AIDS Research and Therapy

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Kidney dysfunction in adults living with HIV and HBV: a 10-year retrospective cohort study across seven Asia-Pacific countries

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Abstract

Background This study investigated kidney dysfunction among people with HIV (PWH), comparing those with and without hepatitis B virus (HBV) co-infection. We further identified predictors of kidney dysfunction in PWH with HBV.

Methods Adult PWH in the TREAT Asia Observational Database—Low Intensity TransfEr cohort, who were on antiretroviral therapy, with follow-up after 2010 were included. HBV co-infection was defined by positive hepatitis B surface antigen. Kidney dysfunction was determined as a single estimated glomerular filtration rate < 60mL/min/1.73m^2. Kaplan-Meier curves were used to evaluate cumulative incidence of kidney dysfunction, and we used Cox proportional hazards model to analyze factors associated with kidney dysfunction in PWH with HBV.

Results Among 23,415 participants (median age = 37 years; interquartile range [IQR]: 31–43), most were male (62.2%), from lower-middle income countries (67.1%), and reported heterosexual HIV transmission (79.3%). The median follow-up time was 5.41 years (IQR: 2.05–8.67). The majority were prescribed NRTI + NNRTI (83.6%), and 4.9% had HBV co-infection. Overall, 8.0% had kidney dysfunction, with a higher proportion among PWH with HBV than those without HBV (14.8% vs. 7.6%, $p < 0.001$). Most cases of kidney dysfunction were stage III (84.2%). Factors associated with kidney dysfunction in PWH with HBV included older age (≥ 50 years: Hazard ratio [HR] = 6.45, 95%CI: 2.31, 18.04) compared to 18–29 years, higher income country (upper-middle income: HR = 1.78, 95%CI: 1.16, 2.74) compared to lower-middle income, low platelet counts (< 150×10^9/L: HR = 2.82, 95%CI: 1.85, 4.31) compared to normal platelets, and ART regimens (NRTI + NNRTI: HR = 0.43, 95%CI: 0.27, 0.70; NRTI + PI: HR = 0.60, 95%CI: 0.36, 1.01) compared to NRTI + INSTI. Higher CD4 counts (200–349 cells/μL: HR = 0.53, 95%CI: 0.31, 0.93; 350–499 cells/μL: HR = 0.45, 95%CI: 0.26, 0.79; ≥500 cells/μL: HR = 0.33, 95%CI: 0.20, 0.56) compared to < 200 cells/μL were associated with lower risk of renal dysfunction. There was no significant difference in kidney dysfunction between those on TDF and TAF (HR = 0.55, 95%CI: 0.25, 1.23).

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Conclusions A high prevalence of kidney dysfunction was observed among PWH with HBV co-infection in the Asia-Pacific. Renal screening and monitoring should prioritize PWH with HBV with older age, low platelets and CD4 counts in low-resource settings.

Keywords Antiretroviral therapy, Renal insufficiency, HIV infection, Hepatitis B, Asia-Pacific, TAF, TDF

Background

In the Asia-Pacific, approximately 9.8% of people with HIV (PWH) have hepatitis B virus (HBV) co-infection [1], which is associated with higher morbidity and mortality than HIV alone [2]. Kidney dysfunction is an important comorbidity among PWH and may be influenced by both HIV/HBV co-infection and antiretroviral therapy, particularly Tenofovir Disoproxil Fumarate (TDF). TDF is used across diverse socioeconomic settings [3]; however, its use is known to be associated with reduced kidney dysfunction, as reflected by decreased estimated glomerular filtration rate (eGFR), and may potentially lead to renal failure [4, 5]. Studies have shown that kidney dysfunction associated with TDF use occurs in a variable proportion of PWH, with those on TDF-based regimens having a 1.7-fold higher risk of renal dysfunction compared to those on other regimens [6]. If TDF is discontinued, about 13% of PWH recovered full kidney function [7]. While the global trend favors Tenofovir Alafenamide Fumarate (TAF) for its renal benefits, TDF remains recommended in the World Health Organization (WHO) guidelines and continues to be widely used in the Asia-Pacific due to limited availability, cost and accessibility of TAF [8]. Many countries in the region use TDF-based combinations such as tenofovir/lamivudine/dolutegravir (TLD) or tenofovir/lamivudine/efavirenz (TLE) [9]. Given these concerns, it is crucial to assess the impact of TDF use in the region, particularly in relation to kidney dysfunction among PWH.

TDF is also commonly used as first-line therapy for HBV due to its high virological efficacy and low drug resistance rates [11, 12]. In PWH with HBV co-infection, treatment guidelines include HBV-active agents [10], typically tenofovir combined with lamivudine (3TC) or emtricitabine (FTC) [13]. TDF has been shown to achieve HBV suppression in PWH with HBV co-infection, ranging from 57.4% to 85.6% [12]. Because TDF is the preferred treatment for both HBV and HIV/HBV co-infection [10, 14], understanding the risk of developing kidney dysfunction among PWH with HBV co-infection is pivotal for optimizing treatment outcomes.

Numerous studies have assessed factors associated with kidney dysfunction among PWH [15–17], however many have involved small sample sizes or limited follow-up [7]. Importantly, there is a scarcity of national and regional data on the association between TDF usage and kidney dysfunction in Asian population [18, 19]. Therefore, this study aimed to examine the occurrence of renal dysfunction in PWH with or without HBV co-infection from 2010 to 2021 in the Asia-Pacific region. Risk factors associated with renal dysfunction were further assessed in the subgroup with HBV co-infection.

