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CURRENT APPROACH TO RENAL TRANSPLANTATION CANDIDATES AND POTENTIAL DONORS WITH VIRAL HEPATITIS

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ABSTRACT

Renal transplantation is the most beneficial treatment in patients with chronic kidney disease (CKD), increasing life expectancy and improving quality of life. A better understanding of organ and tissue functions, the development of surgical techniques, and new and effective immunosuppressive and antimicrobial drugs increase the success of transplantation. However, the number of renal transplantations from living and cadaveric donors is not at the desired frequency. Among the leading causes of the restrictions for transplantation are both the recipients' and donors' chronic diseases. While hepatitis B and C infections are a significant problem affecting the number and success of renal transplantations, the innovation of hepatitis C virus treatments has improved outcomes. Thus, the recipient and donor hepatitis B and C virus infections are no longer considered as relative contraindications for renal transplantation. This review discusses the management of patients and donors with hepatitis B and hepatitis C in renal transplantation.

Keywords: Renal Transplantation, Hepatitis B Virus, Hepatitis C Virus, Direct-acting antiviral agents

INTRODUCTION

Renal transplantation is currently the modality of choice among renal replacement therapies with the least morbidity, mortality, and cost. However, some patients can be challenging while evaluating their transplantation suitability and post-transplantation follow-up. Patients with chronic viral hepatitis are some of the most prominent cases in this group. The risk of reactivation or exacerbation and malignancy caused by chronic viral infections and the management of immunosuppressive agents requires careful patient selection and holistic care. The prevalence of the Hepatitis B virus (HBV) has decreased due to widespread vaccination programs [1]. On the other hand, current Hepatitis C virus (HCV) treatments are also quite promising. HCV infection has evolved into a treatable disease in most patients. According to the 2020 Turkey Registry data, the frequency of Hepatitis B surface antigen (HBsAg) positive patients is 2.4%, and the frequency of Hepatitis C antibody (anti-HCV) positive patients is 2.7% in the hemodialysis (HD) population [2]. Many measures are taken in dialysis centers to reduce these frequencies. These are, in general, strict observance of universal hygiene measures, strict disinfection and sterilization of hemodialysis devices, avoiding unnecessary blood transfusions, and vaccination against HBV. However, chronic viral hepatitis in this population will continue to be a problem in preparation for renal transplantation and the post-transplant period.

This review discusses the current aspects of patient renal transplantation management with HBV and HCV, the most common chronic viral infections in patients with end-stage renal disease (ESRD).

HEPATITIS B INFECTION AND RENAL TRANSPLANTATION

Hepatitis B virus (HBV) belongs to the Hepadnaviridae family and is a DNA virus, a pathogen for which curative treatment is not yet available. HBV primarily infects liver cells, is 100 times more contagious than the human immunodeficiency virus (HIV), and is detectable in the blood and in bodily fluids. HBV infection has a broad clinical presentation spectrum, from a silent latent infection embedded in the DNA of the infected individual to fulminant disease [3]. According to 2019 data from the World Health Organization (WHO), there are 296 million HBV-infected patients across the world, and it is estimated that 1.5 million incidental cases will be added each year [1].

Chronic HBV infection is defined as HbsAg positivity for more than six months, HBV DNA > 20,000 copies, continuous or occasional elevations in aminotransferases, and moderate to severe necroinflammation in the liver biopsy.4 Treatment for chronic HBV infection is determined by the quantity of serum HBV DNA, serum alanine aminotransferase (ALT) level and severity of the liver disease. Medical treatment indications of chronic HBV infection are shown in Table 1 [5].

Medications used to treat chronic HBV infection are interferon, lamivudine, adefovir, telbivudine, tenofovir disoproxil fumarate (TDF), entecavir, and tenofovir alafenamide (TAF), and their mechanisms of action are given in Table 2.

The short-term treatment goals are: inactivating virus replication (serum HBeAg, HBV-DNA negativity), prevention of infectivity, transaminase normalization, and improvement of symptoms.6 The medium-term objectives are: histological improvement and providing HBsAg negativity. Last but not least, the long-term goals are: the prevention of cirrhosis and hepatocellular cancer (HCC).7 The definitions of response to the treatment are shown in Table 3 [8].

