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ORIGINAL ARTICLE

Neutralising Antibodies Response Against SARS-CoV-2 Among People Living With HIV in Dar- es-Salaam Tanzania

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ABSTRACT

Background: Coronavirus Disease 2019 has become endemic and is no longer a global health emergency. However, it remains significant for people living with HIV, whose immune dysregulation may impact antibody responses. Hence this study assessed neutralizing antibody capacity against SARS-CoV-2 variants among HIV-infected adults in Tanzania to inform sero-surveillance and pandemic readiness.

Methods: Seventy HIV-positive adults (≥18 years) were enrolled (53 vaccinated, 17 unvaccinated). These were compared with 89 individuals who were HIV negative (71 vaccinated and 18 unvaccinated). Neutralizing antibodies were quantified using a surrogate virus neutralization assay, with ≥27.7% inhibition defined as protective. Factors associated with neutralization were analyzed using modified Poisson regression (STATA v15), and group differences assessed using Kruskal-Wallis tests (Prism 9), with significance at $P<.05$.

Results: Our study observed a progressive decline in the proportion of participants with protective neutralizing antibodies across the SARS-CoV-2 variants, that is; 93% (65/70) for the wild-type (WT), 77% (54/70) for Delta, and 54% (38/70) for Omicron whether vaccinated or unvaccinated. Additionally, no significant difference in neutralising antibody inhibition capacity was found between participants with CD4 counts above 500 and those below, for any of the three variants, $P>.9999$. Also, the median percentage of neutralizing capacity among the relatively healthy HIV infected individuals (>500 CD4 count) was lower (28%) compared those who were HIV negatives (98%), within the vaccinated group ($P<.0001$) and 29% vs 80% for unvaccinated sub-groups ($P<.005$).

Conclusion: Neutralising antibodies declined significantly across newer SARS-CoV-2 variants among HIV-infected individuals. Moreover, neutralizing capacity was significantly lower among immune competent HIV-positive participants compared to their HIV negative counterparts. We recommend ongoing sero-surveillance to enable timely action.

BACKGROUND

During the early days of the COVID-19 pandemic, it was widely anticipated that individuals living with HIV would be among the most severely affected by SARS-CoV-2 due to their underlying immune dysfunction.[1,2] However, current evidence suggests that people with HIV on effective antiretroviral therapy (ART) can develop and maintain immune responses comparable to those of HIV-negative individuals.[3,4] Although sub-populations with persistently low CD4+ T cell counts and high levels of viremia remain at elevated risk, as they exhibit reduced SARS-CoV-2 IgG seroconversion and diminished antibody longevity.[4] These heterogeneities underscore the need for ongoing population-specific risk assessments, especially for HIV infected individuals particularly as new, highly transmissible pathogens emerge.

The HIV infection landscape in Tanzania have shown a persistence of various challenges overtime, those relevant to SARS-CoV-2 immune response, include one-third of the infected subpopulation are yet to achieve viral suppression,[5] and one third have non communicable disease such hypertension[6,7] and diabetes[8] among others, placing them at a significant risk for severe diseases. Moreover, a significant portion of the population are yet to attain CD4 T cell count above 500.[5] There is evidence for a more severe course of COVID-19 disease in people with cellular immune deficiency. According to Nault et al 2022, individuals with advanced disease (CD4 cell count >200cells/ml) have limited capacity in eliciting neutralisation antibodies after both vaccine or infection.[9,10] Hence although COVID-19

is no longer causing widespread morbidity and mortality,[11] The SARS-CoV-2 viral endemicity places the HIV infected individuals with suboptimal immune response at risk and disproportionally worse outcomes.