Methodology

Study population

We included PWH enrolled in the TREAT Asia HIV Observational Database-Low Intensity TransfEr (TAHOD-LITE) cohort. TAHOD-LITE included over 50,000 PWH who were at least 18 years old from 11 clinical sites in seven Asia-Pacific countries and territories: Cambodia, Hong Kong SAR, India, Indonesia, South Korea, Thailand, and Vietnam. PWH who had initiated ART and were in follow-up after 2010 were included, as routine HBV testing data became available from this date. Eligible PWH were required to have at least one serum creatinine measurements during follow-up. Those with a history of hepatitis C virus (HCV), pre-existing renal impairment, or diagnosed kidney dysfunction at baseline (either at ART initiation or on January 1, 2010 if ART was initiated before 2010) or prior were excluded, to ensure a consistent and reliable baseline assessment of kidney function. Since HCV is a known cause of kidney dysfunction [20], including such individuals would have complicated the assessment of HBV impact on kidney function.

Statistical analysis

Kidney dysfunction was defined as the first instance of an eGFR of <60mL/min/1.73m^2 [18, 19, 21–23]. Kidney dysfunction was classified into five stages based on eGFR: Stage 1 (≥ 90), Stage 2 (60–89), Stage 3 (30–59), Stage 4 (15–29), and Stage 5 (<15) [24]. eGFR was calculated based on serum creatinine, age, and sex using the 2009 CKD-EPI single equation, which was the standard method during the study period. In this equation, S denotes serum creatinine (mg/dL), κ is 0.7 for females and 0.9 for males, and α is –0.329 for females and –0.411 for males, as shown below:

$$141\ x\ \min\left(\frac{S_{creatinine}}{k},\ 1\right)^{\alpha} x\ \max\left(\frac{S_{creatinine}}{k},\ 1\right)^{-1.209} x\ 0.993^{age} x\ 1.018\ [if\ female]$$

Demographic covariates included in the analysis were age, sex, World Bank country income levels, and modes of HIV infection. Clinical and laboratory measures encompassed CD4 cell counts (< 200 cells/μL, 200–349 cells/μL, 350–499 cells/μL, ≥ 500 cells/μL, not reported), viral load levels (≤ 1000 copies/mL, > 1000 copies/mL, not reported), fasting glucose (mg/dL; elevated ≥ 126 mg/dL), platelets (x10^9/L), serum creatinine (mg/dL), HBV infection defined by positive surface antigen (yes vs. no), elevated HBV DNA (IU/mL; elevated ≥ 2,000 IU/mL), anti-HBV medication (TAF, TDF, and not on medication/other), and ARV class (not on treatment, NRTI + NNRTI, NRTI + PI, NRTI + INSTI, and other).

The Kaplan-Meier plot was used to illustrate kidney dysfunction by HBV co-infection status, and log-rank tests were used to evaluate the differences between the two groups. Factors associated with kidney dysfunction among PWH with HBV co-infection were analyzed using Cox proportional hazards model. Risk time was left truncated at ART initiation or January 1, 2010, which ever occurred last, and ended on date of first kidney dysfunction. Those who did not have kidney dysfunction or deceased were censored on the date of their last visit, transfer, death, or loss to follow-up. Missing data for covariates were handled using a last observation carried forward approach [25], where the most recent available value prior to baseline or follow-up was carried forward.

Since emtricitabine/lamivudine (FTC/3TC) is often added to TDF/TAF for PWH with HBV due to its activity against both HIV and HBV [13], a sensitivity analysis was conducted to evaluate the impact of this combination, in addition to the primary effect of TDF. Furthermore, several covariates with an extremely high proportion of missing data, such as platelet count, were excluded in sensitivity analyses (not shown), which showed consistent results with the main model.

All covariates with $p < 0.10$ from univariate analyses were included in the multivariable model, using stepwise backward selection method. In the multivariable model, only statistically significant variables ($p < 0.05$) were maintained.

SAS 9.4 (SAS Institute, Cary NC) and STATA version 18 (STATA Corp., College station, TX) were used to manage the data and perform the statistical analyses.

Results

Characteristics of adults living with HIV at baseline

We analyzed 23,415 adult PWH, with a median follow-up of 4.51 years (Interquartile range [IQR]: 2.05–8.67). Most participants were male (62.2%), with a median age of 37 years (IQR 31–43), and two-thirds were from lower-middle-income countries (67.1%). Heterosexual contact was the predominant mode of HIV acquisition (79.3%). At baseline, PWH had a median CD4 count of 201 cells/μL (IQR 67–331) and a median viral load of 77,348 copies/mL (IQR 14,811–305,072). Median platelets and glucose were 227 × 10^9/L and 86.5 mg/dL, respectively. HBV co-infection was observed in 4.9% of PWH. Most received NRTI + NNRTI regimens (83.6%), while fewer received NNRTI + PI (8.1%) or NRTI + INSTI (5.7%). TDF-based regimens were prescribed in 42%, and TAF in 1.1% (Table 1).

