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ORIGINAL PAPER

Significance of C-reactive protein to albumin ratio and thrombus burden in acute coronary syndrome

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ABSTRACT

Introduction and aim. Thrombus burden (TB) is a critical factor in the pathogenesis of acute coronary syndrome (ACS) pathogenesis with biomarkers such as C-reactive protein (CRP) and serum albumin that reflect systemic inflammatory and nutritional states. The CRP and albumin ratio (CAR) has emerged as a new composite marker, offering enhanced prognostic value. The aim of this study was to evaluate the association between CAR and TB in patients with ACS and to assess the predictive utility of CAR compared to CRP and albumin individually.

Material and methods. A hospital cross-sectional analytical study was conducted among 93 patients ages 18–60 years with ACS who underwent coronary angiography (CAG). CAR was calculated and its association with TB was analyzed.

Results. Of the participants, 9.7% had high tuberculosis. CAR, CRP, and albumin were significantly associated with TB ($p<0.001$). CAR showed the highest correlation ($r=0.728$) and perfect diagnostic accuracy (AUC=1), outperforming CRP (AUC=0.987) and albumin (AUC=0.030). High TB was significantly associated with the presentation of grade 1 TIMI and ST-elevated myocardial infarction (STEMI) presentation ($p<0.05$).

Conclusion. CAR is a reliable, accessible and independent biomarker for predicting TB in ACS, and its incorporation into standard clinical protocols could improve early risk stratification, therapeutic decision-making, and patient outcomes. More multicentric study are warranted to validate its broader clinical applicability.

Keywords. acute coronary syndrome, albumin, C-reactive protein, thrombus burden

Introduction

Acute coronary syndrome (ACS) characterized by sudden, reduced blood flow to the heart, causing symptoms includes unstable angina (UA), non-ST segment elevation myocardial infarction (NSTEMI), and ST segment elevation myocardial infarction (STEMI).[1,2] Despite advances in therapeutic strategies, ACS remains a significant global health problem.[3] The pathophysiology of ACS involves atherosclerotic plaque rupture, endothelial dysfunction, and thrombus formation, leading to partial or complete coronary arteries.[1,2] Among these thrombus formation following plaque rupture plays a critical role in determining the severity of myocardial ischemia and infarction.[4,5]

Thrombus burden (TB) is the amount of thrombus present in the blood vessel,[6] and it is a crucial factor in determining the severity of myocardial ischemia and infarction in ACS and is essential for risk stratification and management.[7] As patients with high TB are more likely to experience extensive myocardial damage, increased coronary artery obstruction, and an increased risk of major adverse cardiac events (MACE).[8] Accurate

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assessment of TB is crucial to determine the severity of ACS and guiding therapeutic interventions. Techniques like angiographic evaluation, intravascular ultrasound (IVUS), and optical coherence tomography (OCT) are used to visualize TB and assess its composition.[9]

Additionally, systemic inflammation is crucial in the pathogenesis of ACS, contributing to plaque formation, instability, and thrombus generation.[5,9,10] C-reactive protein (CRP), an acute phase reactant produced by the liver, is a biomarker of inflammation, associated with worse outcomes in patients with ACS.[11-14] Elevated levels of CRP are associated with increased mortality and a higher incidence of MACE.[15,16] However, relying on a single biomarker may not fully capture the complexity of inflammatory and thrombotic processes in ACS.

Recently, the ratio of C-reactive protein to albumin (CAR) has emerged as a novel prognostic marker that reflects both inflammation and nutritional status.[11–14,17,18] CAR provides a comprehensive understanding of the condition of a patient, including hypoalbuminemia,[19] which has been associated with worse outcomes in cardiovascular disease (CVD).[20,21] Furthermore, since both inflammation and malnutrition have been shown to influence the development of thrombotic complications, it is plausible that CAR may correlate with TB in patients with ACS.[16]

In various studies, CAR has been associated with high coronary artery resistance and significant predictor significance of CAR.[22,23] In another study, in patients with acute myocardial infarction (AMI), high CAR has been associated with larger infarct sizes, more extensive coronary artery disease (CAD), and poorer long-term outcomes.[11]

Aim

Despite these findings, our study addresses the limited evidence on the link between CAR and TB in ACS patients which remains underexplored. Although CRP and albumin have been linked to outcomes in ACS, no previous study has directly compared their CAR with angiographically quantified TB. Therefore, by evaluating CAR along with the standardized TIMI thrombus grading, our research uniquely integrates systemic inflammation, nutritional status, and direct measures of intracoronary clot load. This novel approach aims to determine whether CAR can serve as a readily available biomarker for early risk stratification in patients with ACS. With this background, the present study aims to investigate the relationship between CAR and TB in patients with ACS.