Considering the unique Tanzanian response during the COVID-19 pandemic, where lockdown strategy was not implemented,[12] potentially this led to increased populational viral transmissibility and viral mutations. However, this approach did not influence the magnitude and robustness of the antibody responses for the general population and not much was reported from the HIV infected population in Tanzania.[13,14] Therefore, because the virus evolutionary path is not predictable, and it is not clear whether the accumulating mutations and waning immune responses will give rise to a variant of concern, hence data on immune responses against experienced SARS-CoV-2 variants among HIV infected people is essential to guide strategy in the face of another pandemic. This study will report on neutralizing antibodies against experienced SARS-CoV-2 variants such as the original Wuhan variant, the Delta and the Omicron variants.

MATERIALS AND METHODS

Study Site

Blood samples were obtained from participants recruited from the Muhimbili National Hospital, Mwananyamala and Temeke Regional Referral Hospitals and the Aga Khan Hospital in Dar-es-salaam, Tanzania. Data and sample collection were performed at the Muhimbili University of health and Allied Sciences (MUHAS) in Dar-es-salaam, Tanzania.

Study Design, Study Population and Sampling

A cross-sectional study was conducted among HIV-infected adults aged ≥18 years. Participants were recruited from the same setting and during the same period as the HIV-negative comparison group. All participants reported no history of symptomatic Coronavirus Disease 2019 (COVID-19) based on a self-reported symptom questionnaire. Vaccinated participants had received Ad26.COV2-S (Janssen/Johnson & Johnson), BNT162b2 (BioNTech/Pfizer), or BBIBP-CorV (Sinopharm), and samples were collected between one to four months following vaccination or SARS-CoV-2 infection. Blood samples were collected in ethylenediaminetetraacetic acid (EDTA) tubes, and plasma was separated by centrifugation and stored at −80°C within three hours of collection.

The HIV-negative comparison group (n = 89) was previously described by Nkinda et al.,[13] This cohort comprised 71 vaccinated individuals who had received either Ad26.COV2-S or BNT162b2 vaccines and reported no symptomatic COVID-19, and 18 unvaccinated individuals with prior SARS-CoV-2 infection. HIV-positive and HIV-negative participants were recruited at the same time and were matched based on the duration since vaccination or SARS-CoV-2 infection.

The parent cohort included 220 SARS-CoV-2 infected and/or vaccinated participants, of whom 97 were people living with HIV (PLWH). The original study design involved the use of an HIV-based lentiviral pseudovirus neutralization assay. However, during assay validation, plasma samples from HIV infected participants demonstrated artificially elevated neutralization titres due to interference from antiretroviral drugs, which reduced pseudovirus infectivity and confounded the measurement of true SARS-CoV-2 neutralizing activity. Hence, an alternative assay was validated and adopted for use. Following assay validation,[15] and assay-related eligibility assessment the assay was used to run samples for HIV infected (n=70) and the selected HIV uninfected.

Measurement of Neutralization Antibodies Using the Surrogate Neutralization Assay

The surrogate virus neutralization assay was performed as previously described.15 Briefly plasma samples were heat inactivated, at 56ºC for 45 minutes, prior to further processing. The surrogate virus neutralization test (sVNT) (Gen-Script cPass™, USA, L00847) was performed as per manufacturer's instructions against the wildtype-(D614G) Sars CoV-2 virus, the Delta variant and the BA.1 Omicron variant.16 Briefly, samples, positive and negative controls were diluted 1:10 with sample dilution buffer. The dilutions were mixed with horseradish peroxidase conjugated recombinant SARS-CoV-2 RBD solution and incubated for 30 min at 37 °C. The mixtures were subsequently incubated for 15 min at 37 °C in a capture plate that was pre-coated with hACE2 protein. After a washing step, tetramethylbenzidine (TMB) solution was added and the plate was incubated in the dark at room temperature for 15 min. Stop solution was added to quench the reaction and the absorbance was immediately read at 450 nm on an ELISA microplate reader. The percentage inhibition was calculated as (1− OD value of sample OD value of Negative control) x 100%. Participants with inhibition ≥27.7% were regarded as neutralizers while those with <27.7% were regarded as non-neutralizers as per the established populational cut-off value.15

Statistical Analysis

Modified Poisson logistic regression was used to calculate factors associated with neutralizing antibodies because our outcome was common (>10%). This was done on STATA version 15. While Kruskal Wallis was used to make comparisons between the different exposure groups using Prism 9 (GraphPad Software). P value <5% was considered statistically significant.

