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Outcome of kidney transplant recipients with a history of hepatitis B and C viruses: A single-center cohort study.

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Abstract

Introducción: Kidney transplantation (KT) in patients with viral hepatitis (HBV and HCV) represents a significant challenge, given its high prevalence and its contribution to important posttransplant complications. The objective of this study is to evaluate the posttransplant evolution of patients who were treated and cured of hepatitis B or C before receiving the transplant.

Methods: We conducted a retrospective study at the Ibn Sina University Hospital, Rabat, over 35 years (1983–2018). The study included 20 kidney transplant recipients with cured HBV or HCV before transplantation. Patients were divided into two groups: Group A (no viral reactivation) and Group B (viral reactivation). Various factors, including demographic, clinical, immunosuppressive, and virological parameters, were analyzed.

Results: Of the 20 patients, 16 had cured HCV and 4 had cured HBV. The median age at transplantation was 38.5 years. Among the 8 patients with HCV reactivation, only one had received direct-acting antiviral treatment and achieved a sustained virological response. HBV reactivation occurred in 3 patients, leading to graft rejection in 2. Reactivation of either virus was associated with proteinuria, graft rejection, and return to dialysis. The overall mortality rate was 15%, with two deaths linked to viral reactivation.

Conclusions: Kidney transplant recipients with a history of HBV or HCV face increased risks of graft rejection, proteinuria, and graft loss. Viral reactivation is associated with poor outcomes, underscoring the importance of pretransplant antiviral treatment in improving graft survival. Although viral hepatitis prevalence has decreased due to improved hygiene and vaccination, these infections remain a significant risk factor for kidney graft failure.

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Hepatitis C, hepatitis B, kidney transplant, acute rejection, graft loss.

Kidney transplantation (KT) is the preferred kidney replacement therapy for managing end-stage chronic kidney disease (ESKD), offering improved quality of life and enhanced survival [1]. However, viral hepatitis, particularly hepatitis B (HBV) and hepatitis C (HCV) [2], is a significant challenge. These infections, which are common among dialysis patients, have prevalence rates that vary by geographic region, ranging from 9% to 20% for HBV and 10% to 65% for HCV [3, 4]. They contribute to posttransplant complications such as de novo glomerulonephritis (GN), chronic allograft nephropathy, and posttransplant diabetes mellitus [5-9].

The objective of this study was to assess the posttransplant evolution of patients treated for hepatitis B or C and cured before transplantation.

Materials and methods

Study type and period

This was a retrospective, descriptive, single-center study conducted at the kidney transplant unit of Ibn Sina University Hospital in Rabat, spanning 35 years from December 1983 to February 2018.

Study population

1. All kidney transplant patients from either a living donor or a brain-dead donor with a negative HBV or HCV PCR at the time of transplantation and a follow-up of at least 5 years were included.
2. The exclusion criteria were as follows: patient records with missing data and those lost to follow-up.

We thus distinguished two groups of patients. :

Group A: Patients without viral reactivation of hepatitis B and C after kidney transplantation.

Group B: Patients with hepatitis B and C viral reactivation after kidney transplantation.

Analyzed variables and definitions

We analyzed the following parameters for each patient:

Demographics: the recipient's age at the time of transplantation, the donor's age, and sex.

Clinical: initial nephropathy and comorbidities (diabetes, hypertension, heart disease). Other infections (bacterial, viral, or parasitic), pretransplantation replacement therapy (hemodialysis, peritoneal dialysis, preemptive transplantation), history of blood transfusion, hepatitis B and C, type of graft (living donor kidney, brain-dead donor kidney), episodes of rejection, and de novo glomerulonephritis.

Therapeutic: We specified the induction and maintenance immunosuppressive protocol, as well as the antiviral treatment received before and after kidney transplantation.