Material and methods

This hospital-based analytical cross-sectional study was conducted in the Department of General Medicine at the Mahatma Gandhi Medical College and Research Institute, Puducherry, during the period of November 2022 to September 2024. Ethical approval was obtained from the institutional Human Ethics Committee (IHEC) (MGMCRI/Res/04/2022/121) and all participants gave their written informed consent.

Inclusion criteria for study participants include age group of 18 to 60 years with signs and symptoms of typical chest pain and were diagnosed as STEMI, NSTEMI and ACS who requires coronary angiogram. patient who was treated with primary PCI was included. While patients diagnosed with UA, a history of coronary artery bypass grafting (CABG), active malignancy, infection and connective tissue disorder, and pregnant and lactating women were excluded from the study.

Sample size determination and sampling procedure

Assuming that the prevalence of high TB was 59.6% in ACS patients from the study conducted by Duman et al.,[17] the minimum calculated sample size was 93 using the formula

$$n \geq \frac{Z^2_{\frac{1-\alpha}{2}} \times p\,(1-p)}{d^2} = \frac{(1.96)^2 \times 0.596\,(0.404)}{(0.10)^2} = 93$$

($Z_{\alpha/2}$ – 1.96; proportion (p) – 0.596; precision (d)) – 0.1) with a two-sided confidence interval of 95% and 10% precision. This sample provides >80% power to detect a moderate correlation (r~0.30) between CAR and TB at α 0.05. The consecutive sampling technique was used to include all patients with inclusion criteria until the desired sample size was achieved.

Data collection procedure

Data collection was carried out using a semistructured clinical proforma which included demographic details, risk factor assessment, anthropometric and clinical examination. Electrocardiogram (ECG) was taken at the time of admission. Routine investigations, including complete blood count, renal and liver function tests, serum electrolytes, cardiac enzymes, and measurements of serum CRP and albumin levels, at the time of admission. After the initial evaluation, patients who required coronary angiography (CAG) were performed together with the procedure according to the clinical protocol, without any intervention of the study. Only patients who underwent CAG and satisfied the inclusion criteria were included in the final analysis. The CAR was calculated and compared with the CAG results to assess its association with TB and the degree of arterial occlusion (AO). Patients who did not satisfied inclusion criteria were also treated according to the ACS treatment protocol (American Heart Association (AHA) guidelines) without violating ethical issues.

Blood samples (5 mL) were drawn on admission where hsCRP was measured by immunoturbidimetry (Roche Cobas c501; CV <5%) and serum albumin was

assessed by the bromocresol green method (CV <4%). Further, CAR was calculated as

$$CAR = \frac{CRP\ (\frac{mg}{L})}{albumin\ (\frac{g}{dL})}$$

TB was classified as high and low based on the validated Thrombolysis In Myocardial Infarction (TIMI)[24] classification to allow an objective assessment of the severity of thrombotic in ACS. Grades 0–2 defined as 'low TB' and grades 4–5 as "high TB". Two independent interventional cardiologists, blinded to biomarker levels, classified TB and resolved the discrepancies (where grade 3 adjudicated by consensus). This stratification was helped to analyze its association with biomarkers such as CAR, CRP, and albumin and to evaluate their predictive utility for adverse outcomes.

