Received: 12 September 2023 | Accepted: 2 January 2024

DOI: 10.1111/hiv.13611

SHORT COMMUNICATION

Clinical epidemiology of COVID-19 in people of black ethnicity living with HIV in the UK

Zoe Ottaway[1,2] | **Lucy Campbell**[1,2] | **Laura R. Cechin**[1] | **Nisha Patel**[1] | **Julie Fox**[2,3] | **Fiona Burns**[4,5] | **Lisa Hamzah**[6] | **Stephen Kegg**[7] | **Melanie Rosenvinge**[7] | **Sarah Schoeman**[8] | **David Price**[9] | **Rachael Jones**[10] | **Amanda Clarke**[11] | **Irfaan Mann**[5,12] | **Andrew Ustianowski**[13] | **Denis Onyango**[14] | **Shema Tariq**[5,12] | **Robert F. Miller**[5,12] | **Frank A. Post**[1,2] | **on behalf of the COVID-AFRICA study group**

[1]King's College Hospital NHS Foundation Trust, London, UK

[2]King's College London, London, UK

[3]Guys and St Thomas's NHS Foundation Trust, London, UK

[4]Royal Free London NHS Foundation Trust, London, UK

[5]Institute for Global Health, University College London, London, UK

[6]St George's University Hospital NHS Foundation Trust, London, UK

[7]Lewisham and Greenwich NHS Trust, London, UK

[8]Leeds Teaching Hospitals NHS Trust, Leeds, UK

[9]Newcastle Hospitals NHS Foundation Trust, Newcastle, UK

[10]Chelsea and Westminster NHS Foundation Trust, London, UK

[11]University Hospitals Sussex NHS Foundation Trust, Brighton, UK

[12]Central and North West London NHS Foundation Trust, London, UK

[13]North Manchester General Hospital, Manchester, UK

[14]Africa Advocacy Foundation, London, UK

Correspondence
Zoe Ottaway, School of Immunology and Microbial Sciences, King's College London, Weston Education Centre (rm 2.50), Cutcombe Road, London SE5 9RJ, UK.
Email: zoe.ottaway@nhs.net

Funding information
Medical Research Council (UK) Confidence in Concept scheme, Grant/Award Number: MC_PC_17164; Gilead Sciences, Grant/Award Number: IN-UK-983-6053

Abstract

Objectives: To describe the clinical epidemiology of COVID-19 in people of black ethnicity living with HIV in the UK.

Methods: We investigated the incidence and factors associated with COVID-19 in a previously established and well-characterized cohort of black people with HIV. Primary outcomes were COVID-19 acquisition and severe COVID-19 disease (requiring hospitalization and/or resulting in death). Cumulative incidence was analysed using Nelson–Aalen methods, and associations between demographic, pre-pandemic immune-virological parameters, comorbidity status and (severe) COVID-19 were identified using Cox regression analysis.

Results: COVID-19 status was available for 1847 (74%) of 2495 COVID-AFRICA participants (median age 49.6 years; 56% female; median CD4 cell count = 555 cells/μL; 93% HIV RNA <200 copies/mL), 573 (31%) of whom reported at least one episode of COVID-19. The cumulative incidence rates of COVID-19 and severe COVID-19 were 31.0% and 3.4%, respectively. Region of ancestry (East/Southern/Central vs. West Africa), nadir CD4 count and kidney disease were associated with COVID-19 acquisition. Diabetes mellitus [adjusted hazard ratio (aHR) = 2.39, 95% confidence interval (CI): 1.26–4.53] and kidney disease (aHR = 2.53, 95% CI: 1.26–4.53) were associated with an increased risk, and recent CD4 count >500 cells/μL (aHR = 0.49, 95% CI: 0.25–0.93) with a lower risk of severe COVID-19.

Conclusions: Region of ancestry was associated with COVID-19 acquisition, and immune and comorbidity statuses were associated with COVID-19 disease severity in people of black ethnicity living with HIV in the UK.