Biologically, the following parameters were analyzed: liver enzymes (ASAT, ALAT, PAL, GGT), alpha-fetoprotein, renal function (assessed by blood creatinine levels and estimated glomerular filtration rate (eGFR) via the MDRD formula), and 24-hour proteinuria.

Radiologically, abdominal ultrasound was performed on all patients to check for signs of portal hypertension and liver cirrhosis. The FibroScan measures liver elasticity, thereby assessing the degree of fibrosis in certain patients.

Histological: We studied all liver biopsies performed on the patients as well as graft biopsies collected during the study period.

Serological: We searched for the following parameters in our patients before and after kidney transplantation:

- Hepatitis C virus (HCV):
- The search for anti-HCV antibodies was performed via third- and fourth-generation ELISA techniques.
- HCV infection was confirmed via qualitative PCR (Cobas-Amplicor ROCHE test with a detection threshold of 10 U/ml HCV RNA), followed by quantitative PCR (AmpliPrep/Cobas TaqMan HCV-Roche Diagnostics test, with a detection threshold of 15 IU/ml HCV RNA). Genotypes 1a and 1b were the predominant genotypes.
- Hepatitis B virus (HBV):

We systematically tested all our patients for HBsAg via the ELISA technique, as well as for anti-HBc antibodies, anti-HBs antibodies, and HBV PCR.

Outcome criteria

- HBV- or HCV-positive PCR posttransplantation with a positivity threshold of 20 IU/ml HBV PCR and 12 to 15 IU/ml HCV PCR.

Study size

The sample was nonprobabilistic. All incident data from the study period are reported.

Statistical data analysis

The statistical analysis was performed via JAMOVI software version 2.3.28. Qualitative data are expressed as frequencies (count percentages), and quantitative data are expressed as the means ± standard deviations or medians and interquartile ranges. The data were compared via the chi-square test. A P value of <0.05 was considered to indicate statistical significance.

Results

Participants

Among our 161 kidney transplant recipients, 20 patients were included, of whom 16 had declared cured chronic hepatitis C before transplantation and 4 had declared cured hepatitis B before transplantation.

Group Description

The median age of our patients at the time of transplantation was 38.5 years [28.5--48.5], with extremes ranging from 15--61 years. There was a male predominance: 14 men (70%) and six women, with a male-to-female ratio of 2.3.

Glomerular nephropathy (6 cases) was the most common, with 2 cases secondary to hepatitis B and C, followed by chronic tubulointerstitial nephritis (3 cases), nephrosclerosis (2 cases), polycystic kidney disease, and diabetic nephropathy (1 case each). The cause was undetermined in 7 patients.

Eighteen patients were on chronic hemodialysis, with an average duration of dialysis of 60 ± 15 months, ranging from 12--252 months, whereas two patients received preemptive kidney transplants.

Fourteen (70%) patients received transplants from a living donor, and 6 (30%) patients received transplants from a brain-dead donor (BD).

Among the 20 patients included in the study, 8 (40%) were diabetic, 18 (90%) were hypertensive, 6 (30%) had a history of tuberculosis, and 2 (10%) had heart disease (Table 1).

Table 1. Comorbidities of the patients included.

Comorbidity	Frequency	Percentage
Hypertension	18	90%
Hyperparathyroidism	9	45%
Diabetes	8	40%
History of tuberculosis	6	30%
Cardiopathy	2	10%
Immunosuppression		
ATG + Methylprednisolon	8	40%
Basiliximab + Methylprednisolone	2	10%
Methylprednisolon	10	50%
History of tuberculosis	6	30%
Cardiopathy	2	10%

The immunosuppressive protocol used

The induction treatment consisted of basiliximab in two patients (10%) at a standard dose of 40 mg (day 0 and day 4), combined with a methylprednisolone bolus, and anti-lymphocyte serum in eight patients (40%) at a total average dose of 4 ± 1.5 mg/kg, which was also combined with a methylprednisolone bolus. Ten patients (50%) received only a methylprednisolone bolus as induction therapy. The maintenance immunosuppressive treatment was based on the following combinations:

- Prednisone + Cyclosporine + Mycophenolate mofetil in 9 patients (45%)
- Prednisone + tacrolimus + mycophenolate mofetil in 7 patients (35%)
- Prednisone + azathioprine + cyclosporine was used in 4 patients (20%).