Table 1. Sociodemographic characteristics and comorbid status of study participants*

Variables	Results n (%) or mean±SD
Age (in years)	
21–30	2 (2.2)
31–40	18 (19.4)
41–50	44 (47.3)
51–60	29 (31.2)
Mean±SD	46.20±7.938
Gender	
Male	56 (60.2)
Female	37 (39.8)
Smoking	
Yes	31 (33.3)
No	62 (66.7)
Systemic hypertension	
Yes	87 (93.5)
No	6 (6.5)
Diabetes	
Yes	86 (92.5)
No	7 (7.5)

* SD – standard deviation, n (%) = frequency, and percentages are presented in brackets

Statistical analysis

The data were entered into MS Excel (Ver_2007) and the statistical data were analyzed using SPSS (IBM SPSS Statistics for Windows, Version 26.0, Armonk, NY, USA) software. The normality of the data was assessed using Kolmogorov-Smirnov and Shapiro-Wilks tests and the outliers were handled by exclusion. Descriptive statistics for categorical variables were expressed as frequencies and percentages and for continuous variables as the mean and standard deviation or median without interquartile range (IQR). As for inferential statistics, the chi-square test and one-way ANOVA was used to find the association between TB and CAR. Values of $p<0.05$ were statistically significant. Receiver operating characteristic (ROC) curve and area under curve (AUC) analysis were used to determine the best cutoff point for the diagnosis and resolution of symptoms while on treatment. For all tests, a two-sided p value ≤0.05 was considered statistically significant. Diagnostic values based on AUC were 0.9–1.0 excellent test; 0.8–0.9 good test; 0.7–0.8 fair test; 0.6–0.7 poor test and 0.5–0.6 fail.

Results

The sociodemographic characteristics and the comorbidity status of the study participants are presented in Table 1. The mean CAR among the study participants was 9.38±10.22 and the remaining clinical profiles are presented in Table 2. Among study participants, 32 patients (34.4%) had STEMI, and the remaining 61 patients (65.6%) had NSTEMI in the ECG findings. TB was found among the study participants where 84 patients (90.3%) had low TB, while the remaining nine patients (9.7%) had a high level of TB (Fig. 1).

Table 2. Clinical profile of the study participants with ACS*

Variables	Mean±SD
C-reactive protein (mg/L)	24.77±20.38
Albumin (g/dL)	3.21±0.61
Troponin I (ng/L)	8808.71±13290.78
Low density lipoprotein (mg/dL)	139.86±36.36
High density lipoprotein (mg/dL)	33.16±5.19

* ACS – acute coronary syndrome, SD – standard deviation

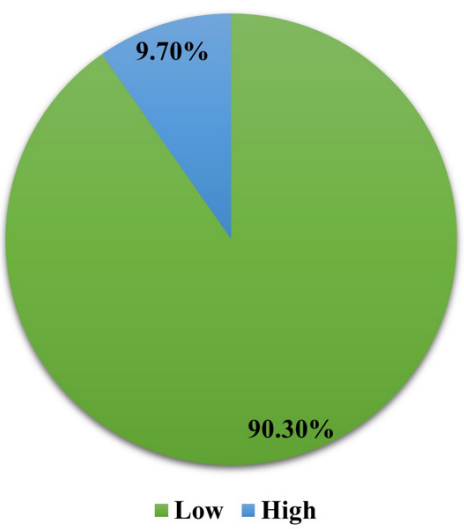

Fig. 1. Thrombus burden among the study participants

Table 3 shows the association of tuberculosis with other findings among study participants. Although the association between TB and ECG findings showed a significant relationship (p = 0.007) showing that the higher TB were more commonly associated with NSTEMI, which is typically associated with partial coronary occlusion, while STEMI often related to competitive occlusion, is mor frequently seen in patients with low TB. The

association between TB and CAR shows a statistically significant relationship, with a p-value of 0.001, indicating that higher TB is strongly associated with elevated CAR suggesting increased inflammation and poor nutritional status in patients with ACS patients (Table 4). Similarly, the association of CAR with other variables did not show a statistically significant result (Table 5).

Table 3. Association between thrombus burden and other variables among study participants*

Thrombus burden	Thrombus burden		p
	Low (n=84) n (%)	High (n=9) n (%)	
Gender			
Male	50 (59.5)	6 (66.7)	0.681
Female	34 (40.5)	3 (33.3)	
CAG TIMI grading			
Grade I	0 (0)	9 (100.0)	0.001
Grade II	25 (29.8)	0 (0)	
Grade III	59 (70.2)	0 (0)	
ECG findings			
STEMI	25 (29.7)	7 (77.8)	0.007
NSTEMI	59 (70.2)	2 (22.2)	

*chi-square test, CAG – coronary angiography, TIMI – thrombolysis in myocardial infarction, ECG – electrocardiography, STEMI – ST-elevated myocardial infarction, NSTEMI – non-ST-elevated myocardial infarction, n (%) – frequency and percentages were presented in brackets

