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Prevalence of cardiovascular risk factors in a historical cohort of people living with human immunodeficiency virus during a 10-year period

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Abstract

Objective: Data on the frequency of cardiovascular disease in people living with human immunodeficiency virus from low- and middle-income countries is scarce.

Methods: We performed an observational study based on data from a historical cohort of people living with human immunodeficiency virus in Colombia during a 10-year follow-up to describe the prevalence of cardiovascular risk factors and their behavior according to CD4 count.

Results: One thousand patients were initially included, out of which 390 had a 10-year follow-up. The mean age was 34 (standard deviation 10) years, and 90% were male. We observed an increase in the prevalence of dyslipidemia (29%–52%, $p < 0.001$) and obesity (1.1%–3.5%, $p < 0.001$). Major cardiovascular events occurred in less than 1% of patients. Patients with a CD4 count <200 cells/mm^3 had a higher frequency of acute myocardial infarction and obesity.

Conclusion: Over time, people living with human immunodeficiency virus present with an increasing prevalence of cardiovascular risk factors, particularly those with a lower CD4 count.

Keywords

HIV, heart disease risk factors, Latin America, prevalence, epidemiology

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Introduction

According to World Health Organization data, by 2021, about 38.4 million people were living with human immunodeficiency virus (HIV) infection, and there were 650,000 acquired immunodeficiency syndrome-related deaths.[1] With the initiation of antiretroviral therapy (ART), patients achieve a reduction in morbidity and mortality and a prolongation of life expectancy.[2,3] However, with HIV infection becoming a chronic disease, likewise, the causes of mortality in this group of patients have changed over time.[4]

As the life expectancy of people living with HIV (PLWH) rises, a simultaneous increase in cardiovascular disease has occurred.[5] The description of coronary artery disease, cerebrovascular disease, heart failure, and arterial hypertension is more frequent.[6–8] A model based on the Dutch ATHENA cohort estimated that from 2010 to 2030, 78% of patients would be diagnosed with cardiovascular disease.[9] However, in low-income countries such as Colombia, data are variable.[10]

Three factors have been proposed to explain this phenomenon, namely, (1) an increasing frequency of traditional risk factors, such as dyslipidemia, metabolic syndrome, and

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smoking; (2) the presence of chronic inflammation due to persistent activation of the immune system; and (3) the effects of certain ARTs.[6,11,12] Some cohorts have reported the follow-up of PLWH and its association with the onset of cardiovascular events.[12] However, the evolution of cardiovascular risk factors over time and their relationship with some disease characteristics (e.g., immune response, viral load control) still needs to be fully elucidated.

This study aims to describe a population of PLWH followed at a teaching hospital in Colombia and to evaluate the prevalence of cardiovascular risk factors during a 10-year follow-up.

Methods

We conducted an observational study using data from a historical cohort to describe the prevalence of cardiovascular risk factors over ten years. We included patients older than 18 years with a diagnosis of HIV infection who attended at the infectious diseases program at the Hospital Universitario San Ignacio, in Bogota, Colombia, between 2004 and 2018. We excluded patients with less than two follow-up visits during the 10 years. Each patient attended medical control at least every 2 months and performed lab workups at least once yearly.

Regarding data collection methodology, the information was obtained from the medical records and included: age, sex, years of follow-up, body mass index, viral load, and CD4+ T cell count. These data were recorded in an electronic database. A search was carried out in the registry of the variables in each clinical assessment the patient had throughout the years of follow-up to obtain data on cardiovascular risk factors.