KEYWORDS

black, COVID-19, hospitalization, HIV, incidence

INTRODUCTION

The global SARS-CoV-2 pandemic disproportionally affected people of African ancestry, with those people of black ethnicity being at greater risk of COVID-19 acquisition, severe morbidity, and mortality [1, 2]. The reasons for such disparity remain poorly understood; an interplay between socioeconomic, cultural and behavioural differences and biological vulnerabilities probably contributed to the increased risk of severe COVID-19 disease [3]. Studies in the general population show severe COVID-19 to be associated with several comorbid conditions, including obesity, hypertension, diabetes mellitus, cardiovascular and kidney disease [4, 5]. Additionally, HIV infection has been associated with higher rates of COVID-19 morbidity and mortality [6–11], with those of black ethnicity, low CD4 cell counts and/or detectable HIV RNA levels and multi-morbidity at greatest risk of adverse COVID-19 outcomes [10, 12–15]. Some observational studies suggest that tenofovir disoproxil (TDF) may affect COVID-19 outcomes [16–19]; TDF, as compared with tenofovir alafenamide (TAF), has been associated with protection against SARS-CoV-2 acquisition [17, 19], severe disease (including hospitalization) [17, 18] and death [16], although this may reflect differences in populations treated with TDF and TAF (Table S1).

The COVID-AFRICA study was conducted to investigate the clinical epidemiology of COVID-19 in people of black ethnicity living with HIV in the UK. This paper describes the incidence of COVID-19, and the demographic, comorbid and immunovirological associations.

METHODS

Study population

People were eligible to participate in the COVID-AFRICA study if they were living with HIV in UK, were of self-reported black African, black Caribbean or other black ethnicity, had previously participated in the Genetic Determinants of Kidney Disease in People of African Ancestry with HIV (GEN-AFRICA) study (NCT05685810), and had remained in HIV care at one of 12 participating sites on 1 January 2020. Participation in GEN-AFRICA was open to all adults of black ethnicity with HIV who were able and willing to provide informed consent, demographic and clinical data, and a blood and urine sample for research. Participation in the COVID-AFRICA study was through actual enrolment between June 2021 and November 2022, after participants had provided written informed consent, or through review of medical records (including of those who died or became lost to follow-up) between June and November 2022; the study was approved by a National Health Service Research Ethics Committee (21/ES/0047) and the Health Research Authority (IRAS 294887).

Exposure variables

The following parameters were obtained from the GEN-AFRICA study visit, conducted between May 2018 and January 2020: age, sex at birth, region of ancestry based

on the country of birth of both parents (East, Southern/Central or West Africa, Caribbean, or other), time of HIV diagnosis and initiation of antiretroviral therapy (ART), details of ART, current and nadir CD4 count, HIV RNA and prior AIDS diagnoses. We also used information from the GEN-AFRICA visit to assess comorbid conditions: diabetes mellitus, hypertension and cardiovascular disease (comprising ischaemic heart disease, congestive cardiac failure, cardiomyopathy, peripheral vascular disease and stroke) were predominantly self-reported diagnoses with corroboration from the medical records; kidney disease was defined by an estimated glomerular filtration rate (eGFR) <60 mL/min per 1.73 m^2, and obesity by a body mass index ≥ 30 kg/m^2.

Outcomes

COVID-19 status, details of the first COVID-19 episode and COVID-19 vaccination status were ascertained through direct participant questioning and additional review of medical records. Enrolled participants were asked to report onset and duration of symptoms, including cough, shortness of breath, constitutional symptoms and loss of smell or taste, and medical complications including cardiovascular events, respiratory or kidney failure, disease severity including details of hospitalization, and the results of COVID diagnostics. Disease severity was categorized as mild (staying at home with mild illness), moderate (staying at home, taking to bed and feeling quite ill) or severe (requiring hospitalization or resulting in death). For those who could not be enrolled in the study, we obtained vital status, and COVID-19 clinical and vaccination status from the medical records, where available.

Statistical analyses

The COVID-Africa cohort is described in terms of demographics and pre-pandemic HIV characteristics and comorbid status, using medians with interquartile ranges (IQRs), or numbers and percentages as appropriate. Baselines characteristics of the study population, stratified by COVID-19 status were compared using Pearson's χ^2 test for categorical variables and Kruskal–Wallis test or analysis of variance for continuous variables, as appropriate. Time to the first episode of COVID-19 was analysed; participants who had not experienced COVID-19 were censored at the last visit up to November 2022 for which the COVID-19 status could be evaluated. We calculated the cumulative incidence of the first episode of COVID-19 (any severity) and severe COVID-19 using Nelson–Aalen methods. As confirmatory diagnostics were severely restricted in the early weeks of the pandemic, we included both laboratory-confirmed and clinical COVID-19 episodes.