Table 4. Association between thrombus burden and clinical profile among study participants

Variable	F value	p
CAR	102.369	0.001
Troponin I	0.345	0.55
CRP	76.824	0.001
Albumin	28.031	0.001

* ANOVA, CAR – C-reactive protein to albumin ratio, CRP – C-reactive protein

Table 5. Association between CAR and other variables among study participants*

Variables	F value	p
Troponin I	0.523	0.97
Gender	1.255	0.28
ECG	1.397	0.19
Age	1.227	0.30
Smoking	1.063	0.45
Diabetes	1.140	0.38

* ANOVA, ECG – electrocardiography

The ROC curve illustrates the diagnostic performance of CRP, albumin, and CAR in predicting TB, demonstrating the highest diagnostic accuracy (Fig. 2). Among the CAR markers, the curve is closest to the top-left corner, indicating superior sensitivity and specificity, making it the most reliable predictor. It emerges as the most effective parameter for distinguishing results, likely linked to its robust ability to reflect the balance of inflammation and nutritional status. Table 6 shows the AUC for TB with CRP, CAR, albumin and troponin I.

Table 6. AUC for CRP, albumin, CAR, troponin I with TB among study participants*

Test result variables	Area	Standard error	p	Asymptotic 95% confidence interval	
				Lower bound	Upper bound
CRP	0.987	0.014	<0.001	0.960	1.000
Albumin	0.030	0.019	<0.001	0.000	0.067
CAR	1.000	0.000	<0.001	1.000	1.000
Troponin-I	0.425	0.087	0.459	0.253	0.596

*AUC – area under curve, CRP – C-reactive protein, CAR – C-reactive protein to albumin ratio

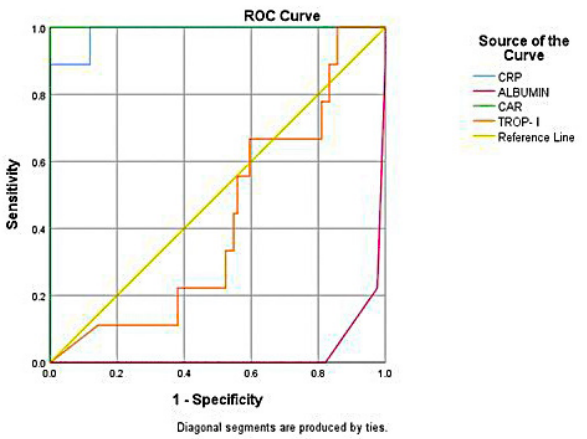

Fig. 2. Analysis of the ROC curve of TB with CAR, CRP, albumin, and troponin I

Discussion

The present study evaluated the significance of CAR as a predictor of TB in patients with ACS. The findings provide valuable information on the relationship between inflammatory markers, TB, and clinical outcomes in ACS. The study found significant associations between TB and CAR, CRP, albumin, TIMI grade, and ECG findings. CAR, with an AUC of 1.000, demonstrated excellent discriminatory power in predicting TB, outperforming individual biomarkers such as CRP and albumin. A strong positive correlation of tuberculosis was observed with CAR (r=0.728, p=0.001) and CRP (r=0.677, p=0.001) and a significant inverse correlation with albumin (r=-0.485; p=0.001). These findings suggest that CAR is not only a powerful prognostic biomarker in ACS but also reflects the systemic inflammatory state and the potential for thrombus formation.

TB plays an important role in the pathophysiology of ACS, contributing to vessel occlusion and myocardial damage.[5,10,25] The high rates of HTN and DM in

our study reflect their strong role as major risk factors in the pathogenesis of ACS. These comorbidities contribute to endothelial dysfunction and atherosclerosis, which are central to thrombus formation and coronary events. In addition, the hospital-based setting can capture more high-risk patients. The TB distribution shows that the majority of study participants (90.3%) had TB, while only 9.7% had a high TB. This suggests that most of the individuals in this cohort had less extensive coronary thrombus formation, which may correlate with less severe cases of ACS. The study by Souteyrand et al. also resulted that 11.8% of the patients had large TB, while remaining 88.2% had low TB, which was similar to our study.[26] Another study by Özkan et al., also resulted that 16.4% had massive TB.[27] These results align with prior studies indicating that inflammation plays a pivotal role in plaque instability, thrombus formation, and adverse outcomes in ACS.[28–30]