Treatment of hepatitis C before kidney transplantation

Table 2 shows the treatment received for hepatitis C virus (HCV) before kidney transplantation. Among the five patients who experienced HCV reactivation, only one patient had received direct-acting antiviral (DAA) treatment with sofosbuvir and daclatasvir, achieving a 100% sustained virological response (SVR). The other cases of HCV reactivation were not treated because of contraindications to interferon-alpha (IFNα) therapy and the unavailability of DAAs. Among the four untreated patients, three lost their grafts and returned to dialysis, whereas one patient continued to experience chronic graft dysfunction with a creatinine clearance of 38 ml/min.

Treatment of hepatitis B before kidney transplantation

Table 3 shows the treatments received for hepatitis B virus infection before kidney transplantation. All of our patients were positive for anti-HBc antibodies but negative for HBsAg. The follow-up primarily involves close monitoring (quarterly) of serological markers for HBV [PCR (HBV), HBsAg, HBeAg] and HCV [PCR (HCV)], with annual liver ultrasound control.

Three patients experienced HBV reactivation after an average postkidney transplantation duration of 10.6 months, and five patients experienced HCV reactivation after an average of 52.4 months postkidney transplantation. There were no cases of acute hepatitis or hepatic cell failure.

All patients with HBV reactivation received antiviral treatment. Owing to resistance to lamivudine, two patients were treated with entecavir and adefovir. One patient was treated initially with entecavir. After treatment for reactivation, only one patient achieved a negative PCR result (Patient No. 4). The three patients with HBV reactivation posttransplantation experienced graft rejection and returned to dialysis.

Table 2. Treatment of hepatitis C before kidney transplantation.

Cases	Therapeutic molecules:	Evolution
Case N°1	Interferon (6 months) Pegylated Interfer (18 months) Interferon pegilado + Rivabirina	Hematological intolerance Negative PCR after 9 months Negative PCR after 4 weeks
Case N°2	Pegylated Interfer (12 months)	Negative PCR
Case N°3	Interferon (12 months)	Negative PCR
Case N°4	Interferon (6 months) Interferón + Ribavirina Interféron (24 months) + Sofosbuvir (21 months)	Treatment failure Treatment failure Negative PCR
Case N°5	Interferon+Ribavirin (6 months)	Negative PCR
Case N°6	Interferon (12 months)	Negative PCR
Case N°7	Pegylated Interféron (6 months)	Negative PCR
Case N°8	Interferon pegilado + Ribavirina	Negative PCR
Case N°9	Interferon (6 months) Interfer + Ribavirin (16 months)	Treatment failure Negative PCR
Case N°10	Interferon (12 months)	Negative PCR

Table 3. Treatment of hepatitis B before kidney transplantation.

Case	Therapeutic molecules	Evolution
Case N°1	Lamivudine (12 months)	Negative PCR
Case N°2	None	Success
Case N°3	Lamivudine (12 months) Lamivudine + Adéfovir (24 months)	Resistance Negative PCR
Case N°4	Entécavir (12 months)	Negative PCR

Table 4. Comparisons between patients with and without hepatitis B and C reactivation.