HBV infection prevalence decreased in patients requiring renal replacement therapy after strict measures for prevention and widespread vaccination programs. According to the Turkish Society of Nephrology data, the prevalence of HBsAg positivity in 2020 was 2.4% in HD and 1.93% in peritoneal dialysis (PD) populations [2].

The risk of HBV reactivation is high in renal transplant recipients due to immunosuppression [9]. Especially in the high HBsAg group, this risk is even higher. However, patients with Hepatitis B e antigen (HBeAg) and/or HBV DNA positivity carry the highest risk among these patients. The highest reactivation rates in HbsAg-negative patients were reported in cases using agents such as rituximab, high-dose glucocorticoids, and tumor necrosis factor inhibitors [10]. Although the risk of reactivation is lower in patients with a protective level of anti-HBs positivity, the risk still exists. In clinical practice, antibodies against Hepatitis B core antigen (anti-HBc) should be screened before renal transplantation. Patients with isolated anti-HBc positivity should be evaluated, and preventive measures should be applied.

The anti-HBs serum levels of patients with CKD should be checked, and the patients should be vaccinated accordingly before transplantation [11]. Antibody response to the vaccine decreases in advanced stages of CKD. Thus, vaccination in the earlier stages can be a better implementation. Seroconversion after vaccination is about 95% in the general population and 50-73% in HD patients [12]. The recommended administration and dose is 40 μg into the deltoid muscle at 0, 1,2, and 6 months, with a response rate of about <85%.

In patients with a renal transplant from a donor with chronic or preexisting HBV infection and no prior HBV infection, the primary goal of treatment is to prevent de novo HBV infection [13]. However, the risk of de novo infection is usually low. High serum titers of HBsAg and HBV DNA of the donor pose risks. In renal transplant recipients with chronic or preexisting HBV infection, the primary goal is to prevent HBV reactivation and the worsening of liver disease.

HBSAG POSITIVE (CHRONIC HBV INFECTION) RECIPIENTS

HBsAg positive patients should be evaluated for HBeAg positivity, quantitative serum HBV DNA level, and the presence of cirrhosis. Chronic HBV infection, including patients with compensated cirrhosis, is not a contraindication for renal transplantation.

Table 1. *Medical Treatment Indications of Chronic HBV Infection*

Recommendations for Medical Treatment	Level of Evidence	Grade of Recommendation
Should be treated		
HBeAg-positive or -negative CHB (HBV DNA >2,000 IU/ml, ALT > ULN and/or at least moderate liver necroinflammation or fibrosis)	I	1
Cirrhotic patients with detectable HBV DNA, regardless of ALT level	I	1
HBV DNA >20,000 IU/mL and ALT >2x ULN, regardless of histology	II-2	1
Can be treated		
>30 years HBeAg-positive CHB (normal ALT and elevated HBV DNA), regardless of histology	III	2
May be treated		
CHB patients with a family history of HCC or cirrhosis	III	2

HBeAg: Hepatitis B e antigen; CHB: Chronic Hepatitis B infection; ALT: Alanine aminotransferase; ULN: Upper limit of normal range; HBV: Hepatitis B virus; HCC: Hepatocellular cancer

Table 2. *Medications used to treat chronic HBV infection* [6, 7].