Definitions

We defined HIV infection as two positive tests using two different methodologies (mainly third and fourth generation Enzyme-Linked Immunosorbent Assay), based on the practice clinical guidelines for diagnosis and management of HIV infection by the Colombian Association of Infectious Diseases[13]; chronic kidney disease (CKD) as the presence of a glomerular filtration rate less than 60 mL/min/1.73 m^2 estimated by the CKD-EPI equation[14]; type 2 diabetes mellitus (DM2) according to the diagnostic criteria of the American Diabetes Association 2020 guideline including HBA1C >6.5% or a random serum glucose measurement >200 mg/dL[15]; arterial hypertension as either systolic blood pressure >140 mmHg or diastolic blood pressure >90 mmHg.[16] To provide an operational definition for dyslipidemia, we adopted the definition provided by the 2019 ESC guidelines.[17] There is a lack of consensus regarding whether these patients should be considered a high-risk group in whom a stricter goal of low-density lipoprotein (LDL) should be achieved. Due to this lack of consensus, we selected a low-risk threshold to avoid overestimation. We evaluated only LDL levels as (1) an evidence-based cutoff point of triglyceride levels is lacking and (2) non-high-density lipoprotein (non-HDL) levels thresholds are proposed for non-low-risk patients; thus, we defined dyslipidemia as an LDL value >116 mg/dL.[17] In addition, the episodes of stroke, transient ischemic attack, and acute myocardial infarction were retrieved from the clinical charts. A low CD4+ T cell count at diagnosis was considered <200 cells/mm^3 to perform the subgroup analysis.[18] The prevalence per year of follow-up was calculated for dyslipidemia, arterial hypertension, DM2, obesity, CKD, and smoking habit.

Statistical analysis

We described quantitative variables using central tendency and measures of the dispersion according to their normality distribution using Shapiro–Wilk and categorical variables as absolute values and percentages. To calculate the prevalence, we divided the number of patients who met the criteria by the total number of patients who completed the specified follow-up. Additionally, we stratified patients according to their CD4+ T cell count (i.e., below or over 200 cells/mm^3 at diagnosis) and calculated the prevalence of the outcomes of interest in these subgroups. We performed a McNemar test[19] to assess the change in reported data on the prevalence of risk factors and cardiovascular outcomes.

Ethical considerations

This study was approved by the ethics and research committee of the Pontificia Universidad Javeriana and the San Ignacio University Hospital (FM-CIE-0619-18).

Results

Our historical cohort initially included 1000 patients and a total of 7301 visits over 10 years, with a mean of 7.7 follow-up visits per patient. As the retrieved data was obtained from yearly lab workups, the mean follow-up time per patient was 7.7 years. There was a reduction in the number of patients seen in the program each year, ending with 390 at 10 years. The mean age at admission was 34 years (standard deviation 10). Regarding the immunovirological variables, a progressive increase in the average CD4 count was observed between the first and the last visit, from 325 to 518 cells/mm^3 ($p < 0.001$). Regarding mortality, only six patients died from unspecified causes during follow-up. Table 1 presents general characteristics by year of follow-up.

Regarding the prevalence of cardiovascular risk factors (Supplemental Appendix 1), arterial hypertension was observed in 5% of patients on the first visit, with an over two-fold increase (10.7%) in the last visit at 10 years ($p = 0.243$). The prevalence of smoking habit was about 30%

Table 1. Sociodemographic characteristics, risk factors, and cardiovascular outcomes in people living with HIV infection by year of follow-up.

