year kidney allograft outcomes. Nonetheless, there has not been a significant change in long-term kidney allograft outcomes. In fact, chronic and acute antibody-mediated rejection (ABMR) and non-immunological complications following kidney transplantation, including multiple incidences of primary kidney disease, as well as complications such as cardiovascular diseases, infections, and malignancy are the major factors that have contributed to the failure of kidney allografts. The use of molecular techniques to enhance histological diagnostics and noninvasive surveillance are what the latest studies in the field of clinical kidney transplant seem to mainly focus upon. Increasingly innovative approaches are being used to discover immunosuppressive methods to overcome critical sensitization, prevent the development of anti-human leukocyte antigen (HLA) antibodies, treat chronic active ABMR, and reduce non-immunological complications following kidney transplantation, such as the recurrence of primary kidney disease and other complications, such as cardiovascular diseases, infections, and malignancy. In the present era of utilizing electronic health records (EHRs), it is strongly believed that big data and artificial intelligence will reshape the research done on kidney transplantation in the near future. In addition, the utilization of telemedicine is increasing, providing benefits such as reaching out to kidney transplant patients in remote areas and helping to make scarce healthcare resources more accessible for kidney transplantation. In this article, we discuss the recent research developments in kidney transplants that may affect long-term allografts, as well as the survival of the patient. The latest developments in living kidney donation are also explored.

Keywords: kidney transplantation; renal transplantation; kidney transplant; renal transplant; transplant recipients; transplantation

1. Introduction

Kidney transplantation is the optimal treatment for improving survival and quality of life for patients with end-stage kidney disease (ESKD) [1]. Advances in surgical, immunosuppressive and monitoring protocols have led to a significant improvement in overall one-year kidney allograft survival of >95% [2]. Nonetheless, there has not been a significant change in long-term kidney allograft outcomes. In fact, chronic and acute antibody-mediated rejection (ABMR) has continued to cause kidney allograft failures [3]. In addition, non-immunological complications following kidney transplantation, such as the recurrence of primary kidney disease and other complications, such as cardiovascular diseases, infections, and malignancy also play important roles in poor long-term allografts and patient survival [4–6].

In their research into immunologic monitoring and diagnostics in kidney transplants [7–14], a number of groups have made attempts in the recent past towards determining the peripheral molecular fingerprints of ongoing rejection [7,8] and predicting acute rejection [7]. Contemporary researchers have measured the levels of donor-derived cell-free DNA (dd-cfDNA) and showed higher predictive abilities for acute rejection [9–12], especially antibody-mediated rejection (ABMR) diagnostics in cases with a combination of donor specific antibodies (DSA) and dd-cfDNA [13,14]. In addition, a molecular microscope diagnostic system for the evaluation of allograft biopsies has been recently introduced within transplant practice, particularly in complex cases. This has mainly been introduced for the purpose of enhancing histological diagnostics [15].

Recent studies have been conducted aimed at preventing or treating ABMR [16,17]. In 2017, imlifidase (IdeS), an endopeptidase derived from Streptococcus pyogenes, was utilized in a desensitization regimen in an open-label phase 1–2 trial [16]. An instant impact was observed by a significant decline in plasma IgG levels. Another single-center phase 2 study that focused mainly on the pharmacokinetics, effectiveness and safety of IdeS treatment was conducted and proved a reduction in anti-human leukocyte antigen (HLA) antibodies using a complement-dependent cytotoxicity test [17].

In recent years, there has been significant progress in research into kidney transplantation and kidney donation [18–84], including articles [20–60] published in our current Special Issue "Recent Advances and Clinical Outcomes of Kidney Transplantation" (https://www.mdpi.com/journal/jcm/special_issues/outcomes_kidney_transplantation).

In this article, we discuss the recent research developments in kidney transplantation that may impact long-term allografts and patient survival, as well as the latest developments in living kidney donation.

2. Non-HLA Antibodies in Transplantation

When it comes to solid organ transplantation, one major immunological obstacle is the detection the non-self structures that exist in the donor cells. Human leukocyte antigens (HLA) are considered the most important non-self allo-antigens in organ transplantation. In addition, patients can form antibodies against targets other than HLA [85]. Multiple targets for these non-HLA antibodies have been studied in kidney transplantation over the last decade (Figure 1). Recent studies have provided findings that suggest the an importance of non-HLA mismatches between donors and recipients in the development of acute rejection and long-term kidney allograft outcomes [68,78,86–92].