Associations between demographic and clinical parameters and (severe) COVID-19 were analysed using Cox proportional hazard models. Factors associated ($p < 0.1$) with COVID-19 in univariable analysis were included in multivariable models. We performed two sensitivity analyses in which data were restricted to (1) sites where the COVID-19 status was available for >90% of participants, and (2) the first year of the pandemic (31 December 2020), before vaccination became available to study participants. All analyses were performed using STATA v17 (StataCorp, College Station, TX, USA).

RESULTS

Participant characteristics

Of the 2907 GEN-AFRICA participants, 2495 (86%) were included in the COVID-AFRICA study: 927 through active enrolment and 1568 through provision of data. The current analyses were restricted to the 1847 (74%) individuals for whom COVID-19 status could be determined (Figure S1); those with undetermined COVID-19 status were younger, more often of West African ancestry, and they had lower nadir CD4 counts and more often reported an AIDS diagnosis (Table S2).

The demographic and clinical characteristics of the 1847 participants, stratified by COVID-19 status, are shown in Table 1. Participants had a median age of 49.6 (IQR: 43.0–55.9) years, 56% were female, and most (80%) were of sub-Saharan African ancestry, with long-standing (median 14.0 years) and well-controlled HIV (median CD4 count = 555 cells/μL, 93% suppressed HIV RNA). Obesity and hypertension were present in 41% and 33% of participants, respectively, and 4–10% had diabetes mellitus, kidney disease or cardiovascular disease.

During median (IQR) follow up of 33.3 (27.9–34.2) months, 573 (31%) participants experienced at least one episode of COVID-19; 510 (89%) of these were mild or moderately severe while 63 (11%) were episodes of severe COVID-19, resulting in death in five participants. Differences in baseline characteristics of those with no, mild-to-moderately severe, and severe COVID-19 were predominantly driven by those who developed severe disease who were older, more often male, of West African ancestry, had lower recent CD4 counts and lower rates of HIV suppression; a greater proportion had hypertension,

TABLE 1 Baseline characteristics of the study participants stratified by COVID-19 status.

Pre-pandemic participant characteristics		Total ($N = 1847$)	No COVID-19 ($N = 1274$)	COVID-19 (mild-to-moderately severe) ($N = 510$)*	COVID-19 (severe) ($N = 63$)	*p*-value
Demographic parameters						
Age (years)	Median (IQR)	49.6 (43.0–55.9)	49.9 (43.4–56.2)	48.4 (41.8–54.9)	53.5 (45.0–59.5)	<0.001
Sex, female	*N* (%)	1035 (56.1)	685 (53.8)	326 (64.0)	24 (38.1)	<0.001
Region of ancestry						
West Africa	*N* (%)	620 (33.6)	463 (36.3)	130 (25.6)	27 (42.9)	<0.001
East Africa		371 (20.1)	233 (18.3)	121 (23.8)	17 (27.0)	
Southern/Central Africa		489 (26.6)	322 (25.3)	156 (30.7)	11 (17.5)	
Caribbean		252 (13.7)	175 (13.8)	73 (14.4)	4 (6.4)	
Other/unknown		110 (6.0)	78 (6.1)	28 (5.5)	4 (6.4)	
HIV parameters						
Time since HIV diagnosis (years)	Median (IQR)	14.0 (9.0–18.0)	14.0 (9.0–18.0)	15.0 (10.0–19.0)	15.0 (11.0–20.0)	0.03
Prior AIDS diagnosis	*N* (%)	187 (10.5)	123 (9.9)	54 (11.1)	10 (17.2)	0.19
Nadir CD4 cell count (cells/μL)	Median (IQR)	214 (86–346)	199 (81–343)	236 (104–362)	202 (65–357)	0.08
Recent CD4 cell count (cells/μL)	Median (IQR)	555 (400–731)	548 (393–731)	587 (430–748)	435 (258–621)	<0.001
HIV RNA <200 copies/mL	*N* (%)	1716 (93.2)	1188 (93.5)	474 (93.3)	54 (85.7)	0.06
Antiretroviral backbone						
Tenofovir disoproxil fumarate	*N* (%)	821 (49.0)	571 (49.3)	236 (50.8)	14 (26.9)	0.002
Tenofovir alafenamide		258 (15.4)	177 (15.3)	64 (13.8)	17 (32.7)	
Abacavir		596 (35.6)	410 (35.4)	165 (35.5)	21 (40.4)	
Antiretroviral third agent						
NNRTI	*N* (%)	697 (40.4)	488 (41.0)	192 (40.3)	17 (29.8)	0.004
INSTI		497 (28.8)	328 (27.5)	157 (32.9)	12 (21.1)	
Protease inhibitor		531 (30.8)	375 (31.5)	128 (26.8)	28 (49.1)	
Comorbid status						
Obesity[a]	*N* (%)	739 (40.9)	493 (39.7)	217 (43.5)	29 (46.0)	0.24
Hypertension	*N* (%)	600 (32.6)	418 (32.9)	148 (29.2)	34 (54.0)	<0.001
Diabetes	*N* (%)	177 (9.7)	118 (9.3)	43 (8.5)	16 (25.4)	<0.001
Kidney disease[b]	*N* (%)	129 (7.0)	80 (6.3)	32 (6.3)	17 (27.0)	<0.001
Cardiovascular disease[c]	*N* (%)	73 (4.0)	50 (3.9)	18 (3.5)	5 (7.94)	0.24