Kidney dysfunction status

During follow-up, approximately 8.0% of the total study population had kidney dysfunction, primarily stage III (84.2%), followed by stage IV (8.1%) and stage V (7.7%). The proportions of kidney dysfunction among PWH with and without HBV co-infection were 14.8% and 7.6%, respectively ($p < 0.001$; Supplemental Table 1). Given the higher proportion of kidney dysfunction among PWH with HBV co-infection, we assessed the time to kidney dysfunction by HBV co-infection status and identified factors associated with kidney dysfunction in PWH with HBV co-infection. This is also supported by a logistic regression model adjusting for demographics (age, sex, country income level, and HIV transmission route), which showed that PWH with HBV co-infection had 1.66 times higher odds of kidney dysfunction compared with PWH without HBV co-infection (aOR = 1.66, 95%CI: 1.39, 1.99) (Supplemental Table 2).

Kidney dysfunction by HBV infection status among PWH

Kaplan-Meier analysis showed that the cumulative incidence of kidney dysfunction at five years was higher among PWH with HBV co-infection (5.69%) than those without HBV (3.91%; Fig. 1). By 10 years, cumulative incidence was 16.68% versus 15.91%, respectively. The difference in cumulative incidence over the entire follow-up period was statistically significant, with a Log-rank test p-value of < 0.001.

Predictors of kidney dysfunction among PWH with HBV co-infection

The incidence rate of kidney dysfunction among PWH with HBV-co-infection was 2.32 per 100 person-years (Table 2). In adjusted analyses, older age, lower CD4 counts, low platelet counts, and residing in upper-middle-income countries were associated with increased risk of kidney dysfunction. Specifically, participants aged ≥ 50 years had greater risk compared to those aged 18–29 years (HR = 6.45, 95%CI: 2.31, 18.04). Participants from upper-middle-income countries had higher risks than those from lower-middle-income countries (HR = 1.78, 95%CI: 1.16, 2.74). Those with platelet counts < 150 × 10^9/L were at higher risk compared to participants with normal platelet counts (HR = 2.82, 95%CI: 1.85, 4.31). Higher CD4 counts were protective compared

Table 1 Demographic and clinical characteristics at baseline among PWH in the Asia-Pacific region, stratified by kidney dysfunction status

	Total	**Without kidney dysfunction**	**With kidney dysfunction***	***p*-value****
	***N*=23,415**	***n*=21,541**	***n*=1,874**	
Age (years)				
Median (IQR)	37.0 (31.0–43.0.0.0)	36.0 (30.0–42.0.0.0)	45.5 (39.0–53.0.0.0)	<0.001
18–29	4,702 (20.1)	4,610 (21.4)	92 (4.9)	<0.001
30–39	9,649 (41.2)	9,272 (43.0)	377 (20.1)	
40–49	6,286 (26.8)	5,564 (25.8)	722 (38.5)	
≥50	2,778 (11.9)	2,095 (9.7)	683 (36.4)	
Sex				<0.001
Male	14,560 (62.2)	13,323 (61.8)	1,237 (66.0)	
Female	8,855 (37.8)	8,218 (38.2)	637 (34.0)	
Country income level				<0.001
Lower middle	15,718 (67.1)	14,709 (68.3)	1,009 (53.8)	
Upper middle	5,840 (24.9)	5,179 (24.0)	661 (35.3)	
High	1,857 (7.9)	1,653 (7.7)	204 (10.9)	
Mode of HIV infection				<0.001
Heterosexual contact	18,568 (79.3)	17,165 (79.7)	1,403 (74.9)	
Male-to-male contact	1,840 (7.9)	1,711 (7.9)	129 (6.9)	
Injection drug use	378 (1.6)	350 (1.6)	28 (1.5)	
Other	2,629 (11.2)	2,315 (10.7)	314 (16.8)	
CD4 at baseline (cells/μL)				
Median (IQR)	201.0 (67.0–331.0.0.0)	205.0 (70.0–333.0.0.0)	130.0 (41.0–288.0.0.0)	<0.001
<200	5,122 (21.9)	4,710 (21.9)	412 (22.0)	<0.001
200–349	2,946 (12.6)	2,794 (13.0)	152 (8.1)	
350–499	1,233 (5.3)	1,176 (5.5)	57 (3.0)	
≥500	998 (4.3)	955 (4.4)	43 (2.3)	
Not reported	13,116 (56.0)	11,906 (55.3)	1,210 (64.6)	
Viral load at baseline (copies/mL)				
Median (IQR)	77,348.0 (14,811.0-305.0,072.0)	73730.5 (14,200.0–296,000.0)	134,686.0 (25,695.0-394.0,145.0)	0.003
≤1000	369 (1.6)	337 (1.6)	32 (1.7)	0.867
>1000	2,548 (10.9)	2,345 (10.9)	203 (10.8)	
Not reported	20,498 (87.5)	18,859 (87.5)	1,639 (87.5)	
Platelet count (x 10^9/L)				
Median (IQR)	227.0 (180.0–281.0.0.0)	227.5 (185.0–282.0.0.0)	207.0 (155.9–265.0.9.0)	0.210
<150 (Low)	64 (0.3)	55 (0.3)	9 (0.5)	0.334
150–450 (Normal)	407 (1.7)	375 (1.7)	32 (1.7)	
>450 (High)	8 (0.0)	8 (0.0)	0 (0.0)	
Not reported	22,936 (98.0)	21,103 (98.0)	1,833 (97.8)	
High fasting glucose (mg/dL)				
Median (IQR)	86.5 (81.0–96.9.0.9)	86.0 (81.0–95.0.0.0)	99.1 (84.0–122.5.0.5)	<0.001
No (<126)	535 (2.3)	508 (2.4)	27 (1.4)	<0.001
Yes (≥126)	36 (0.2)	28 (0.1)	8 (0.4)	
Not reported	22,844 (97.6)	21,005 (97.5)	1,839 (98.1)	
HBV infection defined by positive surface antigen				<0.001
No	22,178 (94.7)	20,487 (95.1)	1,691 990.2)	
Yes	1,237 (5.3)	1,054 (4.9)	183 (9.8)	
ART class				<0.001
Not on treatment	419 (1.8)	380 (1.8)	39 (2.1)	
NRTI+NNRTI	19,574 (83.6)	18,164 (84.3)	1,410 (75.2)	
NRTI+PI	1,902 (8.1)	1,608 (7.5)	294 (15.7)	
NRTI+INSTI	1,341 (5.7)	1,248 (5.8)	93 (5.0)	
Others	179 (0.8)	141 (0.7)	38 (2.0)	
TDF/TAF use				<0.001