Ethical Consideration

Ethical approval to conduct this study was obtained from the MUHAS Institutional Review Board (IRB) (Ref. No.DA.282/289/01.C/) and the National Health Ethics Committee (NatHREC) of the Tanzania National Institute for Medical Research (NIMR/HQ/R.8a/Vol.IX/4051).

Patient Consent Statement

A written informed consent was obtained from each participant before enrollment into the study. Data was stored in an encrypted file and entries were anonymized to ensure confidentiality.

Patient and Public Involvement

Patients or the public were not involved in the design, or conduct, or reporting, or dissemination plans of our research. Because of the complexity of the subject matter.

RESULTS

Baseline Characteristics of the Study Participants

In Table 1, we described a total of 70 participants who were enrolled. These participants were divided into two groups, as per their exposure, that is vaccinated (n=53) and unvaccinated (n=17). For the vaccinated group, more than half were below 50 years [57% (30/53)], majority were females [62.3% (33/53)], most [62% (33/53)] had been on ART for more than 10 years. The median viral load was low -11, close to undetectable levels, however majority [56% (29/53)] had CD4 T cell count below 500 cell/ml. Moreover most 74% (39/53)) were vaccinated with the Jansen vaccine, followed by Pfizer 10% (19/53) then Sinopharm vaccine 8% (4/53. For the unvaccinated group, 71% (12/17) were below 50 years, majority were females 53% (9/17), and more than half (65% 11/17) had been on ART for less than 10 years. The median viral load 11 (11,20) was almost undetectable, however, more than half 59% (10/17) had not attained CD4 count above 500 cells/ml. All participants reported not to have experienced signs or symptoms of COVID-19 disease.

Neutralizing Antibodies Among HIV Infected Participants

In Figure 1A, irrespective of the vaccine exposure or not, majority [(93%), 65/70] of the participants had neutralising antibodies against the Wuhan SARS-CoV-2 variant, although participants with sufficient protective neutralising antibodies reduced with each consequtive variant, ie; [(77%), 54/70] had neutralising antibodies against Delta, and only half of the participants [(54%), 38/70] had protective antibodies against the Omicron variant. The pattern was the same whether one was vaccinated or not, (P>.9999). The trend was the same for the actual antibody inhibition capacity among the vaccinated and unvaccinated sub-groups in Figure 1B, although the decline was significantly steeper for vaccinated participants, from the WT to Delta to the Omicron variant ie; P value=.0018, P value=<.0001, respectively. The unvaccinated individuals on Figure 1B displayed a similar pattern, but the inhibition capacity reduction was not statistically significant, P value=.216, P value=>.9999, P value=.6626.

Antibody Inhibition Capacity Among HIV Infected and HIV Negative Participants

In Figure 2A, we wanted to understand whether the viral inhibition capacity was influenced by the level of CD4 T cell count, considering half of our participants had <500 cell/ml CD4 T cells. Participants with higher CD4 count had comparatively higher median inhibition capacity across the three variants but this difference did not reach statistical significance, P value=>.9999. On Figure 2B we compared, if there was a difference in the inhibition capacity against the Omicron variant when groups of vaccinated and unvaccinated HIV negatives and HIV positives who had viral suppression, optimal CD4 count and similar exposure window of SARS-CoV-2 infection/vaccine, to those who were not infected were compared. We hereby report that the HIV infected individuals had significantly lower median inhibition capacity (28%), against the SARS-CoV-2 Omicron variant compared to their HIV negative counterparts (98%), after vaccination P value=<.0001 while for unvaccinated the antibody inhibition capacity was 29% vs 80% among HIV infected and uninfected respectfully P value=.0029.