	Group A No reactivation (n=12)	**Group B With reactivation (n=8)**	***P***
Proteinuria > 0.3 g/24 h	1 (5%)	12 (60%)	<0.001
Rejection	2 (10%)	6 (30%)	<0.001
Infection	6 (30%)	7 (35%)	0.279
Death	1 (5%)	2 (10%)	0.212
Return to dialysis	1 (5%)	4 (20%)	0.032
Initial immunosuppression			
ATG + Methylprednisolon	7 (35%)	3 (15%)	
Basiliximab + Methylprednisolone	1 (5%)	1 (5%)	0.07
Methylprednisolon	5 (25%)	2 (10%)	
METAVIR Score			
A0F0	1 (5%)	0 (0%)	
A0F1	2 (10%)	0 (0%)	
A1F0	1 (5%)	0 (0%)	
A1F1	3 (15%)	3 (15%)	0.555
A2F2	1 (5%)	0 (0%)	
A2F1	0 (0%)	1 (5%)	
A2F3	1 (5%)	0 (0%)	

Reactivation of Hepatitis B and C

Reactivation of hepatitis B and C viruses after kidney transplantation was significantly associated with the occurrence of proteinuria ($p < 0.001$), graft rejection ($p < 0.001$), and return to dialysis ($P= 0.032$) (Table 4).

Evolution and mortality

Three patients died, one from septic shock and two from reactivation of the hepatitis B virus. Two patients who were carriers of the hepatitis C virus died from a cardiovascular event. There was no significant difference in survival regarding the reactivation of viral hepatitis.

Discussion

In our series, the prevalence of hepatitis B and C viruses cured before kidney transplantation was 12.4%; this high percentage was explained by hemodialysis, which is the replacement technique for the majority of patients and is itself a risk factor for contamination. In France, the prevalence of HBV was 8%, and that of HCV was 5% in the kidney transplant population. This prevalence has significantly decreased owing to the use of erythropoietin, hemovigilance, and universal hygiene precautions [10-13]. Indeed, exposure to multiple blood transfusions and immunosuppression can also promote the transmission of HBV and HCV to dialysis patients and healthcare staff [14-16].

In several cohorts [17–19], the risk factors associated with hepatitis B and hepatitis C virus infections in hemodialysis patients included exposure to multiple blood transfusions, contamination of dialysis machines and equipment, and horizontal interpersonal transmission in dialysis units. Compared with the general population, patients with end-stage renal disease (ESRD) are at increased risk of contracting HBV and HCV infections due to a deficient immune response [20].

Ten patients in our series with hepatitis C virus were treated with interferon, pegylated interferon, and ribavirin during the dialysis period. Until 2014, before the availability of new direct-acting antiviral (DAA) drugs, the treatment of HCV in patients with ESRD was based on INF ± Rib [10, 21]. Interferon is poorly tolerated by patients with chronic renal failure, with relatively low efficacy [22]. International guidelines recommend treating HCV infection before kidney transplantation [10, 23, 24].

Patients carrying hepatitis B virus were treated with lamivudine, adefovir, and entecavir. Two patients developed resistance to lamivudine due to the long duration of treatment. The advent of effective nucleotide analogs (NAs) has provided the opportunity to minimize the consequences of HBV infection in patients with chronic renal failure [22]. The long-term resistance rates to lamivudine are very high, making lamivudine a first-line option that is not recommended for patients who may require prolonged treatment [25, 26].

Reactivation of hepatitis B virus (HBV) in kidney transplant recipients (KTRs) has been reported in 3% to 9% of patients who are positive for anti-HBc antibodies and negative for HBs antigen [27]. Several clinical studies have revealed that the risk of HBV reactivation may be notably greater in patients whose HBV infection is resolved but who are negative for anti-HBs antibodies and positive for anti-HBc antibodies and who do not receive antiviral prophylaxis [28, 29]. This aligns with the cases of HBV reactivation in our series.

Among the five patients with hepatitis C virus (HCV) reactivation in our series, only one patient received direct-acting antiviral (DAA) treatment with sofosbuvir and daclatasvir; this patient achieved a sustained virologic response (SVR) at 12 months posttreatment, with 100% success, and had normal renal function that was still intact eleven years after kidney transplantation. The other cases of HCV reactivation were untreated because of the contraindication of interferon-based treatment after kidney transplantation and the nonavailability of DAAs at the time. Among the four untreated patients, three lost their grafts and returned to dialysis, and one patient had chronic graft dysfunction with a GFR of 38 ml/min at the end of the study.