Table 1 (continued)

	Total	Without kidney dysfunction	With kidney dysfunction*	*p*-value**
	***N*=23,415**	***n*=21,541**	***n*=1,874**	
TDF	9,845 (42.0)	9,124 (42.4)	721 (38.5)	
TAF	263 (1.1)	249 (1.2)	14 (0.7)	
Not on medication/Other	13,307 (56.8)	12,168 (56.5)	1,139 (60.8)	

*Among the 1,874 PWH with kidney dysfunction, 1,577 (84.2%) were in stage III, 152 (8.1%) in stage IV, and 145 (7.7%) in stage V.**Chi-squared test for differences in distributions and Wilcoxon rank-sum test for differences in medians.

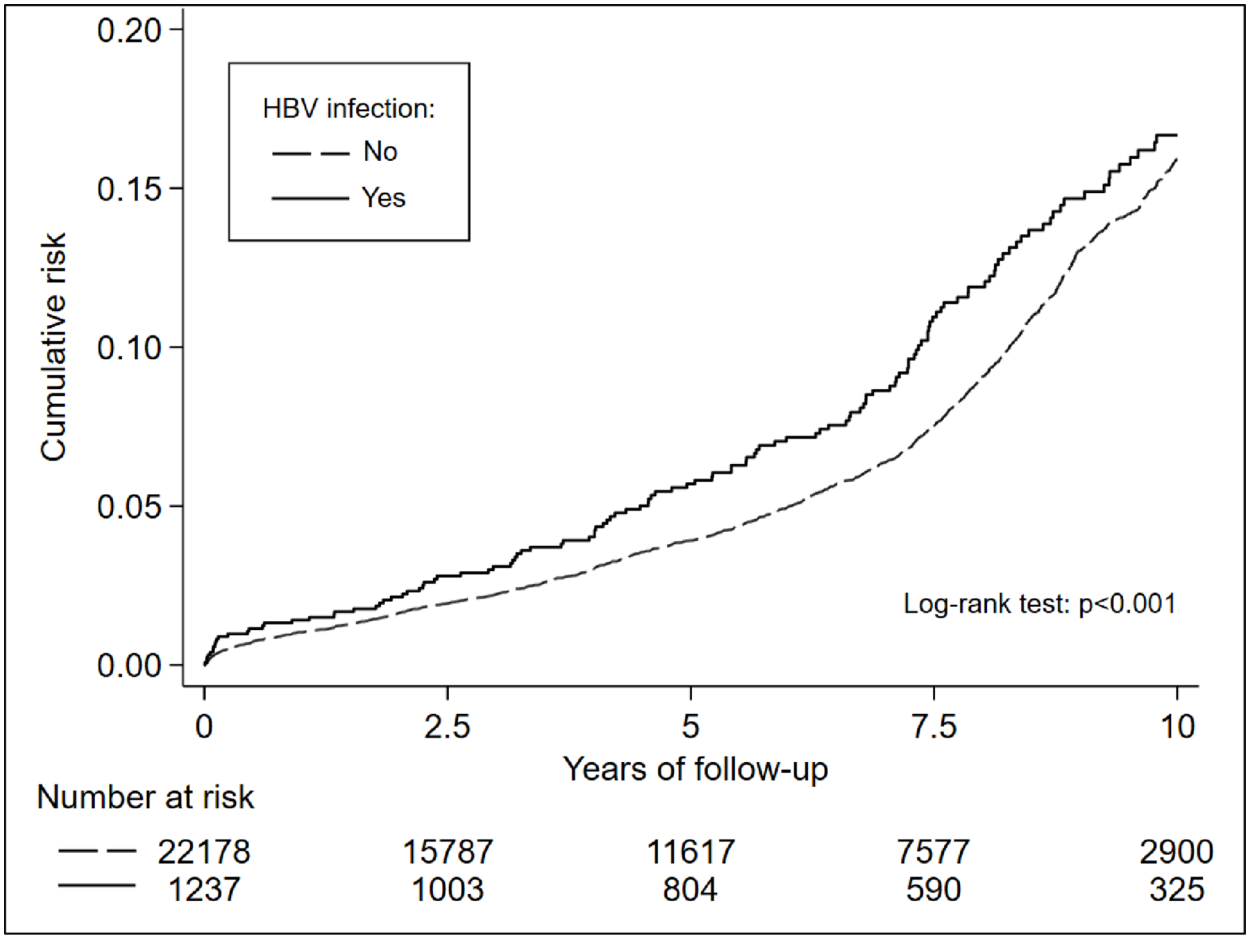

Fig. 1 Cumulative incidence of kidney dysfunction by HBV infection status among PWH in the Asia-Pacific region

to CD4 < 200 cells/μL (200–349 cells/μL: HR = 0.53, 95%CI: 0.31, 0.93; 350–499 cells/μL: HR = 0.45, 95%CI: 0.26, 0.79; ≥500 cells/μL: HR = 0.33, 95%CI: 0.20, 0.56). Use of NRTI + NNRTI (HR = 0.43, 95%CI: 0.27, 0.70) or NRTI + PI (HR = 0.60, 95%CI: 0.36, 1.01) was associated with lower risk compared to NRTI + INSTI. Results of our sensitivity analysis found that the use of TDF/TAF with FTC/3TC was associated with lower risk kidney dysfunction compared to those not on treatment (Table 3).