Factors Associated With Having Neutralizing Antibodies

We further conducted Poisson regression analysis to factors associated to having neutralizing antibodies against the Omicron variant, since it is the variant that is closely related to the current circulating strain. Unfortunately, none of the factors were significant on the univariable analysis, hence we did not proceed to the multivariable analysis on Table 2.

TABLE 1: Baseline Characteristics of the Study Participants

Variable	n	Vaccinated n (%)	Unvaccinated n (%)
Age (years)			
18 – 50	42	30 (56.6)	12 (70.6)
>50	28	23 (43.4)	5 (29.4)
Sex			
Male	28	20 (37.7)	8(47.1)
Female	42	33 (62.3)	9(52.9)
Smoking			
None	60	45 (84.9)	15 (88.2)
Current smoker	3	2 (3.8)	1 (5.9)
Past smoker	7	6 (11.3)	1 (5.9)
Alcohol consumption			
None	33	25 (47.2)	8 (47.1)
Current drinker	14	8 (15.1)	6 (35.3)
Past drunker	23	20 (37.7)	3 (17.6)
Duration on ART			
≤10 years	26	20 (37.7)	6 (35.3)
>10 years	44	33 (62.3)	11 (64.7)

Continue

TABLE 1: Continued

Variable	n	Vaccinated n (%)	Unvaccinated n (%)
ART Treatment change_ 6 months			
Yes	22	20 (37.7)	2 (11.8)
No	48	33 (62.3)	15 (88.2)
Viral Load [median (IQR)]	70	11 (11,20)	11 (11.20)
CD4 T cell count [median (IQR)]	70	476 (336,731)	482 (352, 588)
CD4 T cell count level			
≤500	39	29 (54.7)	10 (58.8)
>500	31	24 (45.2)	7 (41.2)
Vaccine type			
Ad26.COV2-S vaccine	39	39 (73.6)	-
BNT162b2 mRNA vaccine	10	10 (18.8)	-
Sinopharm vaccine	4	4 (7.6)	-
Post vaccine duration			
0-3 months	13	13 (24.5)	-
>3 months	40	40 (75.5)	-
Chronic Disease			
Yes	40	30 (56.6)	10 (58.8)
No	30	23 (43.4)	7 (41.2)

FIGURE 1: Neutralizing antibody response against the wild type, Delta and Omicron SARS-CoV-2 variants (n=70)