Variable	Year										*p*-Value
	1	2	3	4	5	6	7	8	9	10	
Number of patients	1000	997	944	895	840	696	574	504	461	390	
Men *n* (%)	879 (87,9)	877 (88)	829 (87)	791 (88)	743 (88)	617 (88)	509 (88)	451 (89)	410 (88)	352 (90)	
Age in years, mean (SD)	34 (10)	35 (10)	36.9 (10)	38 (9.8)	39.3 (9.8)	40.5 (9.4)	41.8 (9.3)	43.1 (9)	44.3 (9)	45.8 (8.7)	
CD4+ T cell count, mean (SD)	325 (233)	373 (212)	424 (248)	438 (207)	457 (213)	478 (216)	491 (226)	514 (227)	506 (227)	518 (231)	**<0.001**
BMI, mean (SD)	22.3 (3)	23.2 (23.2)	23.5 (3.1)	23.6 (3.1)	23.7 (3.1)	23.7 (3)	23.7 (2.9)	23.8 (2.9)	23.9 (3)	23.9 (3.2)	
Cardiovascular risk factors (%)											
Arterial Hypertension	5	5.32	5.61	5.92	6.43	7.04	8.54	8.53	9.11	10.77	0.243
Type 2 DM	1.5	1.50	1.80	2.01	2.26	3.02	3.31	3.37	3.47	4.10	0.85
Smoking habit	29.4	29.29	29.7	29.72	29.76	30.17	31.36	30.95	30.2	29.74	0.927
Dyslipidemia*	29.1	33.80	39.19	41.90	42.86	48.28	49.30	50.99	52.06	52.56	**0.001**
Obesity**	1.1	1.71	2.97	3.24	2.74	2.59	2.44	2.78	2.82	3.59	**<0.001**
Statins use	2.7	3.61	4.87	6.26	8.57	10.06	12.72	15.87	19.52	22.82	**0.001**
CKD	1.5	1.20	1.27	1.45	2.02	1.44	1.74	1.79	1.52	2.05	0.212
Cardiovascular outcomes (%)											
AMI	0.2	0.30	0.42	0.45	0.48	0.57	0.35	0.40	0.43	0.51	0.084
Stroke	0.3	0.20	0.42	0.22	0.24	0.14	0.00	0.00	0.22	0.51	0.074
Mortality, *n*	1	0	0	1	1	1	0	0	1	1	

AMI: acute myocardial infarction; BMI: body mass index; CKD: chronic kidney disease; DM: diabetes mellitus; SD: standard deviation; HIV: human immunodeficiency virus; LDL: low-density lipoprotein.
*Dyslipidemia was considered with an LDL >116 mg/dL.
**Obesity was considered with a BMI $\geqslant$30 kg/m^2.
The bold entries are statistically significant.

and remained constant throughout the follow-up. Thirty percent of the patients who entered the HIV program had dyslipidemia, and an increase of approximately 23% in the LDL value was documented during follow-up ($p < 0.001$, Supplemental Appendix 2). Likewise, we observed an increase in the use of statins over time, with a prevalence of 2.7% in the first year, reaching 22% in the 10th year ($p < 0.001$, Supplemental Appendix 2).

Regarding the prevalence of DM2, we observed an increase of almost three-fold when compared to the prevalence of the first year (1.5%–4.1%), although this change was not statistically significant ($p = 0.850$). Regarding the prevalence of obesity, we observed that it reached up to 3.5% of the cohort at year 10, with a statically significant change ($p < 0.001$). We observed a low prevalence (less than 1%) of acute myocardial infarction and cerebrovascular disease without variations during follow-up. No statistically significant differences were found for CKD ($p = 0.212$).

Additionally, a subgroup analysis was carried out concerning patients with a CD4 count lower than 200 cells at diagnosis (Figure 1). We observed a statistically significant increase in the frequency of acute myocardial infarction for years 8, 9, and 10 ($p < 0.001$) and in the frequency of obesity in the last year of follow-up in the group with CD4 <200 cells/mm^3. On the other hand, patients with >200 CD4 tended to present with more frequent arterial hypertension.

Discussion

Our study recorded the prevalence of cardiovascular risk factors and outcomes such as acute myocardial infarction, cerebrovascular disease, and mortality in a cohort of PLWH during a 10-year follow-up. We documented an increase in the prevalence of arterial hypertension, DM2, obesity, dyslipidemia, and CKD. However, only obesity and dyslipidemia had a statistically significant change over time. The prevalence of smoking habit was stable during the evaluated period.