Types of Abs against Endothelial antigens in antibody-mediated immune responses	
Alloantibodies	ABO HLA MICA
Autoantibodies	AT1R ETAR Vimentin Perlecan Endoglin FLT3 ligand EDIL3 ICAM4 Fibronectin Collagen type 4

Figure 1. Post-transplant antibodies against human leukocyte antigen (HLA) and non-HLA antigens [68,78,86–92]. Abbreviations: human leukocyte antigen (HLA), major histocompatibility complex class I related chain A antigen (MICA); angiotensin type 1 receptor (AT1R); endothelin-1 type A receptor (Anti-ETAR); FMS-like tyrosine kinase 3 (FLT3); Epidermal growth factor-like repeats and discoidin I-like domain 3 (EDIL3); Intercellular adhesion molecule 4 (ICAM4).

3. Active AMR

Chronic active ABMR is one of the major causes of long-term allograft loss [93–95]. Tocilizumab, a humanized monoclonal antibody targeting the interleukin (IL)-6 receptor, has been assessed in patients with acute and chronic active ABMR [96–98], given that IL-6 mediates various inflammatory and immunomodulatory pathways, including the expansion and activation of T cells and B cells [98]. Furthermore, there is a genetically engineered humanized Immunoglobulin (Ig)G1 monoclonal antibody that binds to IL-6, inhibiting its interaction with IL-6R. Direct inactivation of IL-6 may limit a rebound induced by the accumulation of IL-6 [99,100]. Preliminary investigations from phase 1–2 trials demonstrated the efficacy of the C1q inhibitor for the prevention of a delayed graft function (DGF) and to lessen the occurrence of chronic active ABMR [101,102]. Although the inhibition of the first step in both the classical and lectin pathways of complement activation may serve as another tool to overcome critical sensitization, such data need to be validated in larger cohorts. Several trials are currently being conducted, and new developments will conceivably provide us with practical ways to counteract the deleterious consequences of ABMR [103].

4. Cardiovascular Diseases in Kidney Transplant Recipients

The burden of cardiovascular diseases on ESKD is improved after kidney transplantation [104]. However, it remains the leading cause of reduced early renal graft loss and mortality, as it is associated with significant morbidity and healthcare costs [104]. Major phenotypes of cardiovascular diseases among kidney transplant recipients include ischemic heart disease, congestive heart failure, valvular heart disease, arrhythmias and pulmonary hypertension (Figure 2).

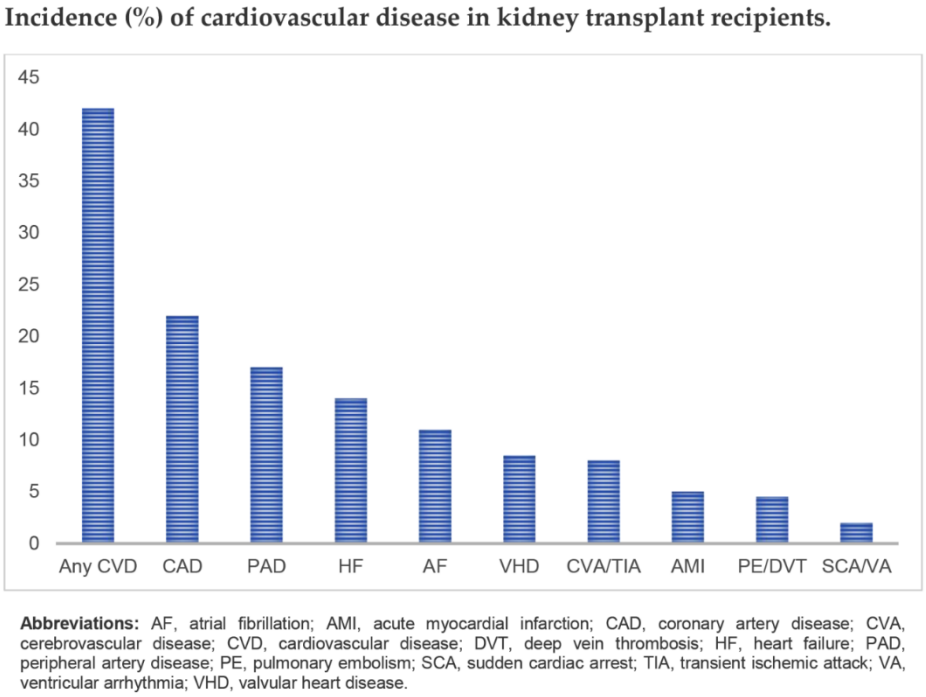

Figure 2. Incidence (%) of cardiovascular disease in kidney transplant recipients.