TIMI classification, a critical tool in assessing thrombus severity, aligns with the pathophysiological impact of high TB in worsening ischemia and altering coronary flow.[24] In our present study, TB assessed using the TIMI classification system during CAG, all patients with high TB were classified as TIMI grade I (100%), while those with low TB were predominantly in TIMI grade III (70.2%) and grade II (29.8%) (p=0.001) indicating a strong association between lower TIMI flow grades and higher TB. In our study, this significant association of TB with the CAG TIMI grade further reinforces its pathophysiological relevance in ACS events. This finding reflects the extent of luminal obstruction and impaired perfusion due to intraluminal thrombi, which has been associated with a worse prognosis in patients with ACS.[31–33] Further, a Pearson correlation analysis revealed a very strong association between TB and CAG TIMI flow grade (r=0.755, p=0.001) that highlights the diagnostic value of angiographic assessment in identifying patients with a high thrombotic load. The study by Kume et al. resulted that patients with high TIMI had higher TB compared to the patients with low TB.[34] These findings are consistent with previous literature that emphasized the clinical importance of quantifying TB for prognostication and to guide interventional strategies, including the use of thrombectomy or powerful antithrombotic therapy.[31–33] Thus, accurate evaluation of thrombuses is essential for optimizing ACS and improving patient outcomes.

The ECG itself does not directly quantify the thrombus load; it provides valuable indirect evidence of ischemic load and coronary obstruction. In our study, 65.6% presented with NSTEMI, while 34.4% of the patients had STEMI. This predominance of NSTEMI reflects the evolving trends in ACS presentations, where NSTEMI is increasingly recognized due to advances in diagnostic tools and heightened clinical vigilance.[27,35,36] The higher proportion of NSTEMI cases may suggest a more chronic atherosclerotic process with less abrupt plaque rupture.[36,37] A significant association was observed between TB and ECG findings in our study. Participants with low TB were predominantly diagnosed with NSTEMI, while those with high TB were more likely to have STEMI (p=0.007) in our patients. Furthermore, the correlation showed a modest but significant inverse relationship between the ECG findings and TB (r=-0.299, p=0.004) indicating that as TB increases, the probability of presenting with STEMI also increased. The study by Scarparo et al. showed that patients with high TB had STEMI with higher mortality compared to low TB in patients with NSTEMI.[25] Study by Coşkun et al., found that patients with high TB were higher in STEMI.[38] Scarparo et al. also showed that higher TB observed among the STEMI patients with increased 30-day mortality and 3-day MACE.[25] This finding highlights the interaction between thrombus composition, degree of coronary occlusion, and the severity of the infarction.[6,26] STEMI, characterized by complete occlusion of a major coronary artery, is typically associated with higher TB, reflecting its direct impact on myocardial perfusion, in contrast, NSTEMI, often involving partial occlusion, corresponds to a lower TB.[6,8,25,27] Thus, supporting that thrombus rich lesions are more common in total occlusions characteristics of STEMI events.

Both CRP and albumin, individually and in a combined ratio, are important for evaluating the inflammatory and nutritional status of patients with ACS.[15,17,21,23,29] The mean level of CRP and albumin among ACS patients in our study was 24.77±20.38 mg/L and 3.21±0.612 g/dL and CRP showed a strong positive correlation with TB (r=0.677, p=0.001),while albumin showed an inverse correlation (r=-0.485, p=0.001). Both were significantly associated with TB (p=0.001 for both). Furthermore, CRP exhibited a high predictive value for TB (AUC=0.987), while albumin alone had a very low discriminatory power (ACU=0.030), suggesting that albumin alone is not as reliable for thrombus risk. Elevated CRP levels in patients with ACS are associated with worse outcomes and have been shown to promote endothelial dysfunction, upregulated tissue factor and enhance platelet activation.[15,39,40] Conversely, albumin function as a negative acute phase protein with antioxidative and anti-inflammatory response are associated with increased cardiovascular mortality.[21,41–43] Together, CRP and albumin provide the complementary insights into the pathophysiological processes of ACS, where CRP reflects the inflammation and thrombotic activation, and albumin reflects systemic resilience and repair mechanisms. This interplay justifies the rationale behind using the CAR for enhanced prognostic accuracy in ACS management.