Interferon	Affects the HBV life cycle at many stages, such as HBV entry into the cell, virion uncoating, DNA transcription, and collection of viral particles. In CHB, pegylated interferon alfa-2a 180 micrograms are applied subcutaneously once a week for 48 weeks.
Lamivudine	Cytidine analog: competitively inhibits the reverse transcriptase enzyme and terminates viral DNA chain elongation. In patients with normal renal function, the daily dose is 100 mg. Lamivudine has a mild side effect profile. Drug resistance in long-term use and consequent virological and biochemical exacerbations limit its use.
Adefovir	A prodrug with a nucleotide analog. Since resistance rates are high, used as an add-on treatment to lamivudine-resistant cases. The most critical side effect of adefovir is dose-dependent renal toxicity. It may cause Fanconi Syndrome by creating proximal tubular dysfunction, elevated creatinine values, and hypophosphatemia.
Telbivudine	A thymidine nucleoside analog, providing a more potent viral inhibition than lamivudine and adefovir. The high frequency of drug resistance development limits its use.
Tenofovir Disoproxil Fumarate (TDF)	A nucleotide analog prodrug, which received FDA approval in 2001 for the treatment of HIV and in 2008 for the treatment of CHB. There is no identified drug resistance. The most common side effects are: headache, nasopharyngitis, back pain, and nausea. With long-term use of TDF, osteomalacia, decrease in bone mineral density, renal impairment, and hypophosphatemia.
Entecavir	Guanosine nucleoside analogue. It inhibits viral replication in 3 stages, and the frequency of drug resistance is low. It is given at a dose of 0.5 mg in naive patients and 1 mg/day in Lamivudine-resistant patients. Side effects due to entecavir are not dose-dependent, and side effects are similar in doses of 0.5 mg and 1 mg.
Tenofovir Alafenamide (TAF)	At least as effective as TDF in chronic HBV patients. It is safer than TDF in terms of side effect profile.

Table 3. *Definitions of response to the treatment in HBV infection.*

Virologic response	The reduction of HBV DNA <10 IU/ml (undetectable) with treatment. *Permanent response after treatment*: <2,000 IU/ml of HBV DNA after 12 months of treatment. *Primary non-response*: Less than 1 log drop in DNA despite three months of treatment *Partial response:* Detectable HBV DNA despite more than 1 log reduction in DNA at one year *Virological* progression: Detection of HBV DNA 1 log higher than the lowest detectable HBV DNA level under treatment
Serologic Response	*For HBeAg;* HBeAg loss and development of seroconversion in HBeAg-positive patients *For HBsAg;* Loss of HBsAg and development of anti-HBs.
Biochemical Response	ALT normalization with treatment (<40 IU/ml) Persistent biochemical response: ALT within the reference range measured at three-month intervals for one year
Histologic Response	Improvements in necroinflammatory activity and a rise of ≥2 scores Ishak Fibrosis Score after treatment.

ISOLATED ANTI-HBC POSITIVE RECIPIENT

It is necessary to measure the quantitative serum HBV DNA in such patients. The vast majority of patients are HBV DNA negative, and it is reasonable to give a booster dose of the hepatitis B vaccine to these patients. If the recipient develops an anti-HBs titer of ≥10 mIU/mL, they will likely have had a prior HBV infection.14 However, if there is no response to the booster dose, the patient is likely to have chronic HBV infection with loss of HBsAg or an occult HBV infection. Cirrhosis should be investigated considering clinical or biochemical evaluation if HBV DNA is detected in the patient's serum.

Potential kidney donors should be tested for HBsAg, anti-HBc, and HBV DNA. Tests should be done within 28 days before organ harvesting for living donors. For cadaveric donors, tests should be done 96 hours before organ harvesting [14]/ This may not be possible in clinical practice. Evaluation of HBV DNA results may be delayed. If the serum quantitative HBV DNA measurement is not available, an HBV DNA test may be reserved for HBsAg or anti-HBc positive donors.

RECIPIENTS WITHOUT PREVIOUS HBV INFECTION (INCLUDING VACCINATED PATIENTS)

Renal transplantation from donors with chronic HBV infection should be avoided. However, transplantation may be reasonable in cases where there is an urgent need. The de novo HBV infection risk is low, but there is still a risk. Hepatitis B Immunoglobulin (HBIG) is administered to recipients who receive a kidney from a donor with chronic HBV and no prior HBV immunity or infection [15]. This provides passive immunity to cover the period with the most risk of transmission, similar to preventing acute hepatitis B infection after exposure or perinatal transmission. The utility of perioperative HBIG in preventing HBV transmission in vaccinated HBV-immune kidney recipients is unclear. Still, it may be considered perioperatively if the anti-HBs level of the recipient is below 100 mIU/mL.

Antiviral therapy should be initiated at the time of transplantation [16]. The optimal duration of treatment is unknown. It is generally applied for one year. HBsAg, anti-HBc, and HBV DNA levels should be checked every three months [15, 16]. If these parameters remain negative after one year, antiviral therapy can be discontinued. After discontinuation of treatment, HBsAg and HBV DNA monitoring frequency is gradually reduced from every three months to annual [16].