Abbreviations: IQR, interquartile range; INSTI, integrase strand-transfer inhibitor; NNRTI, non-nucleoside reverse-transcriptase inhibitor.
*Including those with unknown severity status; $N = 44$.
[a]Obesity is defined as body mass index ≥30 kg/m^2.
[b]Kidney disease is defined as estimated glomerular filtration rate <60 mL/min/1.73 m^2.
[c]Cardiovascular disease is defined as myocardial infarction, coronary artery disease, peripheral vascular disease, stroke, heart failure and/or cardiomyopathy.

diabetes or kidney disease, and their ART more often included a protease inhibitor (PI) and less often TDF (Table 1). A total of 1563 (88%) had received at least one dose of SARS-CoV-2 vaccine from January 2021 onwards.

The overall incidence rates of COVID-19 and severe COVID-19 by December 2022 were 31% and 3.4%, respectively. By May 2020, February 2021, September 2021 and February 2022, when high rates of infections

TABLE 2 Associations between demographic, clinical and immunovirological parameters and COVID-19.

		COVID-19 (any severity)				Severe COVID-19			
		Univariable		Multivariable		Univariable		Multivariable	
		HR (95% CI)	*p*-value	HR (95% CI)	*p*-value	HR (95% CI)	*p*-value	HR (95% CI)	*p*-value
Age	<50 years	1				1		1	
	50–59 years	0.92 (0.77–1.10)	0.34			1.67 (0.95–2.95)	0.08	1.18 (0.62–2.24)	0.62
	≥60 years	0.87 (0.67–1.15)	0.33			2.91 (1.51–5.61)	0.001	1.62 (0.76–3.47)	0.21
Sex	Male (vs. female)	0.79 (0.67–0.94)	0.01	0.89 (0.74–1.07)	0.22	2.04 (1.23–3.39)	0.01	1.64 (0.94–2.87)	0.08
Region of ancestry	West Africa	1		1		1		1	
	East Africa	1.56 (1.24–1.97)	<0.001	1.64 (1.28–2.11)	<0.001	1.11 (0.61–2.04)	0.73	1.38 (0.71–2.67)	0.34
	Southern/Central Africa	1.40 (1.13–1.75)	0.002	1.41 (1.11–1.78)	0.004	0.54 (0.27–1.08)	0.08	0.71 (0.33–1.50)	0.37
	Caribbean	1.24 (0.94–1.62)	0.13	1.32 (0.99–1.76)	0.06	0.37 (0.13–1.06)	0.06	0.40 (0.14–1.16)	0.09
	Other/unknown	1.14 (0.78–1.67)	0.49	1.21 (0.81–1.82)	0.35	0.84 (0.29–2.39)	0.74	1.29 (0.44–3.83)	0.64
Time since HIV diagnosis	Per additional year	1.02 (1.00–1.03)	0.02	1.00 (0.99–1.02)	0.79	1.02 (0.99–1.06)	0.24		
Prior AIDS	Yes (vs. no)	1.21 (0.93–1.57)	0.15			1.90 (0.96–3.76)	0.07	1.37 (0.68–2.74)	0.37
Nadir CD4 cell count	<200 (vs. ≥200) cells/μL	0.80 (0.67–0.95)	0.01	0.79 (0.66–0.94)	0.01	1.05 (0.64–1.74)	0.85		
Recent CD4 cell count	<350 cells/μL	1				1		1	
	350–500 cells/μL	1.11 (0.85–1.45)	0.44			0.57 (0.30–1.08)	0.09	0.72 (0.36–1.44)	0.35
	>500 cells/μL	1.20 (0.96–1.51)	0.11			0.36 (0.20–0.64)	<0.001	0.49 (0.25–0.93)	0.03
HIV RNA	<200 (vs. ≥200) copies/mL	0.94 (0.69–1.28)	0.69			0.45 (0.22–0.91)	0.03	0.53 (0.24–1.15)	0.11
Obesity[a]	Yes (vs. no)	1.18 (1.00–1.40)	0.05	1.20 (1.00–1.43)	0.05	1.28 (0.78–2.10)	0.33		
Hypertension	Yes (vs. no)	0.99 (0.83–1.18)	0.89			2.47 (1.50–4.05)	<0.001	1.40 (0.74–2.64)	0.30
Diabetes	Yes (vs. no)	1.16 (0.88–1.51)	0.29			3.37 (1.91–5.79)	<0.001	2.39 (1.26–4.53)	0.01
Kidney disease[b]	Yes (vs. no)	1.37 (1.02–1.84)	0.03	1.67 (1.22–2.28)	0.001	5.36 (3.07–9.35)	<0.001	2.53 (1.25–5.12)	0.01
Cardiovascular disease[c]	Yes (vs. no)	1.02 (0.67–1.55)	0.91			2.09 (0.84–5.22)	0.11		