CAR has recently emerged as a powerful biomarker that reflects both inflammatory status and nutrition-

al reserve. In the findings of our study, the mean CAR was 9.38±10.222, highlights a significant inflammatory burden relative to nutritional status. CAR also showed a highly significant association with tuberculosis (F=102.369, p=0.001) and demonstrated the strongest correlation among the biomarkers evaluated (r=0.728, p=0.001). ROC analysis revealed an AUC of 1.000 for CAR, indicating perfect discriminatory power in the identification of patients with high RB, far surpassing individual predictive capacities. CAR makes it a superior marker for risk stratification in ACS, as elevated levels indicate a pro-inflammatory, prothrombotic state, closely linked with plaque rupture, endothelial injury, and intraluminal thrombus formation.[44,45] The study by Askin et al., showed that CRP or albumin levels had lower sensitivity and specificity when compared to CAR had higher specificity, but lower sensitivity.[46] Thus, CAR with its high diagnostic accuracy showed that it has potential to predict the TB in ACS patients, yet with intracoronary TB at high risk, had difficulty to predict by CAR.[46] Unlike single biomarkers, CAR integrates two opposing biological process, systemic inflammation and nutritional status, providing a more comprehensive view of disease burden. As an inflammatory marker of CRP and albumin alone has less diagnostic accuracy than compared to the combined marker.[46] Previous studies have validated CAR as a predictor of mortality and adverse cardiac events in patients with ACS.[39,41–43,45,47] The findings in our study strongly support its routine inclusion in clinical practice as a cost-effective, accessible marker to evaluate thrombotic risk and guiding therapeutic decisions in ACS.

The major strengths include that integrating CAR into existing risk scores which is TIMI that improved the calibration, and that even propose the prospective validation in the multicentre cohorts. Despite these encouraging findings, several limitations warrant careful consideration. Firstly, it was conducted in a single-centric study that poses constraints, which may limit the generalizability of the findings to broader populations. In addition, referral bias in a tertiary care setting could have inflated the prevalence of hypertension and diabetes and thus the observed biomarker associations. Second, the cross-sectional design precludes the establishment of causality between inflammatory markers, tuberculosis, and clinical outcomes. Additionally, the lack of long-term follow-up limits the ability to assess the predictive value of CAR for adverse cardiovascular events. Also, potential confounders including medications and time to angiography, were not captured. The grade 3 TB required adjudication, introducing potential classification bias despite blind dual review. Finally, we focused solely on CRP and albumin, hence the incorporation of additional inflammatory or nutritional biomarkers and the advancement of imaging techniques can further refine risk stratification.

To address these gaps, future multicentre prospective studies with larger, more diverse populations are essential. Longitudinal follow-ups must be incorporated to validate the prognostic significance of CAR for hard endpoints, such as reinfarction, heart failure, and mortality. Randomized trials could evaluate whether CAR-guided therapeutic intensification improves outcomes. Further studies could also explore the integration of CAR with other biomarkers and imaging modalities for comprehensive evaluation of thrombus risk in ACS patients.

Conclusion

Therefore, this study underscores the clinical relevance of integrating biochemical and angiographic markers, specifically CAR and stratification of TB in the evaluation and risk of patients with ACS. Our findings demonstrate a significant association between elevated CAR and increased TB, suggesting a direct relationship between systemic inflammation, impaired nutritional status, and the extent of coronary thrombosis. The findings advocate for the inclusion of CAR in routine clinical assessment due to its simplicity, cost-effectiveness, and diagnostic power.

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Declarations

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Author contributions

Conceptualization, V.R.; Methodology, V.R..; Software, P.V.R.I.; Validation, V.R.; Formal Analysis, P.V.R.I.; Investigation, P.V.R.I.; Resources, P.V.R.I.; Data Curation, P.V.R.I.; Writing – Original Draft Preparation, P.V.R.I.; Writing – Review & Editing, V.R.; Visualization, V.R.; Supervision, V.R.; Project Administration, V.R.

Conflicts of interest

The author(s) reported no potential conflicts of interest regarding the research, authorship, or publication of this article.

Data availability

All data generated or analyzed in this study are included in this published article.

Ethics approval

The Institutional Ethical Committee (MGMCRI/Res/04/2022/121) approved this study.

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