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C-REACTIVE PROTEIN-TO-ALBUMIN RATIO AND ITS ASSOCIATION WITH STROKE AMONG HYPERTENSIVE PATIENTS IN SOUTH-EASTERN NIGERIA: A PILOT CROSS-SECTIONAL STUDY

Ofordum CJ[1], Igbokwe EG[1], Ibezim HU[2,3], Ubaoji IK[1], Nwangwu CS[3], Ekuma TE[4]

Department of Applied Biochemistry, Nnamdi Azikiwe University, Awka[1]
Department of Biochemistry, Igbinedion University, Okada[2]
Department of Internal Medicine, Igbinedion University Teaching Hospital, Okada[3]
Department of Science Laboratory Technology, Akanu Ibiam Federal Polytechnic, Unwana[4]

ABSTRACT

BACKGROUND:

Chronic inflammation is central to the development of hypertension and its complications, including stroke. The C-reactive protein-to-albumin ratio (CAR), which combines a positive and a negative acute-phase reactant, has emerged as a potential marker of systemic inflammatory burden. However, evidence from sub-Saharan Africa remains limited. This study aimed to evaluate the CAR among hypertensive patients and assess its association with stroke status in a South-Eastern Nigerian population.

METHODOLOGY:

This pilot case–control study included 20 participants, comprising 10 hypertensive patients and 10 apparently healthy normotensive controls. Serum albumin was measured using the bromocresol green method, while C-reactive protein was determined by an immunoenzymometric assay. CAR was calculated as the ratio of CRP to albumin. Blood pressure was measured using standard protocols and stroke status was determined and supported by clinical findings and neuroimaging reports. Data

RESULTS

Hypertensive patients had significantly lower serum albumin levels and higher CRP concentrations compared with controls ($p = 0.004$). CAR values were significantly higher among hypertensive participants (range: 1.077–4.667) compared with controls (range: 0.265–0.842) ($p = 0.023$). A strong positive association was observed between CAR and stroke status (tau-b = 0.618, $p = 0.001$).

CONCLUSION

CAR is elevated among hypertensive patients and is significantly associated with stroke status, suggesting its potential role as a simple and cost-effective prognostic inflammatory marker. Larger longitudinal studies are needed to validate these findings.

KEYWORDS

C-reactive protein; albumin; C-reactive protein to albumin ratio; hypertension; stroke; inflammation

CORRESPONDING AUTHOR

Ibezim Harmony Uchenna
Department of Biochemistry,
Igbinedion University, Okada
Department of Internal Medicine,
Igbinedion University Teaching Hospital, Okada
ibezim.harmony@iuokada.edu.ng
+2347066377286

INTRODUCTION:

Stroke remains one of the leading causes of mortality and long-term disability worldwide and represents a growing public health challenge, particularly in low- and middle-income countries [1]. In sub-Saharan Africa, the burden of stroke has risen substantially over the past two decades,

driven largely by the increasing prevalence of hypertension, population ageing, and limited access to preventive healthcare services [2]. Hypertension is the most important modifiable risk factor for stroke and contributes significantly to both ischaemic and haemorrhagic cerebrovascular events [3]. Despite advances in clinical management, stroke-related morbidity and mortality remain disproportionately high in resource-limited settings such as Nigeria [4]. Beyond elevated blood pressure, accumulating evidence suggests that systemic inflammation plays a central role in the pathophysiology of hypertension and stroke. Chronic inflammatory processes contribute to endothelial dysfunction, vascular remodelling, atherosclerosis, and thrombogenesis, thereby increasing cerebrovascular vulnerability [5,6]. Although several inflammatory biomarkers including E-selectin, fibrinogen, homocysteine, and lipoprotein(a) have demonstrated predictive value in hypertension and cardiovascular disease, CRP was selected in this study because of its widespread availability, relatively low assay cost, and established clinical relevance in cardiovascular risk stratification. [7,43]. CRP is also incorporated into validated cardiovascular risk prediction models such as the Reynolds Risk Score, further supporting its translational utility in clinical practice [44]. In resource-limited settings such as sub-Saharan Africa, biomarkers that are inexpensive, reproducible, and routinely available are particularly valuable for large-scale cardiovascular risk assessment [45].