Abbreviations: CI, confidence interval; HR, hazard ratio.

[a]Obesity is defined as body mass index ≥30 kg/m^2.

[b]Kidney disease is defined as estimated glomerular filtration rate <60 mL/min/1.73 m^2.

[c]Cardiovascular disease is defined as myocardial infarction, coronary artery disease, peripheral vascular disease, stroke, heart failure and/or cardiomyopathy.

from the founder strain and the alpha, delta and omicron variants had largely subsided, the cumulative incidences of any COVID-19 were 6.9%, 14.5%, 18.3% and 25.7%, and for severe disease were 0.8%, 2.0%, 2.5% and 3.1%, respectively (Figure S2).

Female sex, region of ancestry, (greater) nadir CD4 count, obesity and kidney disease were associated with COVID-19 acquisition (any symptom severity) in univariable analysis. Of these, East and Southern/Central African ancestry, nadir CD4 count, and kidney disease remained associated with COVID-19 acquisition in the adjusted analyses. Age, recent CD4 count, HIV RNA, hypertension and diabetes mellitus were not associated with COVID-19 acquisition (Table 2). In analyses of severe COVID-19, age ≥60 years, male sex, current CD4 count, HIV RNA, hypertension, diabetes mellitus and kidney disease were associated in univariable analysis; of these, a current CD4 count >500 cells/μL was protective against severe COVID-19, while diabetes mellitus and kidney disease remained associated with an increased risk of severe COVID-19 in multivariable analysis.

We additionally investigated whether ART exposures were associated with COVID-19 acquisition or severe COVID-19 disease (Table S3). None of the commonly used ART backbone agents (TDF, TAF or abacavir) and classes of ART third agents [non-nucleoside reverse transcriptase inhibitors (NNRTIs), integrase strand-transfer inhibitors or PIs] were associated with COVID-19 acquisition. TDF [vs. abacavir; hazard ratio (HR) = 0.48, 95% confidence interval (CI): 0.24–0.94], TAF (vs. abacavir; HR = 1.92, 95% CI: 1.01–3.64) and protease inhibitors (vs. NNRTI; HR = 1.94, 95% CI: 1.06–3.56) were significantly associated with severe COVID-19 in univariable analysis. The associations with TAF and PI were attenuated and no longer significant in multivariable analysis, while the association with TDF was largely unaffected (adjusted HR = 0.48, 95% CI: 0.22–1.03).

We performed two sensitivity analyses: factors associated with COVID-19 (any severity), restricted to two sites where the COVID-19 status was available for >90% of participants (Table S4), and factors associated with COVID-19 (any severity) and severe COVID-19, restricted to the first year of the pandemic before SARS-CoV-2 vaccines became available (Table S5). The results of these analyses were consistent with the findings reported for the full cohort.

DISCUSSION

We analysed COVID-19 incidence and risk factors in a large, well-characterized cohort of people of African ancestry with HIV. Although the incidence of COVID-19 was in line with the general UK population, the proportion who developed severe disease requiring hospitalization and/or resulting in death was approximately two-fold higher [20]. Immunovirological and comorbidity status were predictors of severe COVID-19, while antiretroviral exposures were no longer associated with severe disease after adjustment for confounders.

