

## **Erratum**

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In the article "Similarities and Differences between COVID-19-Associated Nephropathy and HIV-Associated Nephropathy" [Kidney Dis. 2022;8:1–12. DOI: 10.1159/000 520235] by Chen et al., in Table 1, the first two entries in the column "Virus" have been interchanged. The corrected Table 1 is shown here.

Table 1. Comparison between COVID-19- and HIV-associated kidney diseases

	COVID-19-associated kidney disease	HIV-associated kidney disease
Virus	Coronaviruses, enveloped, positive single-stranded RNA viruses	Lentivirus, enveloped, positive single-stranded RNA viruses
Incidences	2–46% COVID-19 patients	6–48.5% HIV-infected patients
Clinical presentations	AKI, proteinuria, hematuria	AKI, proteinuria, enlarged kidney
Pathology	ATI, cFSGS, MCD, MN, lupus nephritis, and TMA	cFSGS, HIVCK, ATN, tubulointerstitial disease
Infection of kidney cells	Unknown, likely via ACE2	Yes, via cell-cell transmission (lymphocytes-kidney cells)
APOL1 risk alleles	Yes for cFSGS	Yes for cFSGS
Systemic effects	Yes with cytokine storm	Yes with sepsis
Drug toxicity	Yes, but not well-determined	Yes with tenofovir and others
CKD	AKI to CKD or AKI on CKD	Affect CKD progression
Treatment	No effective antiviral drugs, transient infection with development	Effective viral suppressive drugs, persistent and not cur-
	of neutralizing antibodies, vaccine available	able, kidney is viral reservoir, no vaccine

AKI, acute kidney injury; CKD, chronic kidney disease; APOL1, apolipoprotein L1; cFSGS, collapsing focal segmental glomerulosclerosis; HIVCK, HIV-associated immune complex kidney disease; ATN, acute tubular necrosis; MN, membranous nephropathy; TMA, thrombotic microangiopathy; MCD, minimal change disease; ATI, acute tubular injury; HIV, human immunodeficiency virus.



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