Regarding the presence of obesity, in 2010, Malaza et al. published a study carried out in the rural area of KwaZulu-Natal, in South Africa, where the prevalence of HIV is the highest worldwide. The authors reported a prevalence of overweight and obesity of 45% and 24.5%, respectively. The high prevalence of obesity and overweight in this population was probably related to changes in agriculture and nutrition, with an increase in carbohydrate, highly refined fats, and oils intake, as well as an increase in sedentary behavior.[20] Comparing it with our cohort, men also had a mean body mass index within the normal range. However, the progression to obesity reached 4% of our population at 10 years. Different causes have been proposed to explain this finding. First, HIV infection *per se* and ART may alter lipid metabolism and distribution of adipose tissue with ectopic lipid deposits. Some studies have linked certain drugs to weight gain in HIV patients, including regimens based on integrase inhibitors, such as raltegravir, which is associated with a seven-fold increase in the risk of developing obesity.[21] Due to the lack of adequate data, we did not explore the effect of ART or lifestyle habits on the development of obesity. We may infer that PLWH requires strict monitoring of both nutritional and anthropometric variables to avoid weight gain and progression to obesity and to evaluate their treatment regime regularly.

Regarding the prevalence of arterial hypertension, we observed a trend toward an increase, with a two-fold surge when comparing the population in the first versus the 10th year. A previous study in the United States with a patient profile similar to ours reported that the prevalence of arterial hypertension and DM2 was lower than that of our cohort.[12] On the other hand, in a Spanish cohort of over 11,000 patients, up to 25% had arterial hypertension.[22] This finding is consistent with a high prevalence of hypertension in the Spanish population (approximately 40% over 18 years of age).[23] To explain the relationship between arterial hypertension and HIV infection, vascular dysfunction associated with HIV infection has been described. This phenomenon is attributed to a proinflammatory and prothrombotic state, endothelial dysfunction, direct viral infection to endothelial and vascular smooth muscle cells, and viral protein-mediated endothelial activation.[24] In this line, the follow-up of patients should include recording blood pressure to carry out early detection of arterial hypertension and, thus, define the requirement of pharmacological initiation and non-pharmacological management.

In our cohort, we observed an increase in the prevalence of DM2 over time, although it was not statistically significant. This phenomenon has been analyzed in different studies worldwide. For instance, the RAPID II study included 4000 Latin American patients, out of which 420 were Colombian, and a prevalence of diabetes mellitus of 0.8% was reported, which remained stable over time. However, an increase in the prevalence of DM2 was observed in a Spanish cohort, reaching 19%.[22] Further, in a prospective observational cohort in 12 Asian countries, the TAHOD-LITE Study, the authors found that among 4629 patients who started ART with normal fasting plasma glucose, 20% and 7% developed prediabetes and diabetes, respectively.[25] Despite this documented variability in prevalence, monitoring for DM2 is required to be systematic, which would allow lifestyle changes and nutritional monitoring to avoid the complications of this disease.

Regarding dyslipidemia, a cross-sectional study conducted in China that included 1581 newly diagnosed HIV-infected patients and 347 HIV-negative patients found a dyslipidemia prevalence (defined as either high total cholesterol (⩾200 mg/dL), high triglycerides levels (⩾150 mg/dL), high LDL levels (⩾130 mg/dL), or low HDL (<40 mg/dL)) of 75.6% among PLWH (no statistically difference with HIV—negative people), with high TC, high TG, high LDL, and low HDL in 8.4%, 33.9%, 8.5%, and 59.6%,