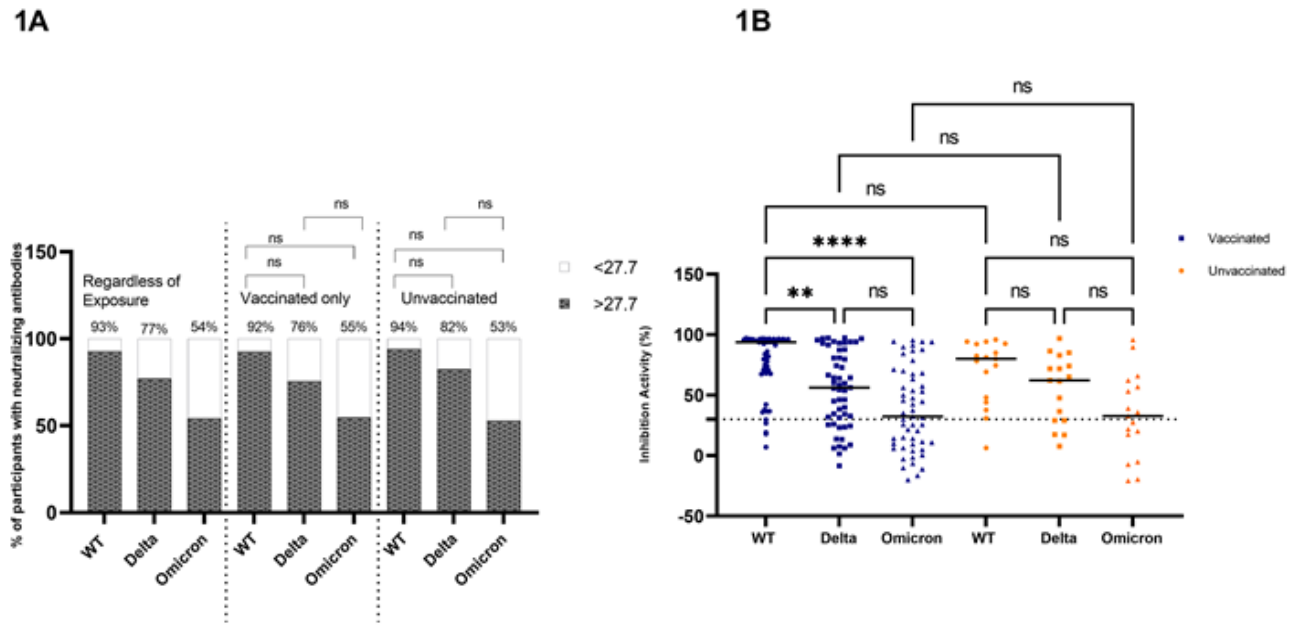


Fig 1A- Proportions of HIV infected participants with neutralising antibody titre against the respective variant; regardless of exposure, for vaccinated only participans and unvaccinated group. Fig 1B – Comparisons of antibody inhibition capacity against SARS-CoV-2 among vaccinated (blue symbols) and unvaccinated participats (orange symbols). The Horizontal dotted line indicate the cut-off threshold for protective antibody titres (27.7%). Comparisons were made using Kruskal Wallis test, p-values are displayed as ns = $p>.05$, **: $p<.01$, ****: $p<.00001$

FIGURE 2: Antibody inhibition response against SARS-CoV-2 among HIV negative (n= 89) and HIV positive individuals (n=70)

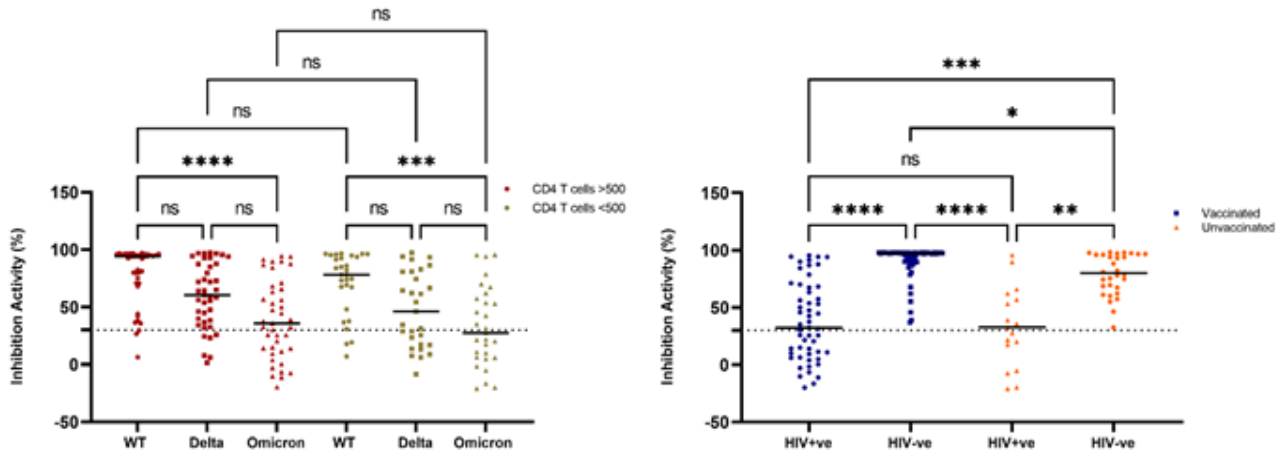


Figure2A shows comparison of antibody inhbition capacity among HIV infected with CD4 count above (red symbols) and below (green symbols) 500 cell/ml. Figure2B shows a comparison of inhibition capacity between vaccinated HIV positive and HIV negative participats (Blue symbols, and HIV positive and HIV negative unvaccinated participants (orange symbols). The Horizontal dotted line indicate the cut-off threshold for protective antibody titres (27.7%). Comparisons were made using Kruskal Wallis test, p-values are displayed as ns = p>.05, *: p<.05, **: p<.01, ***: p<.001, ****: p<.00001.