Reported risk factors for cardiovascular disease in kidney transplant recipients include inflammatory and immunosuppressive agents, episodes of allograft rejection, as well as traditional cardiovascular risk factors, such as hypertension, hyperlipidemia, smoking, obesity, chronic kidney disease, proteinuria, and diabetes mellitus, all of which add to a transplant recipient's cardiovascular risk profile [104]. Hypertension is common among kidney transplant recipients and uncontrolled hypertension in kidney transplant recipients is associated with increased cardiovascular mortality and morbidity, and reduced allograft survival [105]. Furthermore, weight gain is also a significant problem in post-kidney transplant patients. Weight gain after transplantation can unfavorably affect patient outcomes [106]. Identifying these risk factors and adopting strategies to abolish these risk factors may potentially prevent, and help manage, post-transplant obesity. The underlying mechanisms for the increased occurrence of dyslipidemia post-transplant are due to immunosuppressive medications, proteinuria, and post-transplant diabetes [107,108].

The medical management of risk factors includes strategies employed in the chronic kidney disease (CKD) population, with credence given to approaches specific for kidney transplant recipients, such as the choice of maintenance immunosuppression, steroid tapering or withdrawal, and particular anti-hypertensive regimens (Table 1). Overall, cardiovascular morbidity and mortality in kidney transplant recipients has decreased over the last few decades, likely due to improved detection and the timely management of risk factors. Recognition of these complications is important in assessing cardiovascular disease risk in kidney transplant recipients, and optimizing screening and therapeutic approaches. These include lifestyle and immunosuppressive regimen modification, as well as the best feasible regimen for glycemic and lipid controls according to an individual's metabolic profile and medical history.

Table 1. Cardiovascular risk factors among kidney transplant recipients and suggested management.

Cardiovascular Risk Factor	Suggested Management	Reference
Traditional risk factors		
Hypertension	• Monitor each visit • Target BP < 130/80 mmHg (ACC/AHA, 2017) • Initial treatment with CCB • ACEI/ARB if > 1 g/day proteinuria	[109–112]
Diabetes	• Monitor for post-transplant DM annually • Target HbA1c 7.0–7.5% (KDIGO, 2009) • Low-dose ASA in all atherosclerotic CVD	[109,113]
Cigarette smoking	• Screen annually • Offer intervention for smoke cessation	[109,114]
Dyslipidemia	• Monitor annually • Use of statins favored in all KTx (KDIGO, 2014)	[109,115]
Obesity	• Monitor BMI and weight circumference • Healthy diet and exercise • BMI target < 35 kg/m2	[109,116]
Non-traditional risk factors		
eGFR < 45 ml/min/1.73m2	• Increased use of living donor organs if possible • Check serum creatinine at least annually • Avoid nephrotoxic medications	[109,117]
Proteinuria	• ACEI/ARB if > 1 g/day proteinuria • Check urine analysis at least annually	[109,118,119]
Left ventricular hypertrophy	• Check ECG, echocardiography • Treat underlying hypertension	[109,120]
Anemia	• Treatment similar to CKD guidelines • Check CBC	[109,121,122]
Acute rejection episodes	• Treat rejections as per KDIGO, 2009	[109,123,124]

American College of Cardiology (ACC); angiotensin-converting enzyme inhibitor (ACEI); American Heart Association (AHA); angiotensin-II receptor blocker (ARB); aspirin (ASA); body mass index (BMI); blood pressure (BP); complete blood count (CBC); calcium-channel blockers (CCB); chronic kidney disease (CKD); cardiovascular disease (CVD); diabetes mellitus (DM); electrocardiography (ECG); estimated glomerular filtration rate (eGFR); Kidney Diseases Improving Global Outcomes (KDIGO); kidney transplant (KTx).