ART management after kidney dysfunction

Among 107 PWH who had kidney dysfunction and received TDF at the time of the event, 77 (72.0%) continued TDF, 3 (2.8%) switched to TAF, 24 (22.4%) switched to other ART, and 3 (2.8%) had ART stopped after developing kidney dysfunction (Table 2).

Discussion

Our findings revealed a higher proportion of kidney dysfunction among PWH with HBV co-infection compared to PWH alone. Factors associated with an increased risk of kidney dysfunction in this group included older age, higher World Bank country income level, and lower platelet count. In contrast, higher CD4 count and the use of NRTI + NNRTI or NRTI + PI were associated with a lower risk of kidney dysfunction compared to NRTI + INSTI.

Table 2 Factors associated with kidney dysfunction among PWH with HBV co-infection in the Asia-Pacific region

	No. of patients	Follow up (years)	No. of event	Incidence rate (/100pys)	Univariate model			Multivariable model		
					HR	95% CI	*p*-value	HR	95% CI	*p*-value
Total	1,237	7873.94	183	2.32						
*Age (years)**							**<0.001**			**<0.001**
18–29	~	773.34	4	0.52	1.00			1.00		
30–39	~	2320.11	14	0.60	1.04	0.34, 3.16	0.951	1.07	0.35, 3.29	0.901
40–49	~	3091.07	55	1.78	2.50	0.90, 7.00	0.080	2.39	0.85, 6.72	0.099
≥50	~	1689.42	110	6.51	7.15	2.58, 19.82	**<0.001**	6.45	2.31, 18.04	**<0.001**
Sex							0.057			
Male	847 (68.5)	5023.47	125	2.49	1.00					
Female	390 (31.5)	2850.47	58	2.03	0.74	0.54, 1.01	0.057			
Country income level							**0.022**			**0.010**
Lower middle	396 (32.0)	2294.18	33	1.44	1			1.00		
Upper middle	728 (58.9)	5048.73	136	2.69	1.66	1.13, 2.45	**0.010**	1.78	1.16, 2.74	**0.008**
High	113 (9.1)	531.03	14	2.64	2.01	1.07, 3.78	**0.030**	0.94	0.47, 1.87	0.850
Modes of HIV infection							0.526			
Heterosexual contact	899 (72.7)	5855.68	143	2.44	1.00					
Male-to-male contact	177 (14.3)	979.81	19	1.94	0.89	0.55, 1.44	0.643			
Injection drug use	39 (3.2)	284.42	5	1.76	0.58	0.23, 1.42	0.231			
Other	122 (9.9)	754.03	16	2.12	0.79	0.47, 1.32	0.361			
*CD4 (cells/μL)**							**0.004**			**<0.001**
<200	~	761.71	25	3.28	1.00			1.00		
200–349	~	1171.66	32	2.73	0.58	0.34, 1.01	0.053	0.53	0.31, 0.93	**0.026**
350–499	~	1461.29	39	2.67	0.49	0.29, 0.84	**0.010**	0.45	0.26, 0.79	**0.005**
≥500	~	3121.36	76	2.43	0.40	0.25, 0.66	**<0.001**	0.33	0.20, 0.56	**<0.001**
Not reported	~	1357.92	11	0.81	–	–	**–**	–	–	–
*Viral load (copies/mL)**							0.528			
≤1000	~	5367.00	149	2.78	1.00					
>1000	~	268.85	8	2.98	1.27	0.60, 2.69	0.528			
Not reported	~	2238.09	26	1.16	–	–	–			
*Glucose (mg/dL)**							**0.002**			
Not high (<126)	~	4520.85	124	2.74	1.00					
High (≥126)	~	387.06	26	6.72	1.96	1.28, 3.00	**0.002**			
Not reported	~	2966.03	33	1.11	–	–	**–**			
*Elevated HBV DNA (IU/mL)**							0.416			
No (<2000)	~	1072.77	45	4.19	1.00					
Yes (≥2000)	~	131.72	2	1.52	0.55	0.13, 2.30	0.416			
Not reported	~	6669.45	136	2.04	–	–	**–**			
*Platelets (x 10^9/L)**							**<0.001**			**<0.001**
Normal (150–450)	~	5331.39	131	2.46	1.00			1.00		
Low (<150)	~	371.23	28	7.54	3.17	2.11, 4.79	**<0.001**	2.82	1.85, 4.31	**<0.001**
High (>450)	~	95.04	3	3.16	1.68	0.53, 5.29	0.375	2.08	0.66, 6.61	0.213
Not reported	~	2076.27	21	1.01	–	–	**–**	–	–	–
*HBV medication**							**0.002**			0.203
TAF	~	73.14	8	10.94	1.00			1.00		
TDF**	~	5097.04	107	2.10	0.29	0.14, 0.60	**0.001**	0.55	0.25, 1.23	0.146
Not on medication/Other	~	2703.76	68	2.52	0.38	0.18, 0.80	**0.011**	0.68	0.31, 1.52	0.343
*ART class**							**<0.001**			**0.002**
NRTI+INSTI	~	420.64	29	6.89	1.00			1.00		
NRTI+NNRTI	~	5350.91	92	1.72	0.42	0.28, 0.65	**<0.001**	0.43	0.27, 0.70	**0.001**