Our data support the assertion that comorbidities such as diabetes mellitus and kidney disease increase the risk of severe COVID-19 [6, 10, 11, 15]. Kidney disease was also associated with COVID-19 acquisition, which may reflect increased community or hospital-associated exposure to, or regular testing of individuals with severe kidney disease for, COVID-19. Unlike reports from the general population [4, 5], we did not observe an association between obesity and severe COVID-19. The association between hypertension and severe COVID-19 was attenuated following multivariable adjustment and no longer significant. Our study had limited power to detect associations between cardiovascular disease and (severe) COVID-19.

The notion that higher CD4 counts were associated with protection against severe COVID-19 outcomes in our participants is consistent with previous reports [10, 12, 13, 15]. The association between HIV RNA level and severe COVID-19 outcomes was attenuated after adjustment [7]. We found no evidence that specific antiretrovirals, including TDF, provided protection against COVID-19 acquisition. However, TDF was associated with a reduced hazard of severe COVID-19, while exposure to TAF and PI were associated with an increased risk; the associations with TAF and PI were attenuated in the adjusted analyses and no longer statistically significant. The association with TDF was largely unaffected by the adjustment for other factors and of borderline statistical significance; a possible contribution of unmeasured bias or confounding cannot be excluded.

The strengths of our study include the ethnic diversity of the study participants and the use of both community and hospital COVID-19 episodes. The inclusion of early, commonly unconfirmed episodes of COVID-19 provides an important insight into the early stages of the pandemic but may have resulted in misclassification of some cases. Potential bias from non-availability of COVID-19 status was explored in a sensitivity analysis, which was consistent with the findings in all participants. Although we were unable to analyse the effects of COVID-19 vaccination, analyses restricted to COVID-19 episodes before the vaccines became available were consistent with the main analyses. Unfortunately, we were unable to link our study participants to the national COVID-19 registry, which means that mortality may have been underestimated in this geographically mobile cohort.

In summary, severe COVID-19 was associated with poor HIV immune-virological control and pre-pandemic comorbidity status in people of African ancestry in the UK, a population under-represented in previous studies. We found no evidence that specific ART provided protection against COVID-19 acquisition, and no robust evidence that TDF provided protection against severe COVID-19 disease. Further work is needed to explore genetic susceptibility to COVID-19, and the role of social determinants of health and health beliefs in these communities, ahead of future pandemics.

AUTHOR CONTRIBUTIONS

The study was designed by ZO, FP and LC, with input from the community (DO). FAP, JF, FB, LH, SK, MR, SS, DP, RJ, AC and AU were site principal investigators, and FAP, JF, FB, LH, SK, MR, SS, DP, RJ, AC, IM and AU coordinated recruitment and data collection at their sites. LC assisted with logistic and governance aspects. ZO performed the analyses with input from FAP and LC. ZO, FAP, LC and RFM interpreted the findings. ZO wrote the first draft of the manuscript with input from FAP. All authors revised and approved the final version of the manuscript.

ACKNOWLEDGEMENTS

The authors would like to thank the study participants, participating sites and all members of the COVID-AFRICA study group (Appendix A).

FUNDING INFORMATION

The GEN-AFRICA study was supported by the Medical Research Council (UK) Confidence in Concept scheme (MC_PC_17164); the COVID-AFRICA study was funded through a grant from Gilead Sciences (IN-UK-983-6053).

CONFLICT OF INTEREST STATEMENT

ZO, LC, LRC, NP, JF, FB, LH, SK, MR, SS, DP, RJ, IM, AU, DO, ST and RFM declared no competing interests. AC declares no competing interests for this paper but reports advisory boards and speaker fees from Gilead Sciences, MSD and Viiv Healthcare; conference travel support from Gilead Sciences and ViiV healthcare. FAP reports grants and personal fees and non-financial support from Gilead, grants and personal fees and non-financial support from ViiV, and grants and personal fees and non-financial support from MSD during the conduct of the study.

DATA AVAILABILITY STATEMENT

The database contains personal and sensitive information and is therefore not publicly available. Access to the study data and/or samples is governed by the National Health Service data access policy and those of King's College Hospital NHS Foundation Trust, the study sponsor. The Gen-AFRICA and COVID-AFRICA studies are open to collaborations, and all requests from researchers who meet the criteria for access to fully anonymized patient-level data will be considered. Concepts can be submitted for review to the principal investigator (Prof. Frank Post; email: frank.post@kcl.ac.uk).