5. Preexisting Diabetes and Post-Transplantation Diabetes

Preexisting diabetes and post-transplantation diabetes confer reduced patient and graft survival in kidney transplant recipients [71,73,125]. Hyperglycemia is present in nearly 90% of kidney transplant recipients in the immediate postoperative period, but it is not sustained in the majority [126]. In addition to the general risk factors for diabetes, there are also certain transplantation-related factors (e.g., specific immunosuppressive agents, surgical stress and inflammation, nutritional interventions) placing kidney transplant recipients at elevated risk of hyperglycemia [126]. Some transplant immunosuppressive medications, including corticosteroids, calcineurin Inhibitors (CNIs), and mammalian target of rapamycin (mTOR) inhibitors, are associated with a higher incidence of metabolic complications such as post-transplantation diabetes. CNIs impair insulin secretion and sensitivity and directly damage pancreatic islet cells [127].

A robust evidence base guiding precise glycemic goals is currently lacking in kidney transplant recipients. Management is largely guided by evidence from the general diabetes population [71,73,125]. Hospital management of hyperglycemia is primarily achieved through an insulin regimen that takes into account rapid changes in glucocorticoid doses, nutritional modalities and renal function during the immediate post-transplantation period. There is an opportunity to use oral or non-insulin injectable agents in a considerable number of patients by the time they are discharged from the hospital, or in

the long run. The use of specific oral or non-insulin injectable agents is guided by patient specifics and the pharmacologic properties of medications. Although several studies have suggested the safe use of sodium glucose transport 2 (SGLT2) inhibitors in kidney transplant recipients [128], future studies assessing their efficacy and safety are needed, since SGLT2 inhibitor treatment also carries an increased risk of genital tract infections and, possibly, of urinary tract infections [129]; kidney transplant recipients are particularly susceptible to infections due to immunosuppressive regimens.

6. Posttransplant Malignancy

Cancer is one of the three major causes of death after kidney transplantation [130,131]. Posttransplant malignancy occurrence is widely recognized (Table 2). The effect of viral infections, induction and immunosuppressive maintenance regimens have been proposed as important risk factors for posttransplant malignancy. The increased risk of cancer may be due to viral reactivation induced by immunosuppressive agents or impaired immune surveillance leading to faster tumor growth [132]. A higher degree of immunosuppression is associated with an increased risk of malignancy, and calcineurin inhibitors can promote carcinogenesis [132].

Table 2. Standardized incidence ratio of cancers in kidney transplant recipients [133].

Cancer	Standardized Incidence Ratio (95% CI)
Lip cancer	29.45 (17.85–48.59)
Non-melanoma skin cancer	12.14 (6.37–23.13)
Renal cell carcinoma	10.77 (6.40–18.12)
Non-Hodgkin lymphoma	10.66 (8.54–13.31)
Thyroid cancer	5.04 (3.79–6.71)
Hodgkin lymphoma	4.90 (3.09–7.78)
Urinary bladder cancer	3.52 (1.48–8.37)
Melanoma	2.48 (1.08–5.67)
Hepatocellular carcinoma	2.45 (1.63–3.66)
Gastric cancer	1.93 (1.60–2.34)
Colon cancer	1.85 (1.53–2.23)
Lung cancer	1.68 (1.29–2.19)
Ovarian cancer	1.60 (1.23–2.07)
Pancreatic cancer	1.53 (1.23–1.91)
Breast cancer	1.11 (1.11–1.24)

Confidence Interval (CI).

7. Infection

Solid organ transplant recipients are at greater risk of infection than the non-immunosuppressed population (Table 3) [134]. Infections are the most common non-cardiovascular causes of mortality following kidney transplantation, accounting for 15%–20% of mortality [131,135]. The first six months post-transplant is the time of greatest infection risk. There are also times when patients encounter adverse reactions to immunosuppressive agents [136,137]. Among all infectious complications, viruses are considered to be the most common agents [138]. Herpes simplex virus, varicella zoster virus, BK polyomavirus, cytomegalovirus, Epstein–Barr virus, hepatitis B virus, and adenovirus are well-known etiologic agents of viral infections in kidney transplant patients worldwide [138]. In order to prevent opportunistic infections in kidney transplant recipients, antimicrobial prophylaxis is recommended after kidney transplantation. The recommended prophylactic method after transplant differs based on the organism, as well as individual patient characteristics.

Table 3. Infection post kidney transplantation.