RECIPIENTS WITH PREVIOUS OR CHRONIC HBV INFECTION

All recipients with chronic or preexisting HBV infection should receive antiviral therapy. The risk of HBV reactivation following transplantation is related to the recipient's condition at the time of renal transplantation (i.e., HBsAg positive or negative) and the type of immunosuppressive

therapy used. All patients with chronic or preexisting HBV infection (i.e., anti-HBc positive) and receiving rituximab (even a single dose) should initiate preventive antiviral therapy, regardless of HBsAg or anti-HBs status. Antiviral therapy is indicated in HBsAg negative and anti-HBc positive patients, even if anti-HBs is positive. Entecavir or TAD is recommended to prevent HBV reactivation [17, 18]. Therefore, antiviral treatment should be initiated at the time of transplantation. The duration of antiviral therapy may vary in patients with preexisting HBV, and lifelong antiviral treatment is required.

Recipients with a chronic hepatitis B virus infection should receive antiviral therapy indefinitely after transplantation [17, 18]. Patients who previously received entecavir or TAD can be treated with the same agent. Treatment of patients receiving lamivudine should be converted to TAD. Entecavir or TAD should be started if no previous treatment has been received [17, 18]. Recipients receiving antiviral therapy should have their serum aminotransferases and HBV DNA monitored every six months or more frequently. Some centers may discontinue treatment in recipients with preexisting HBV infection when immunosuppressive doses are decreased. If antiviral therapy is discontinued, aminotransferases, HBV DNA, and HBsAg are monitored every three months for the following year. For patients with normal liver enzymes, monitoring every six months after the first year may be reasonable. However, more frequent monitoring should be continued if the immunosuppressive regimen is intensified. In addition, if an agent such as rituximab is added to the treatment, preemptive antiviral therapy should be restarted. Interferon therapy should not be used in renal transplant patients as it may trigger acute allograft rejection [6].

Medication doses for HBV treatment should be adjusted for the renal transplant patients' glomerular filtration rate (GFR). Recommended doses are shown in Table 4 [18].

Studies show that renal transplant recipients with HBV infection have a shorter life expectancy than those without HBV infection. However, reports indicate that the first five-year life expectancy is not affected by HBV infection. Ng et al. compared 38 HBsAg positive patients with the same number of negative patients after renal transplantation. They found that the graft loss rate was higher in the HBsAg positive group. The mean graft survival was 12 years in the HBsAg negative group and 11 years in the positive group. Proteinuria, glomerular filtration rates, and patient survival rates were similar [19]. In a study by Wang et al., 83 HBsAg positive and 384 HbsAg negative donor transplants with similar characteristics were compared, and excellent graft and patient survival rates were observed in the 36-month follow-up. Hepatitis transmission was not observed [20].

Appropriate and prompt antiviral treatment has a positive effect on life expectancy. However, it is reported that renal transplantation under appropriate antiviral therapy may also positively affect life expectancy in compensated cirrhotic cases, but data are quite limited. For example, in a single-center study, 12 cirrhotic patients with HBV infection had an excellent five-year survival rate (94%) after renal transplantation under antiviral therapy [21].

HEPATITIS C INFECTION AND RENAL TRANSPLANTATION

Hepatitis C virus (HCV) is an enveloped, single-stranded, positive-sense RNA virus, a member of the Flaviviridae family. HCV does not enter the cell nucleus during replication, and unlike HBV and HIV, complete eradication is possible [22]. There are six genotypes (1-6) and subtypes (a, b, c). According to WHO data, as of 2019, there are 58 million people with HCV worldwide and 1.5 million new patients are added every year.1 HCV infection causes 55-85% chronicity and cirrhosis in 5-25% of the chronically affected cases [23].