ETHICS STATEMENT

The study was approved by the local Research Ethics Committee (21/ES/0047) and the Health Research Authority (IRAS 294887).

ORCID

Zoe Ottaway https://orcid.org/0009-0008-9425-7662
Irfaan Mann https://orcid.org/0000-0003-2178-7263
Shema Tariq https://orcid.org/0000-0001-9802-7727
Frank A. Post https://orcid.org/0000-0002-2844-1612

REFERENCES

1. Apea VJ, Wan YI, Dhairyawan R, et al. Ethnicity and outcomes in patients hospitalised with COVID-19 infection in East London: an observational cohort study. *BMJ Open*. 2021;11(1):e042140.
2. Sze S, Pan D, Nevill CR, et al. Ethnicity and clinical outcomes in COVID-19: a systematic review and meta-analysis. *EClinicalMedicine*. 2020;29:100630.
3. Pareek M, Bangash MN, Pareek N, et al. Ethnicity and COVID-19: an urgent public health research priority. *Lancet*. 2020;395(10234):1421-1422.
4. Williamson EJ, Walker AJ, Bhaskaran K, et al. Factors associated with COVID-19-related death using OpenSAFELY. *Nature*. 2020;584(7821):430-436.
5. McFarlane E, Linschoten M, Asselbergs FW, Lacy PS, Jedrzejewski D, Williams B. The impact of pre-existing hypertension and its treatment on outcomes in patients admitted to hospital with COVID-19. *Hypertension Research*. 2022;45(5):834-845.
6. Geretti AM, Stockdale AJ, Kelly SH, et al. Outcomes of coronavirus disease 2019 (COVID-19) related hospitalization among people with human immunodeficiency virus (HIV) in the ISARIC World Health Organization (WHO) clinical characterization protocol (UK): a prospective observational study. *Clin Infect Dis*. 2021;73(7):e2095-106.
7. Brown AE, Croxford SE, Nash S, et al. COVID-19 mortality among people with diagnosed HIV compared to those without during the first wave of the COVID-19 pandemic in England. *HIV Med*. 2022;23(1):90-102.
8. Bhaskaran K, Rentsch CT, MacKenna B, et al. HIV infection and COVID-19 death: a population-based cohort analysis of UK primary care data and linked national death registrations within the OpenSAFELY platform. *Lancet HIV*. 2021;8(1):e24-e32.
9. Biccard BM, Gopalan PD, Miller M, et al. Patient care and clinical outcomes for patients with COVID-19 infection admitted

to African high-care or intensive care units (ACCCOS): a multicentre, prospective, observational cohort study. *Lancet*. 2021; 397(10288):1885.
10. Jassat W, Cohen C, Tempia S, et al. Risk factors for COVID-19-related in-hospital mortality in a high HIV and tuberculosis prevalence setting in South Africa: a cohort study. *Lancet HIV*. 2021;8(9):e554-67.
11. Bertagnolio S, Thwin SS, Silva R, et al. Clinical features of, and risk factors for, severe or fatal COVID-19 among people living with HIV admitted to hospital: analysis of data from the WHO global clinical platform of COVID-19. *Lancet HIV*. 2022;9(7): e486-e495.
12. Nomah DK, Reyes-Urueña J, Díaz Y, et al. Sociodemographic, clinical, and immunological factors associated with SARS-CoV-2 diagnosis and severe COVID-19 outcomes in people living with HIV: a retrospective cohort study. *Lancet HIV*. 2021; 8(11):e701-e710.
13. Wit FW, Reiss P, Rijnders B, et al. COVID-19 in people with HIV in the Netherlands. *AIDS*. 2023;37(11):1671-1681.
14. Childs K, Post FA, Norcross C, et al. Hospitalized patients with COVID-19 and human immunodeficiency virus: a case series. *Clin Infect Dis*. 2020;71(8):2021-2022.
15. Shapiro AE, Bender Ignacio RA, Whitney BM, et al. Factors associated with severity of COVID-19 disease in a multicenter cohort of people with HIV in the United States, march-December 2020. *J Acquir Immune Defic Syndr*. 2022;90(4): 369-376.
16. Boulle A, Davies MA, Hussey H, et al. Risk factors for coronavirus disease 2019 (COVID-19) death in a population cohort study from the Western Cape Province. *SA Clin Infect Dis*. 2021;73(7):e2005-15.
17. Li G, Park LS, Lodi S, et al. Tenofovir disoproxil fumarate and coronavirus disease 2019 outcomes in men with HIV. *Aids*. 2022;36(12):1689-1696.
18. Del Amo J, Polo R, Moreno S, et al. Tenofovir disoproxil fumarate/emtricitabine and severity of coronavirus disease 2019 in people with HIV infection. *Aids*. 2022;36(15):2171-2179.
19. Berenguer J, Díez C, Martín-Vicente M, et al. Prevalence and factors associated with SARS-CoV-2 seropositivity in the Spanish HIV research network cohort. *Clin Microbiol Infect*. 2021; 27(11):1678-1684.
20. UK Health Security Agency. Coronavirus (COVID-19) in the UK: Cases. 2023 Available from: https://coronavirus.data.gov.uk/details/cases