Table 2 (continued)

	No. of patients	Follow up (years)	No. of event	Incidence rate (/100pys)	Univariate model			Multivariable model		
					HR	95% CI	*p*-value	HR	95% CI	*p*-value
NRTI+PI	~	1796.48	48	2.67	0.65	0.41, 1.05	0.077	0.60	0.36, 1.01	**0.053**
Not on treatment/Other	~	305.91	14	4.58	1.22	0.64, 2.34	0.544	0.93	0.47, 1.84	0.945

*Covariates are time-updated

**Among 107 participants on TDF at the time of event, 77 took TDF, 3 took TAF, 24 took other, and 3 not on medication after the event

Global p-values are test for heterogeneity excluding "not reported" values

P-values in bold represent significant covariates

In this study, 8% of PWH experienced kidney dysfunction, which is higher than the global average among PWH from 60 countries at 4.8% [26]. The relatively low prevalence of end-stage renal disease or eGFR stage V (0.62%; 145/23,425) among PWH in our study is similar to Japan (0.5%) [27], but lower than the USA (1.5%) [28], suggesting that different regions may reflect variations in genetic susceptibility, racial or ethnic composition, ART regimen, healthcare access, and screening practices. Furthermore, Kaplan-Meier analysis showed that PWH with HBV co-infection had a significantly higher cumulative incidence of kidney dysfunction compared to PWH alone, with the separation between the two survival curves occurring very close to time zero. This early divergence may reflect underlying differences not captured in our analysis. While HIV can cause HIV-related nephropathy, resulting in a decline in kidney function [29], HBV has been known to lead to glomerulonephritis [30], and these mechanisms, individually or combined, may contribute to the rapid separation of the two groups. Previous studies also demonstrated that HBV infection was associated with a higher risk of kidney dysfunction [31–33]. Particularly, HBV infection was linked to a 20% increased risk of chronic kidney dysfunction in the general population [32] and a 96% increased risk among PWH [31].

We found that high HBV viral load was not significantly associated with kidney dysfunction. HBV DNA testing was performed inconsistently in this cohort, and the large amount of missing data limits our ability to draw firm conclusions about the potential role of HBV replication in renal dysfunction. Prospective studies with more detailed clinical and treatment data, including cumulative TDF exposure, ART regimen variations, and timing of ART initiation are needed to better elucidate the underlying mechanisms. Older PWH with HBV had a higher hazard for kidney dysfunction, as supported by existing literature [21, 23, 34]. Using data from the same region, PWH aged > 50 who have ever received TDF had been shown to have 5.39 times greater risk of developing kidney dysfunction compared to those aged 30 or younger in the Asia-Pacific between 1996 and 2013 [23]. Similarly, compared to Australian PWH aged 30–39, individuals aged 40–49 had at least a 222% increased risk of kidney dysfunction [21]. Older PWH are at a significantly higher risk of developing kidney dysfunction compared to younger PWH, as aging naturally leads to a decline in kidney function and increases the likelihood of developing related health complications, such as hypertension, which are associated with kidney dysfunction [15].

We found that PWH from upper-middle income countries were more likely to have kidney dysfunction compared to those from lower-middle income countries. Insufficient healthcare infrastructure and limited access to care in lower-middle-income regions might contribute to the significant differences in the prevalence of chronic kidney disease [35, 36]. Notably, early diagnosis of kidney dysfunction is rare in resource-limited areas [36], with nine out of 10 individuals with kidney dysfunction unaware of their condition [35].

The association with lower platelet counts, observed in our study is consistent with previous findings [37, 38]. Platelets may accelerate kidney disease by mediating inflammatory processes and influencing the immune system [38]. In PWH with HBV co-infection, particularly older individuals, HBV-induced cirrhosis can significantly impair both liver and kidney function, leading to thrombocytopenia [39], which suggests a bidirectional relationship between platelet counts and kidney dysfunction. Furthermore, low CD4 cell counts was associated with higher risks of kidney dysfunction, likely due to susceptibility to infections, leading to increased inflammation, which can damage the kidneys [40]. Our findings were consistent with a prior investigation in Ethiopia, which found that PWH with low CD4 counts (< 200 cells/μL) were 2.5 times more likely to develop kidney dysfunction compared to those with CD4 ≥ 200 cells/μL [41].