One of the most important developments in medicine for HCV treatment arose in the last decade. HCV infection, a cause of high chronicity, cirrhosis, and hepatocellular carcinoma, has now turned into a disease that can be cured in over 95% of the cases. According to previous data, HCV infection was associated with a poor prognosis after renal transplantation [24]. In addition, interferon-based treatments were not curative in most cases and triggered acute rejection. Today, it is observed that very successful outcomes can be obtained in renal transplant patients with direct-acting antiviral agents (DAA) without any impairment in the graft function. In the light of all these developments and ongoing clinical research, the Kidney Disease Improving Global Outcomes (KDIGO) Clinical Practice Guideline

Table 4. *Dose Adjustements for Glomerular Filtration Rates in HBV Treatment*

ENTECAVIR (Nucleozide, Pure)	
GFR	**Dosage**
≥50	0,5 mg daily
30 to 49	0,25 mg daily or 0,5 mg every 48 hours
10 to 29	0,15 mg daily or 0,5 mg every 72 hours
<10 or hemodilysis/peritoneal dialysis	0,05 mg daily or 0,5 mg every 7 days
ENTECAVIR for LAMIVUDINE REFRACTORINESS/RESISTANCE	
GFR	**Dosage**
≥50	1 mg daily
30 to 49	0,5 mg daily or 1 mg every 48 hours
10 to 29	0,3 mg daily or 1 mg every 72 hours
<10 or hemodialysis/ continuous ambulatory peritoneal dialysis	0,1 mg daily or 1 mg every 7 days
TENOFOVIR DISOPROXIL FUMARATE (TDF, Nucleotide)	
GFR	**Dosage**
≥50	300 mg daily
30 to 49	300 mg every 48 hours
10 to 29	300 mg every 72 or 96 hours
<10 with hemodialysis	300 mg once a week or after a total of approximately 12 hours of dialysis
<10, without hemodialysis	Insufficient data; no recommendations.
Continuous ambulatory peritoneal dialysis	Insufficient data; no recommendations.
TENOFOVIR ALAFENAMIDE (TAF, Nucleotide)	
GFR	**Dosage**
≥15	25 mg daily
<15 with hemodialysis	25 mg; on hemodialysis days, after hemodialysis
<15 without hemodialysis	Insufficient data; no recommendations.
LAMIVUDINE (3TC, Nucleoside)	
GFR	**Dosage**
≥50	100 mg daily
30 to 49	100 mg first dose, than 50 mg daily
15 to 29	100 mg first dose, than 25 mg daily
5 to 14	35 mg first dose, than 15 mg daily
<5 or hemodialysis/ continuous ambulatory peritoneal dialysis	35 mg first dose, than 10 mg daily
ADEFOVIR DIPIVOXYL (Nucleotide)	
GFR	**Dosage**
≥50	10 mg daily
30 to 49	10 mg every 48 hours
10 to 29	10 mg every 72 hours
<15 without hemodialysis	Insufficient data; no recommendations.
Hemodialysis	10 mg every 7 days, after hemodialysis
Continuous ambulatory peritoneal dialysis	Insufficient data; no recommendations.

GFR: Glomerular Filtration Rate

for the Prevention, Diagnosis, Evaluation, and Treatment of Hepatitis C in Chronic Kidney Disease was published in 2018 for the management of HCV-infected renal transplantation candidates and recipients [25].