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Ottaway Z, Campbell L, Cechin LR, et al. Clinical epidemiology of COVID-19 in people of black ethnicity living with HIV in the UK. *HIV Med*. 2024;1-8. doi:10.1111/hiv.13611

APPENDIX A

A.1 | COVID-AFRICA GROUP: STUDY SITES AND STAFF

University Hospitals Sussex NHS Foundation Trust, Brighton [Amanda Clarke (PI), Marion Campbell, Alyson Knott, Lisa Barbour, Vittorio Trevitt].

Chelsea and Westminster NHS Foundation Trust, London [Rachael Jones (PI), Alex Schoolmeesters, Shoreh Ghazali].

Guys and St Thomas's NHS Foundation Trust, London [Julie Fox (PI), Anele Waters, Mariusz Racz, Louise Terry, Kathy Arbis].

King's College Hospital NHS Foundation Trust, London [Frank Post (CI), Zoe Ottaway, Beatriz Santana-Suarez, Leigh McQueen, Lucy Campbell, Bee Barbini, Emily Wandolo, Rachel Hung, Luella Hanbury, Sarah Barber, Chris Taylor, Mary Poulton, Holly Middleditch, Itty Samuel, Candice McDonald, Vladimir Kolodin, Laura Cechin, Larissa Mulka, Amy John, Sally Hawkins, Madelaine Angus, Tim Appleby, Katherine Bainbridge, Hayley Cheetham, Julie Barker, Kate Childs, Rousell Roberts, Nivenjit Kaur, Lisa Shorrock, Kate El Bouzidi, Nisha Patel, Amelia Oliveira, Ayoma Ratnappuli, Kate Flanagan, Daniel Trotman, Nisha Mody, Michael Brady, Killian Quinn, Elizabeth Hamlyn, Gurjinder Sandhu, Verity Sullivan, Naomi Fitzgerald].

Leeds Teaching Hospitals NHS Trust, Leeds [Sarah Schoeman (PI), Gary Lamont, Natasha Calder-Smith, Tadas Mazeika, Kiran Chana].

North Manchester General Hospital, Manchester [Andrew Ustianowski (PI), Gabriella Lindergard, Jan Flaherty, Valerie George, Denise Kadiu, Alice Hendy, Kevin Kuriakose].

Mortimer Market Centre; Central and North West London NHS Foundation Trust, London [Sarah Pett (PI), Mah Jabeen Qamar, Jose Paredes Sosa, Konstantina Tetorou, Michelle Beynon, Adesola Yinka-Ogunleye, Gosala Gopalakrishnan, Irfaan Maan, Claire Mullender].

Newcastle Hospitals NHS Foundation Trust, Newcastle [David Ashley Price (PI), Bijal Patel, Ian McVittie].

Queen Elizabeth Hospital, Woolwich; Lewisham and Greenwich NHS Trust, London [Stephen Kegg (PI), Chloe Saad, Rosa Harrington, Kirsty Cunningham, Mandy Lewis].

Royal Free London NHS Foundation Trust, London [Fiona Burns (PI), Jonathan Edwards, Tom Fernandez, Qayo Egeh, Megan Bailey, Katie Spears].

St George's University Hospital NHS Foundation Trust, London [Lisa Hamzah (PI), Catherine Cosgrove, Hannah Laurence, Ameen Rahman, Katie Toler].

University Hospital Lewisham; Lewisham and Greenwich NHS Trust, London [Melanie Rosenvinge (PI), Claudia Adade, Shelley Campbell].

Africa Advocacy Foundation (Denis Onyango).