<1 Month	1–6 Month	>6 Month
Bacterial infection *	**Bacterial infection**	**Bacterial infection**
• UTI (mainly E Coli, Enterobacteriaceae, Pseudomonas, Enterococcus) • Respiratory • Catheter, drainage sites, wound, perinephric fluid collection, urinary stent infections • Bacteremia • C diff colitis	• With prophylaxis ** -C diff colitis, Mycobacterium species • Without prophylaxis - Listeria, Nocardia	• UTI • Pneumonia
Viral infection	**Viral infection**	**Viral infection**
• HSV • Donor-derived—HIV, Hepatitis, CMV, BK, LCM virus, West Nile virus, Rabies	• With prophylaxis—BK, Adenovirus, Influenza, EBV, HCV, Parvovirus • Without prophylaxis—HSV, CMV, VZV,	• CMV (colitis or retinitis) • Hepatitis (B and C) • EBV, HSV, HHV-8, papillomavirus (associated with malignancy) • VZV, BK virus, parvovirus
Fungal infection	**Fungal infection**	**Fungal infection**
• Candida (can be donor derived or pre—TX colonization)	• With prophylaxis—Aspergillus, Cryptococcus, Mucor • Without prophylaxis—Pneumocystis jiroveci	• Cryptococcus, Rhodococcus, Aspergillus, pneumocystis, Mucor
Parasitic infection	**Parasitic infection**	
• Donor-derived—Malaria, Babesia, Balamuthia, T. cruzi	• Toxoplasma, Strongyloides, T. cruzi, Leishmaniasis	

* Center-dependent multidrug resistant bacteria like Methicillin-resistant Staphylococcus aureus (MRSA), Vancomycin-resistant Enterococcus (VRE), extended-spectrum beta-lactamases (ESBLs); ** With prophylaxis – with Bactrim and Gancyclovir/Valganciclovir; Abbreviations: cytomegalovirus (CMV), lymphocytic choriomeningitis virus (LCM), Epstein-Barr Virus (EBV), hepatitis B virus (HBV), hepatitis C virus (HCV), Trypanosoma cruzi (T. cruzi), Varicella Zoster virus (VZV), human herpes virus 8 (HHV-8).

8. Latest Developments in Living Kidney Donation

Living donor kidney transplants are the best option for many patients with ESKD for several reasons, including (1) better long-term graft survival, (2) no need to wait on the transplant waiting list for a kidney from a deceased donor, (3) transplant surgery can be planned and (4) lower risks of rejection and DGF [139]. Living donor kidney transplantation is the optimal treatment for patients with ESKD [139]. The expansion of living donor programs was made possible by new modes of living donation and by the extension of the living donor pool [139].

To expand the donor pool, a well-developed paired kidney donation program and the adequate reimbursement of costs associated with donation are fundamental elements [140]. Paired kidney donation provides living kidney donation for noncompatible donor/recipient pairs that otherwise would not be feasible or need desensitization [141]. Other possible approaches for increasing the donor pool include ABO-incompatible transplantation [142], the utilization of higher risk donors, advanced donation with a voucher system, and providing donors with financial incentives [141,143,144].

Over the past decade, the long-term risks of kidney donation have been described. Living donors seem to have a higher risk of ESKD, particularly in obese donors and also for African American donors with an apolipoprotein L1 (APOL1) high-risk genotype. In African American living kidney donors, those with the APOL1 high-risk genotype (prevalent in about 13% of African Americans in the United

States) had an almost three times more accelerated decline in estimated glomerular filtration rate (eGFR) after adjusting for pre-donation eGFR than those with a low-risk genotype [145].

9. Post-Transplant Hyperparathyroidism and Bone Disease

Successful renal transplantation results in a reduction in parathyroid hormone (PTH), especially during the first 3 months after transplantation [146]. However, elevated PTH levels can still be found in 30% to 60% of patients 1 year after transplantation. Persistent hyperparathyroidism following kidney transplantation can result in notable complications, such as fracture/bone diseases, cardiovascular disease, vascular calcification, and allograft dysfunction (Figure 3). Associated factors for persistent hyperparathyroidism are long dialysis duration, high PTH levels prior to transplantation, lower eGFR post-transplant, post-transplant hypercalcemia, and post-transplant high alkaline phosphatase.