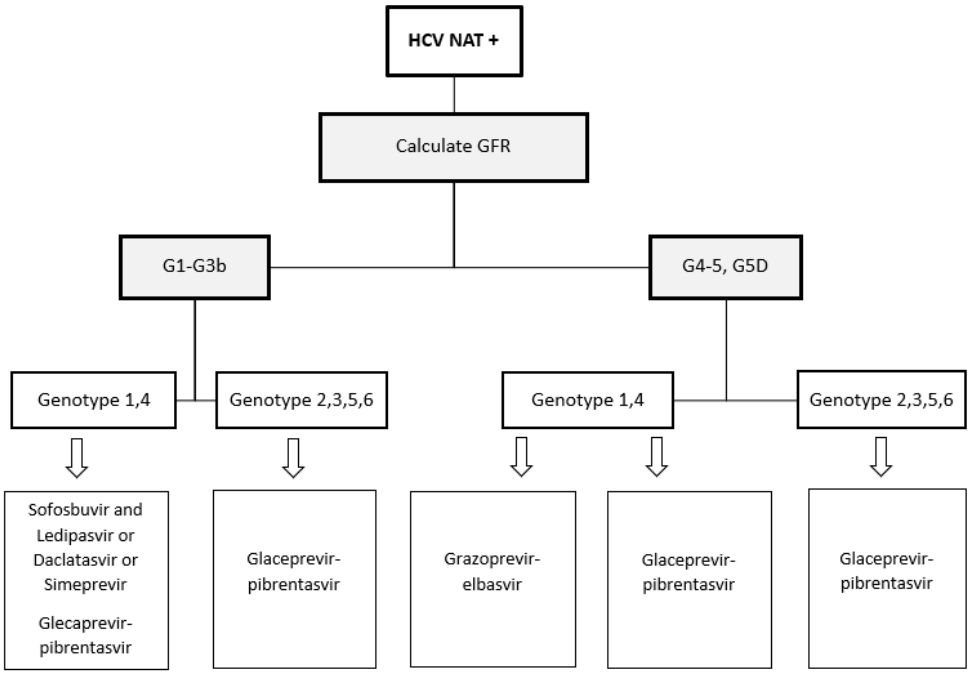
Every renal transplant candidate should be screened for HCV infection. HCV RNA should be checked in patients with true HCV antibody positivity [25]. Patients with HCV infection should be screened for cirrhosis by non-invasive or invasive methods [25]. Renal transplantation is associated with a poor prognosis in cirrhotic patients. Combined liver-kidney transplantation can be considered in cirrhotic patients, with multidisciplinary evaluation on a case-by-case basis [25]. However, limited data indicate the superiority of combined transplantation over renal transplantation alone in compensated cirrhotic cases. The 2008 American Combined Liver-Kidney Transplantation Consensus Congress reported that the subjects with portal vein pressure ≥10 mmHg are the most likely to benefit from combined transplantation [26]. However, in many countries, patients may not have access to liver transplantation on this criterion alone. Therefore, the decision should be made on a region and case basis.

Although there has been a decrease in post-transplantation complications in HCV-infected patients, such complications are still observed. These complications are: liver disease after renal transplantation, de novo or recurrent glomerular diseases, new-onset diabetes after transplantation, and lymphoproliferative diseases [25]. The most common glomerular diseases associated with HCV after renal transplantation are membranous glomerulonephritis, membranoproliferative glomerulonephritis, cryoglobulinemia, and thrombotic microangiopathies [27–29]. HCV infection is also associated with transplant glomerulopathies. Therefore, it is crucial to monitor proteinuria and hematuria in HCV-positive renal transplant recipients.

It is not clear whether the antiviral treatment of the HCV-infected patients should be started in the pretransplant period or postponed for the post-transplant period with improved kidney functions and broader DAA options. If the patient has stable liver disease and no extrahepatic disease, it may be appropriate to start treatment in the early post-transplantation period [25]. However, if the patient requires HCV treatment while waiting for transplantation, among the agents used today, glecaprevir-pibrentasvir is effective in all genotypes, and elbasvir-grazoprevir-ombitasvir and ombitasvir-paritaprevir-ritonavir combinations are effective in genotypes 1 and 4.25 These agents do not require renal dosing and can be used in the pretransplant period. The first-line treatments recommended for renal transplant recipients in the KDIGO 2018 HCV Guidelines for Chronic Kidney Disease are summarized in Figure 1 [25].

Figure 1. Treatment Scheme for Renal Transplant Recipients

NAT: Nucleic Acid Amplification Testing, GFR: Glomerular Filtration Rate



Although the kidney function is usually sufficient after renal transplantation to allow broader treatment options, the most crucial point in the use of DAA is the drug interactions. Cyclosporine can significantly increase the level of glaveprevir-pibrentasvir and elbasvir-grazoprevir combinations, and high-dose cyclosporine is not preferred with these agents. Cyclosporine is contraindicated with the elbasvir-grazoprevir combination for the same reason. These DAA combinations increase tacrolimus and everolimus serum levels [30, 31]. Particularly, tacrolimus can reach very high serum levels. The ombitasvir-paritaprevir-ritonavir-dasabuvir combination is contraindicated in renal transplantation, as these are the most likely to interact with agents used in renal transplantation. Therefore, immunosuppressive drug levels should be closely monitored in these patients. However, sofosbuvir and daclatasvir do not interact with the calcineurin inhibitors, everolimus, sirolimus, and mycophenolate mofetil, and many studies have recently been published showing the efficacy and safety of these agents in renal transplantation [31]. In addition to immunosuppressives, antibiotics and antifungal agents are frequently used in renal transplant recipients, and interactions should also be checked for these agents.