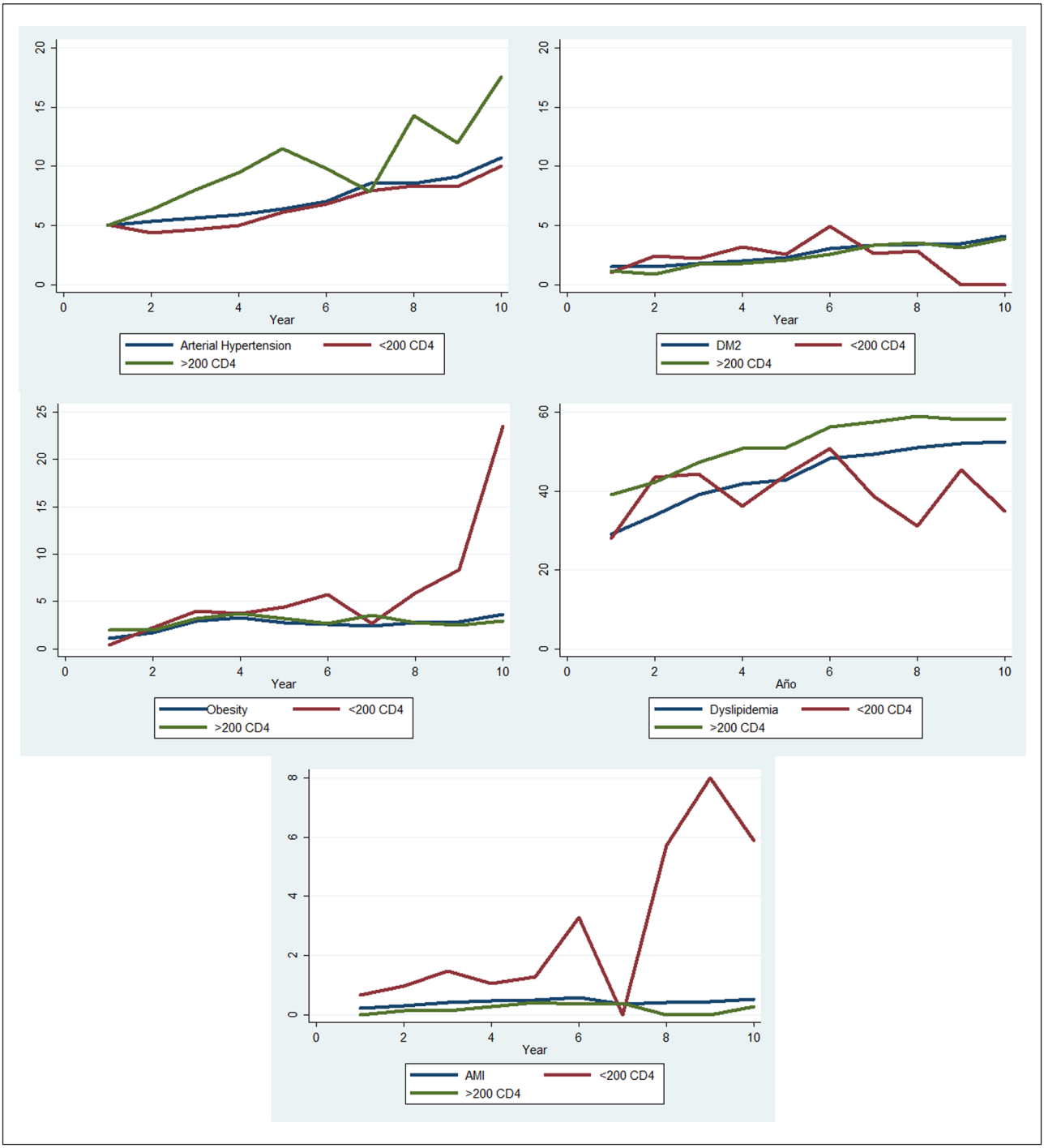

Figure 1. Comparison of patients with CD4 <200 cells/mm^3 versus >200 cells/mm^3.

respectively.[26] Although it is unfeasible to make a direct comparison with this study due to the different dyslipidemia definitions, more than 50% of both populations exhibit lipid abnormalities, highlighting the importance of this cardiovascular risk factor among PLWH. Another study published by El-Sadr et al. in 2005 analyzed the effect of some variables on the behavior of the lipid profile and blood glucose in a cohort without ART. Most patients were men (79%), with a mean age of 38.1 years. Regarding the lipid profile, they had a mean LDL of 98.3 mg/dL and triglycerides of 132.9 mg/dL,[27] with a prevalence of dyslipidemia of approximately 40%, similar to our cohort.