TABLE 2: Factor Associated With Neutralizing Antibodies

Variable	n	Negative	Positive	*P value*	Univariate analysis cPR	95%CI	*P value*
Age (years)							
18 – 50	42	28 (66.7)	14 (33.3)	.611	Ref		
>50	28	17 (60.7	11 (39.3)		0.97	0.62 – 1.52	.923
Sex							
Male	28	19 (67.9)	9 (32.1)	.611	Ref		
Female	42	26 (61.9)	16 (38.1)		0.74	0.45 – 1.65	.665
Smoking							
None	60	39 (65.0)	21 (35.0)	.243	Ref		
Current smoker	3	3 (100)	0 (00)		1.09	0.45 – 2.61	.842
Past smoker	7	3 (42.9)	4 (57.1)		0.71	0.29 – 1.58	.374
Alcohol consumption					1.04		
None	33	20 (60.6)	13 (39.4)	.789	Ref		
Current drinker	14	9 (64.3)	5 (35.7)		1.02	0.77 – 1.41	.770
Past drunker	23	16 (69.6)	7 (30.4)		1.23	0.79 – 1.92	.898
Duration on ART							
≤10 years	26	14 (53.8)	12 (46.2)	.161	Ref		
>10 years	44	31 (70.5)	13 (29.5)		0.90	0.58 – 1.40	.658
ART Treatment change 6 months							
Yes	22	33 (68.8)	15 (31.3)	.250	1.27	0.26 – 0.83	.268
No	48	12 (54.5)	10 (45.5)		Ref		
Viral Load							
Detectable	47	32 (68.1)	15 (31.9)	.343	1.33	0.87 – 2.02	.181
Undetectable	23	13 (56.5)	10 (43.5)		Ref		
Chronic disease							

Continue

TABLE 2: Continued

Variable	n	Negative	Positive	*P value*	Univariate analysis cPR	95%CI	*P value*
Chronic disease							
no	30	21 (70.0)	9 (30,0)	.388	Ref		
yes	40	24 (60.0)	16 (40.0		1.28	0.81 – 2.04	.286
CD4 T cell count							
≤500	39	23 (59.0)	16 (41.0)	.298	Ref		
>500	31	22 (71.0)	9 (29.0)		0.82	0.52 – 1.28	.390
Vaccination Period							
1-3 months	17	11 (64.7)	6 (35.3)	.967	Ref		
>3 months	53	34 (64.2)	19 (35.8)		0.85	0.50 – 1.44	.554
Vaccine type							
Ad26.COV2-S vaccine	39	25 (64.1)	14 (35.9)	.792	0.90	0.57 – 1.43	.348
BNT162b2 mRNA vaccine	10	7 (70.0)	3 (30.0		0.74	0.35 – 1.57	.447
Sinopharm vaccine	4	2 (50.0)	2 (50.0		Ref		

DISCUSSION

Our study observed a progressive decline in the proportion of participants with protective neutralizing antibodies across the SARS-CoV-2 variants, that is; 93% for the wild-type (WT), 77% for Delta, and 54% for Omicron regardless of prior exposure. Additionally, no significant difference in neutralizing antibody inhibition capacity was found between participants with CD4 counts above 500 and those below, for any of the three variants. However, a statistically significant lower neutralizing capacity against the Omicron variant was noted among HIV infected individuals with optimal CD4 T cell count when compared to the HIV negative participants.