This study also found that PWH with HBV receiving NRTI + NNRTI or NRTI + PI regimens were less likely to experience kidney dysfunction in comparison to those who received NRTI + INSTI. INSTIs, which commonly include dolutegravir and raltegravir, can cause mild elevations in serum creatinine, potentially leading to slight increases in kidney dysfunction [42]. Alternative methods to estimate GFR, such as cystatin C, were not available in this cohort, limiting our ability to distinguish

Table 3 Sensitivity analysis for factors associated with kidney dysfunction among PWH HBV co-infection in the Asia-Pacific region

	No. of patients	Follow up (years)	No. of event	Incidence rate (/100pys)	Univariate model			Multivariable model		
					HR	95% CI	p-value	HR	95% CI	p-value
Total	**1,237**	**7873.94**	**183**	**2.32**						
Age (years)[*]							**<0.001**			**<0.001**
18-29	~	773.34	4	0.52	1.00			1.00		
30-39	~	2320.11	14	0.60	1.04	0.34, 3.16	0.951	1.10	0.36, 3.36	0.872
40-49	~	3091.07	55	1.78	2.50	0.90, 7.00	0.080	2.58	0.92, 7.26	0.072
≥50	~	1689.42	110	6.51	7.15	2.58, 19.82	**<0.001**	6.94	2.48, 19.40	**<0.001**
Sex							0.057			
Male	847 (68.5)	5023.47	125	2.49	1.00					
Female	390 (31.5)	2850.47	58	2.03	0.74	0.54, 1.01	0.057			
Country income level							**0.022**			**0.033**
Lower middle	396 (32.0)	2294.18	33	1.44	1.00			1.00		
Upper middle	728 (58.9)	5048.73	136	2.69	1.66	1.13, 2.45	**0.010**	1.76	1.12, 2.75	**0.013**
High	113 (9.1)	531.03	14	2.64	2.01	1.07, 3.78	**0.030**	1.24	0.60, 2.53	0.564
Modes of HIV infection							0.526			
Heterosexual contact	899 (72.7)	5855.68	143	2.44	1.00					
Male-to-male contact	177 (14.3)	979.81	19	1.94	0.89	0.55, 1.44	0.643			
Injection drug use	39 (3.2)	284.42	5	1.76	0.58	0.23, 1.42	0.231			
Other	122 (9.9)	754.03	16	2.12	0.79	0.47, 1.32	0.361			
CD4 (cells/μL)[*]							**0.004**			**<0.001**
<200	~	761.71	25	3.28	1.00			1.00		
200-349	~	1171.66	32	2.73	0.58	0.34, 1.01	0.053	0.54	0.31, 0.94	0.030
350-499	~	1461.29	39	2.67	0.49	0.29, 0.84	**0.010**	0.43	0.25, 0.75	0.003
≥500	~	3121.36	76	2.43	0.40	0.25, 0.66	**<0.001**	0.32	0.19, 0.55	<0.001
Not reported	~	1357.92	11	0.81	–	–	**–**	–	–	–
Viral load (copies/mL)[*]							0.528			
≤1000	~	5367.00	149	2.78	1.00					
>1000	~	268.85	8	2.98	1.27	0.60, 2.69	0.528			
Not reported	~	2238.09	26	1.16	–	–	–			
Glucose (mg/dL)[*]							**0.002**			
Not high (<126)	~	4520.85	124	2.74	1.00					
High (≥126)	~	387.06	26	6.72	1.96	1.28, 3.00	**0.002**			
Not reported	~	2966.03	33	1.11	–	–	**–**			
Elevated HBV DNA (IU/mL)[*]							0.416			
No (<2000)	~	1072.77	45	4.19	1.00					
Yes (≥2000)	~	131.72	2	1.52	0.55	0.13, 2.30	0.416			
Not reported	~	6669.45	136	2.04	–	–	**–**			
Platelets (x 10^9/L)[*]							**<0.001**			**<0.001**
Normal (150-450)	~	5331.39	131	2.46	1.00			1.00		
Low (<150)	~	371.23	28	7.54	3.17	2.11, 4.79	**<0.001**	2.70	1.78, 4.11	**<0.001**
High (>450)	~	95.04	3	3.16	1.68	0.53, 5.29	0.375	2.13	0.67, 6.77	0.200
Not reported	~	2076.27	21	1.01	–	–	**–**	–	–	–
HBV medication[*]							**<0.001**			**0.006**
Not on medication		571.55	29	5.07	1.00			1.00		
TDF+FTC	~	1123.65	23	2.05	0.52	0.30, 0.91	0.022	0.51	0.29, 0.91	**0.022**
TDF+3TC	~	2548.65	58	2.28	0.49	0.31, 0.76	0.002	0.52	0.33, 0.81	**0.004**
TAF+FTC	~	67.12	7	10.43	1.55	0.67, 3.57	0.301	1.45	0.62, 3.58	0.617
TAF+3TC	~	6.03	1	16.59	4.23	0.57, 31.37	0.158	3.42	0.45, 25.93	0.450
Other	~	3556.94	65	1.83	0.47	0.30, 0.73	0.001	0.66	0.41, 1.06	0.406

[*]Covariates are time-updated. P-values in bold represent significant covariates.

true kidney impairment from INSTI-related creatinine changes. Our sensitivity analyses showed that TDF + FTC or TDF + 3TC were associated with a lower risk of kidney dysfunction, consistent with findings that the doravirine, 3TC, and TDF combination may improve estimated kidney function [43]. However, a systematic review of 26 clinical studies found TAF to be safer for the kidneys than TDF [44]. Additionally, TDF has been associated with a 63% increased risk of kidney dysfunction compared to other ARV medications [45]. We did not observe a significant difference in our analysis, likely due to limited number of participants on TAF who experienced kidney dysfunction events, which reduced statistical power. TDF/TAF use in the cohort was recorded separately when prescribed for HBV treatment; however, given their role in ART backbones, their use may also reflect co-treatment decisions driven by HBV status, even when not explicitly recorded as HBV therapy.