Savas et al. retrospectively reviewed data from 1,274 kidney transplant recipients over 21 years (1985--2006), forty-three of whom were positive for anti-HCV antibodies at the time of transplantation. HCV reactivation was observed in 19 patients (45.2%), with a mean time of 20.8 ± 5.7 months posttransplantation [30].

In our series, the reactivation of both hepatitis B virus (HBV) and hepatitis C virus (HCV) after kidney transplantation was significantly associated with the occurrence of proteinuria ($P<0.001$), rejection ($P <0.001$), and return to dialysis ($P= 0.032$).

According to a historical cohort study of 337 kidney transplant recipients, including 35 patients with active HCV during the kidney transplantation period, there were no significant differences in terms of the number of graft rejection episodes between the two groups. Although survival rates for both patients and grafts were lower in the HCV-positive group at 2 and 5 years, the differences between the HCV-positive and HCV-negative groups were not significant [31, 32].

According to a medical literature review [13], an association has been shown between hepatitis B virus infection and decreased patient and graft survival after kidney transplantation; the adjusted relative risk (aRR) for all-cause mortality and graft loss was 2.85 (95% CI: 2.36; 3.33, $P< 0.0001$) and 1.26 (95% CI: 1.02; 1.51, $P < 0.0001$), respectively.

This study had several limitations due to its retrospective nature, the small sample size, and the heterogeneity of the cases collected. However, larger studies are necessary to validate our findings.

Conclusion

Kidney transplantation is the best treatment for patients with end-stage chronic kidney disease who are carriers of hepatitis B virus (HBV) and hepatitis C virus (HCV).

Infections with HBV and HCV currently have a low prevalence among kidney transplant recipients because of the systematic screening of blood and organ donations. Adherence to universal hygiene

protocols, along with vaccination against hepatitis B, has helped reduce their prevalence in this population. Compared with seronegative patients, kidney transplant recipients who are carriers of HCV and HBV are more likely to develop proteinuria, and graft survival is lower.

Infection with HBV and HCV must be recognized as a significant risk factor for graft loss. It is necessary to initiate antiviral treatment for viremic patients before kidney transplantation to improve the likelihood of kidney graft survival.

Abbreviations

CKD: End-stage chronic kidney disease.
HD: hemodialysis.
PCR: polymerase chain reaction.
KT: Kidney transplant.
HCV: hepatitis C virus.
HBV: Hepatitis B virus.

Supplemental Information

The supplementary materials have not been included.

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Not declared.

Contributions from authors

Hajar Fitah: Conceptualization, Methodology, Research, Writing – Original Draft, Project Management, Supervision, Validation, Visualization, Writing – Proofreading and Editing.
Hajar El Allagui,: Conceptualization, Project Management, Supervision, Validation, Visualization, Writing – Revision and Editing.
Nabil Hmaidouch,: Conceptualization, methodology, research, Writing – Original draft.
Mariam El Galiou: Conceptualization, methodology, research, Writing – Original draft.
Naima Ouzeddoun: Conceptualization, Methodology, Research, Writing – Original Draft.
Loubna Benamar: Conceptualization, methodology, research, Writing – Original draft.
Tarik Bouattar: Conceptualization, methodology, research, Writing – Original draft.:
All the authors read and approved the final version of the manuscript.

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Availability of data or materials
Not applicable.

Declarations

Ethics Committee Approval and Consent to Participate

The study was approved by the ethics committee of the Faculty of Medicine and Pharmacy, Mohammed V University in Rabat, Morocco.

Consent to publication
It does not apply when specific images, X-rays or photographs of patients are not published.

Conflicts of interest
The authors declare that they have no conflicts of interest.

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