In the current study, we observed a progressive decline in SARS-CoV-2 neutralization capacity across the consecutive variants, consistent with our previous findings among HIV-negative participants[13,14] who showed a decreasing antibody inhibition capacity as the viral mutations accumulated [13]. While the pattern seems to be the same regardless of the HIV status, in the current study, the decline was significantly steeper among individuals with HIV infection. According to Hoft et al 2024, people with HIV, particularly those with CD4 counts below 200 cells/ml, may have impaired seroconversion and consequently lower antibody titers,[4,6]although we also have studies which noted impaired antibody response at a CD4 count threshold of >200 cells/ml. In our study, there was a significantly lower neutralization capacity against the Omicron variant among HIV infected who had viral suppression, optimal CD4 count and similar exposure window of SARS-CoV-2 infection/vaccine, to those who were not infected. This suggests that we cannot completely equate the HIV infected individuals on effective ART with the HIV negative individuals, in terms of immune response, because of the underlying immune disfunction. Moreover, since approximately 30% of people living with HIV may never experience complete immune recovery despite sustained virological suppression,[4] ongoing sero-surveillance and vaccination emphasis is a critical public health strategy, because of their heightened risk of severe infection or re-infection as the evolutionary trajectory of SARS-CoV-2 remains uncertain. This need is urgent in Tanzania, where no published data currently exist on neutralizing antibody levels among individuals living with HIV and there are still high levels of vaccine hesitancy.

In the current study there was no difference in the neutralizing antibodies, between vaccinated and unvaccinated HIV infected individuals. Similar findings were reported by Batchi-Bouyou et al 2024[17] Of note vaccinated participants in the current study (76%, 53/70) received the primary series vaccines, as this is still the only available option in Tanzania to-date (2025). Regardless of minor difference stemming from the types of vaccine in boosting immune response,[1] it is generally agreeable that the primary series vaccines fall short in boosting immune response with respect to the current mutated variant.[13,14] The HIV participants could have benefited from the current WHO recommended variant specific vaccine as reported elsewhere.[18] Furthermore, we could not determine whether vaccine-induced immune responses reflected vaccination alone or were boosted by prior asymptomatic SARS-CoV-2 infection. Identification of previous infection using anti-nucleocapsid serology was limited by the inclusion of recipients of the whole-virus BBIBP-CorV vaccine, whose post vaccine response is similar to infection antibody response. Hence, the observed responses likely represent a pattern of vaccinated and unvaccinated individuals in a real-world setting.

Our study is not without limitations, first is the relatively small sample size of people living with HIV (PLWH), which may have reduced the statistical power to detect differences in subgroup analyses, including comparisons by CD4 count strata and vaccine type. Also, non-significant findings should be interpreted with caution, as they may reflect insufficient power rather than the absence of true associations. Future studies with larger sample sizes are warranted to confirm these findings. Secondly, although HIV-positive and HIV-negative participants were recruited at the same time, they were matched for duration of exposure since infection/vaccination only, hence there could be some confounding stemming from differences in

age/ sex, although for both groups, majority were below 50 years. Regardless our findings discuss an important phenomenal on whether HIV infected individuals with optimal CD4 count can elicit protective antibodies post vaccination similar to the HIV negative individuals. Lastly, we did not assess differences stemming from the different types of vaccines because majority received the Ad26.COV2-S (J&J), ie. 76% HIV infected and 80-100% for HIV negatives.

In conclusion, our study showed a progressive decline of neutralizing antibodies against the Delta and Omicron SARS-CoV-2 among both vaccinated and unvaccinated individuals. Moreover, there was no intragroup difference in mounting antibody protective titers when participants with CD 4 count above and below 500 were compared. Importantly, we observed a statistically significant lower neutralizing capacity against the Omicron variant among HIV infected individuals with optimal CD4 T cell count when compared to the HIV negative participants. For this population, we recommend continued sero-surveillance, in order to remain informed on the extent to which they require additional vaccinations. Moreover, future studies should evaluate variant-specific vaccines, their immunogenicity and effectiveness among HIV infected individuals in African settings, given current WHO recommendations and the continued evolution of SARS-CoV-2 variants.

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