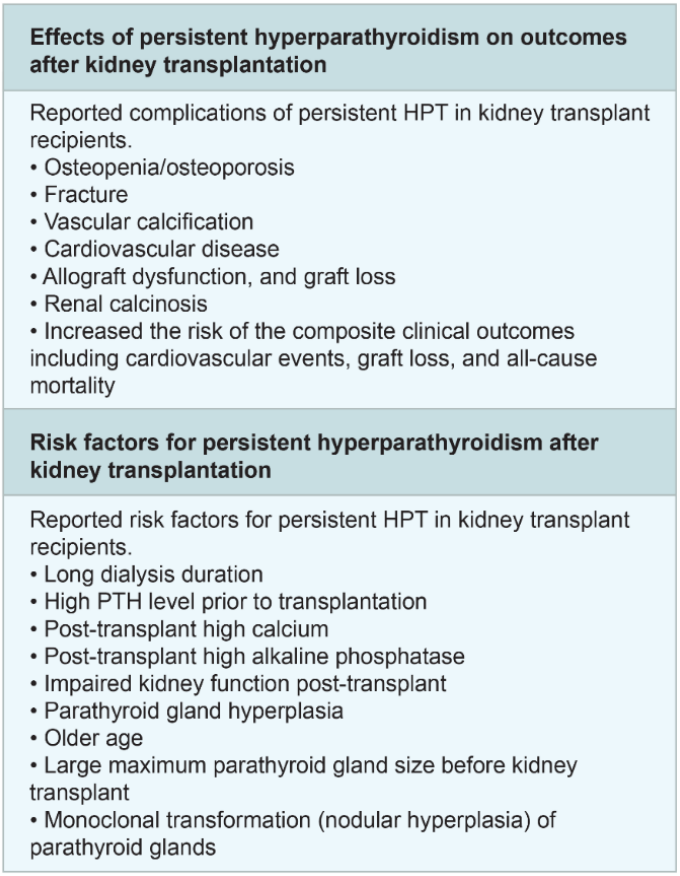

Figure 3. Effects and risk factors of post-transplant hyperparathyroidism.

10. Potential Directions and Future Scope

Researchers need to instantly shift their focus on the unaddressed concerns with respect to kidney transplants. Because of the limited supply of organs, numerous potential recipients still have to spend more time in dialysis, waiting for a transplant. Sensitization to HLA antigens inhibits the recipients' access to transplants, compromising the survival of the graft due to chronic and acute AMR. The publication of complete data from a multi-center second-phase test that explores how IdeS is useful in desensitization is underway (NCT02790437). The phase 3 trial, uncovering the impact of clazakizumab following transplantation, was launched recently, with the outcomes of the phase 2 trial to be released soon.

Moreover, the lack of experienced and skilled professionals could hinder the diagnostic correctness of complications following transplantation. Furthermore, medication non-adherence among patients

could increase the alloimune reaction. Notably, medical research on the costimulation blockade during kidney transplantation is underway. A randomized sixty-month multi-center study (CIRRUS, NCT03663335) in kidney transplant is also underway, with the aim of defining the range of dosage and assessing the tolerability, safety, and effectiveness of some newly developed anti-CD40 monoclonal antibodies in two distinct cohorts in comparison to a tacrolimus-based regimen. Recently, a phase 2a clinical trial, with the purpose of assessing how effective the dual costimulation blockade with anti-CD40 (VIB4920) is when combined with belatacept in kidney transplantation patients (NCT04046549), was registered.

Big data is increasingly being utilized, with the establishment of a large collection of cohorts and the usage of electronic health records (EHRs) in kidney transplantation and artificial intelligence, which might be useful in solving problems related to the survival analysis of patients who have gone through kidney transplantation [147–155]. In the present era, it is strongly believed that big data and artificial intelligence will greatly reshape the research done on kidney disease and, consequently, improve the general clinical practice of nephrology [156].

The benefits of telemedicine include reaching out to patients in remote areas and helping to make scarce healthcare resources more accessible. As telemedicine applications continue to proliferate, studies have demonstrated that telehealth for transplant care may be associated with a reduction in cost and time, and may also improve access to transplantation for ESKD patients [157,158].

11. Conclusions

The most recent endeavors in kidney transplantation tend to mainly focus on noninvasive monitoring, as well as the improvement of histological diagnostics with the aid of molecular techniques. Such studies offer creative means that can be used to find immunosuppressive agents, which can effectively overcome critical sensitization, prevent the creation of anti-HLA antibodies, treat chronic active ABMR, and reduce non-immunological complications following kidney transplantation, such as the recurrence of primary kidney disease and other complications, such as cardiovascular diseases, infections, and malignancy. In the present era of utilizing EHRs, it is strongly believed that big data and artificial intelligence will reshape the research done on kidney transplantation in the near future. In addition, the utilization of telemedicine is increasing, providing benefits such as reaching out to kidney transplant patients in remote areas and helping to make scarce healthcare resources more accessible for kidney transplantation.

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