Morbidity and mortality rates are higher in HCV-infected patients than HCV-negative renal transplant recipients. However, transplantation is associated with better life expectancy than HCV-positive dialysis patients. Although it is not yet clear how close the mortality rates are in HCV-positive patients is with DAA regimens compared to HCV-negative recipients, it has been shown that the morbidity of the patients is reduced. Renal transplantation is also the best option in HCV-positive end-stage renal disease patients unless there are definite contraindications.

CONCLUSIONS

Chronic viral hepatitis in renal transplantation candidates and recipients may be challenging in many ways, especially those who require close follow-up and a multidisciplinary approach. In the light of new treatment options, publications, and guidelines, renal transplantation is the treatment of choice for patients with ESRD with chronic viral infections in carefully selected patients. The issues to be considered are: the timing of transplantation, the evaluation of treatment requirement, selection of antiviral drugs, the decision to administer induction therapy, close follow-up of maintenance immunosuppressive agents and renal functions, screening for infection, and malignancy in this patient group. Renal transplantation in eligible patients reduces morbidity, mortality, and treatment costs, and renal transplantation should be supported in this population. And finally, essential treatment of chronic viral hepatitis should be available and programmed if the patient is being transplanted via the "primum non nocere" principle.

ALT, Alanine aminotransferase
Anti-HBc, Hepatitis B core antigen
Anti-HCV, Hepatitis C antibody
CKD, Chronic kidney disease
DAA, Direct-acting antiviral agents
DNA, deoxyribonucleic acid
ESRD, End stage renal disease
GFR, Glomerular filtration rate
HBsAg, Hepatitis B surface antigen
HBV, Hepatitis B virus
HCC, Hepatocellular cancer
HCV, Hepatitis C virus
HD, Hemodialysis
HIV, Human immunodeficiency virus
KDIGO, Kidney Disease Improving Global Outcomes
PD, Peritoneal dialysis
RNA, Ribonucleic acid
TAF, Tenofovir alafenamide
TDF, Tenofovir disoproxil fumarate
WHO, World Health Organization

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Резиме

ТЕКОВЕН ПРИСТАП КОН КАНДИДАТИТЕ ЗА РЕНАЛНА ТРАНСПЛАНТАЦИЈА И ПОТЕНЦИЈАЛНИТЕ ДОНАТОРИ СО ВИРУСЕН ХЕПАТИТ

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Реналната трансплантација е најкорисен третман кај пациентите со хронична бубрежна болест (ХББ) со зголемувањето на животниот век и подобрувањето на квалитетот на животот. Подоброто разбирање на функциите на органите и на ткивата, развојот на хируршки техники и нови и ефективни имуносупресивни и антимикробни лекови го зголемуаат успехот на трансплантацијата. Сепак, бројот на ренални трансплантации од живи и кадаверски донатори не е на саканата фреквенција. Меѓу водечките причини за ограничувањата за трансплантацијата се хроничните болести и на примателите и на донаторите. Додека инфекциите со хепатитис Б и Ц се значаен проблем што влијае на бројот и на успехот на реналните трансплантации, иновацијата на третманите на хепатитис со вирусот Ц ги подобрија резултатите. Така, вирусните инфекции со хепатитис Б и Ц на примателот и на донаторот повеќе не се сметаат за релативни контраиндикации за ренална трансплантација. Овој преглед зборува за менаџирањето со пациентите и донаторите со хепатитис Б и хепатитис Ц при ренална трансплантација.

Клучни зборови: ренална трансплантација, вирус на хепатитис Б, вирус на хепатитис Ц, антивирусни агенси со директно дејство