While we were able to assess kidney dysfunction among PWH with HBV co-infection using large regional cohort data in the Asia-Pacific, there were some limitations. Variations in creatinine measurement practices and available resources across study settings may affect the accuracy of detecting renal dysfunction, potentially leading to underreporting of events [21, 23]. eGFR was estimated using the CKD-EPI formula, previously used in studies evaluating kidney dysfunction [18, 19, 21–23], which is clinically reliable but not the most accurate; Cystatin C–based measures, though more precise, are rarely used in routine care. Assessment at a single time point may also miss transient or falsely elevated creatinine levels, potentially leading to misclassification of kidney dysfunction. Repeated measurements would better capture kidney dysfunction but were not consistently available in this cohort. Additionally, we did not collect data on comorbidities (e.g., hypertension, and cardiovascular disease) [21] or HIV-related factors (e.g., WHO clinical stages) [15] which are associated with an increased risk of renal dysfunction. Given the nature of the database, we were also unable to account for all potential confounders, including clinical decision-making processes, such as contraindications or comorbidities, that may have influenced ART or HBV treatment choices. As treatment allocation (e.g., TDF vs. TAF) was not randomized, indication bias cannot be excluded. We also acknowledge that there are additional ART-related factors that could impact the development of kidney dysfunction that were not assessed in this cohort. Specifically, cumulative exposure to different ART regimens and regimen switching are key factors that may influence renal outcomes among PWH with HBV co-infection. Further analyses are needed to evaluate these variables and their potential impact on kidney dysfunction in this population. We plan to investigate these factors in future research to gain a more comprehensive understanding of ART's role in kidney health. Finally, our subgroup analysis among PWH with HBV co-infection has 183 events. Future research with longer follow-up time is necessary to improve statistical power and accuracy of the findings. In addition, acquisition of HBV infection and other information such as HBV vaccination status was not collected in the cohort. This limits our ability to assess prevention efforts and timing of infection.

Conclusion

This study reveals a higher prevalence of kidney dysfunction among PWH with HBV compared to PWH alone. With kidney dysfunction rates in the Asia-Pacific region higher than global average, there is a need for regular renal screening and monitoring, particularly for PWH with HBV co-infection with older age, low platelets and CD4 counts in low-resource settings. Future research should address comorbidities and refine measurement practices (e.g., duration of TDF exposure and specific ARV medications) to enhance our understanding of renal health impacts within this population.

Abbreviations

3TC	Lamivudine
ARV	Antiretroviral
FTC	Emtricitabine
HBV	Hepatitis B virus
INSTI	Integrase Strand Transfer Inhibitor
NNRTI	Non-Nucleoside Reverse Transcriptase Inhibitor
NRTI	Nucleoside Reverse Transcriptase Inhibitor
PI	Protease Inhibitor
PWH	People with HIV
TAF	Tenofovir lafenamide
TDF	Tenofovir disoproxil fumarate
WHO	World Health Organization

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12981-025-00831-8.

Supplementary Material 1

Acknowledgements

The authors would like to thank Professor Joseph P. Dario of the Icahn School of Medicine at Mount Sinai, United States for his valuable comments on the manuscript and assistance with language editing. This work was presented at the International AIDS Conference 2025 in Kigali, Rwanda.

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TTV contributed to the conceptualization and design of the study. TTV led data management and analysis. TTV drafted the initial versions of this manuscript. All authors provided feedback on and revised the previous versions of the manuscript. Finally, all authors reviewed and approved the final manuscript.

Funding
The TREAT Asia HIV Observational Database Low-Intensity TransfEr study is an initiative of TREAT Asia, a program of amfAR, The Foundation for AIDS Research, with support from the U.S. National Institutes of Health's (NIH) National Institute of Allergy and Infectious Diseases, the Eunice Kennedy Shriver National Institute of Child Health and Human Development, the National Cancer Institute, the National Institute of Mental Health, the National Institute on Drug Abuse, the National Heart, Lung, and Blood Institute, the National Institute on Alcohol Abuse and Alcoholism, the National Institute of Diabetes and Digestive and Kidney Diseases, and the Fogarty International Center, as part of the International Epidemiology Databases to Evaluate AIDS (IeDEA; U01AI069907). The Kirby Institute is funded by the Australian Government Department of Health and Ageing, and is affiliated with the Faculty of Medicine, UNSW Sydney. This publication is the result of funding in whole or in part by the NIH. It is subject to the NIH Public Access Policy. Through acceptance of this federal funding, NIH has been given a right to make this manuscript publicly available in PubMed Central upon the Official Date of Publication, as defined by NIH. The content of this publication is solely the responsibility of the authors and does not necessarily represent the official views of any of the governments or institutions mentioned above.

Data availability
Data cannot be shared publicly because of confidentiality concerns and because it is considered as owned by contributing study sites. Anonymized data are available on reasonable request with the agreement of the study and site principal investigators (contact via study project manager: tor.peterson@treatasia.org), for researchers who meet the criteria for access to confidential data.

Declarations

Ethics approval and consent to participate
Ethics approval for the study was obtained from the ethics committees of each participating site, the data management and biostatistical center at the Kirby Institute [The University of New South Wales (UNSW) Human Ethics committee], and the coordinating center at TREAT Asia/amfAR. This study was conducted in accordance with the Declaration of Helsinki.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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Received: 1 September 2025 / Accepted: 22 November 2025
Published online: 12 December 2025

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