Different medications have been associated with the alteration of the lipid profile. Both nucleoside and non-nucleoside inhibitors of reverse transcriptase and protease inhibitors stand out, mainly due to hypertriglyceridemia and elevation of LDL values. In the D: A: D registry (Data Collection on Adverse Events of Anti-HIV Drugs), ritonavir-based treatment regimens more markedly increase triglyceride and LDL values.[28] The increase in the use of statins observed in our population may be related to the change in the indications and treatment goals established over time.

Several studies have evaluated the impact of a low CD4 count on the presence of cardiovascular risk factors. In the HOPS study, the authors documented that a CD4 count of fewer than 100 cells/mm^3 is associated with increased cardiovascular outcomes with a hazard ratio of 1.08 (confidence interval (CI) 1.01; 1.14). These events are probably related to a marked chronic inflammatory process that drives accelerated atherosclerosis and an increased risk of atherosclerotic plaque rupture.[23] Among patients with <200 CD4 cells/mm^3 at diagnosis, we found an increased prevalence of obesity at the end of follow-up and a statistically significant increase in the frequency of acute myocardial infarction, similar to the meta-analysis performed by Eyawo et al.[29] When compared with the VACS study, the authors observed that the cohort of patients with HIV infection has an increased risk of acute myocardial infarction when compared to the general population, HR of 1.48 (CI 1.27; 1.72).[30] Our study could not corroborate this phenomenon due to the low frequency of cardiovascular events, mainly because of the young age of the patients during follow-up.

It should be noted that our study describes the Colombian population with HIV and reports the prevalence of cardiovascular risk factors in this population, in whom controls were carried out systematically following national recommendations. We acknowledge some drawbacks. First, the patients in the cohort did not complete the stipulated time of follow-up, which could have generated that the changes in prevalence over time did not become statistically significant despite having a trend toward the increase in cardiovascular risk factors. However, with the obtained data, it was possible to shed light on the behavior of these factors over time. Second, as the mean age of our patients at the beginning of follow-up was that of young adults, a longer-term analysis is required to evaluate the behavior of cardiovascular outcomes as the population ages. Third, as we did not obtain the ART received by the patients from the registry, it is impossible to analyze the behavior of cardiovascular risk factors concerning this variable. Fourth, although a statistical analysis between comorbidities and virological variables, such as viral load, would have been of significant relevance, we could not explore these associations as most (>95%) of our patients achieved an undetectable viral load after six months on ART. Fifth, the lack of an aging control group undermined statistical analysis to establish whether HIV infection is associated with an increased risk of developing cardiovascular risk factors over time. Sixth, due to the nature of our historical cohort, we did not perform a sample size calculation.

Conclusion

In PLWH, the prevalence of cardiovascular risk factors appears to increase over time, particularly an increase in LDL levels and obesity. This suggests that patients with HIV infection require multidisciplinary follow-up and strict metabolic control to carry out interventions promptly.

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Author contributions

All authors contributed to the conception and design of the study. Material preparation, data collection, and analysis were performed by Ángel García-Peña, Jairo Aldana, Juan David Botero, Juan Manuel Vasquez, Miguel León, Diego Rodríguez-Lugo, Lina Villamil, Julián E. Barahona-Correa, and Jose Tamara. Statistical analysis was performed by Ángel García-Peña and Miguel León. The manuscript was drafted by Ángel García-Peña, Miguel León, Diego Rodríguez-Lugo, Lina Villamil, and Julián E. Barahona-Correa; all authors commented on previous versions of the manuscript and revised it critically for important intellectual content. All authors read and approved the final manuscript. All authors have participated sufficiently in the work to take public responsibility for appropriate portions of the content.

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Data availability statement

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Supplemental material

Supplemental material for this article is available online.

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