C-reactive protein (CRP) is a well-established acute-phase reactant that rises in response to inflammation and tissue injury and has been associated with an increased risk of stroke as well as poorer outcomes following cerebrovascular events [8,9]. Conversely, serum albumin, a negative acute-phase protein, tends to decrease during inflammatory states and reflects both nutritional status and systemic illness severity [10]. Individually, both CRP and albumin have been linked to stroke risk and outcomes; however, their combined assessment may provide a more robust reflection of the inflammatory milieu [11].

The C-reactive protein–to–albumin ratio (CAR) has emerged as a novel composite biomarker that integrates inflammatory burden and physiological reserve. Elevated CAR has been associated with adverse outcomes in various clinical conditions, including acute ischaemic stroke [12,13]. Several studies have demonstrated that higher CAR values correlate with stroke severity, functional disability, and mortality, suggesting its potential value in clinical risk assessment [14]. By capturing both heightened inflammation and reduced albumin levels, CAR may offer superior prognostic insight compared with either biomarker alone [15].
Despite growing evidence supporting the clinical relevance of CAR, data from African populations remain scarce, and its utility among hypertensive patients in sub-Saharan Africa has not been well explored [16]. Given the high prevalence of hypertension and the rising burden of stroke in Nigeria, there is a pressing need to identify simple, affordable biomarkers that can aid in early risk identification and clinical decision-making [17]. CAR is particularly attractive in this context because CRP and albumin are routinely available laboratory parameters in most healthcare settings [18].

Therefore, this pilot cross-sectional study was designed to evaluate the association between the C-reactive protein–to–albumin ratio and stroke among hypertensive patients in South-Eastern Nigeria. By generating preliminary data on CAR levels in hypertensive patients with and without stroke, and comparing these findings with those of apparently healthy controls, this study seeks to contribute to the limited regional evidence base and provide a foundation for larger, prospective investigations.

METHODS

Study Design and Setting

This study was a pilot comparative cross-sectional study conducted at Ifebi Medical Center and Regina Specialist Hospital, Awka, Anambra State, located in the South-Eastern geopolitical zone of Nigeria. The study was designed to generate preliminary data on the association between systemic inflammation, assessed using the C-reactive protein–to–albumin ratio (CAR), and stroke among hypertensive patients in a resource-limited setting.

Study Population

A total of 20 adult participants were recruited in this pilot exploratory study, using convenience sampling in order to generate preliminary data and assess feasibility within a limited study period and resource-constrained setting.

The approach enabled the recruitment of readily available eligible participants and is consistent with methodologies commonly used in pilot biomedical research [46-48]. The study population comprised 10 hypertensive patients, some of whom had a documented history of stroke, and 10 apparently healthy controls without known chronic medical conditions. Hypertensive patients were recruited from the Medical Outpatient Clinic of Ifebi Medical Center, while control participants and laboratory analyses were obtained from Regina Specialist Hospital.

Eligibility Criteria

Inclusion Criteria

Adults aged 18 years and above who provided written informed consent were eligible for inclusion. Participants in the hypertensive group had a confirmed diagnosis of hypertension based on the Eighth Joint National Committee (JNC 8) and World Health Organization/International Society of Hypertension (WHO/ISH) guidelines [19,20]. Control participants were adults without a prior diagnosis of hypertension, stroke, diabetes mellitus, or other chronic inflammatory conditions.

Exclusion Criteria

Although participants receiving steroid therapy and other potent anti-inflammatory medications were excluded, hypertensive participants receiving routine antihypertensive medications such as ACE inhibitors or angiotensin receptor blockers (ARBs), as well as statins, were not excluded. This was because these medications form part of standard hypertension management in routine clinical practice. However, their potential anti-inflammatory effects may have influenced inflammatory biomarker levels and should be considered when interpreting the findings. [21].

Stroke Ascertainment

Stroke status among hypertensive patients was determined from clinical records, based on documented physician diagnosis supported by relevant clinical findings and neuroimaging reports where available. A stroke was defined in accordance with the World Health Organisation criteria as a rapidly developing clinical sign of focal or global disturbance of cerebral function lasting more than 24 hours with no apparent non-vascular cause [22].

Sample Size Justification

As this was a pilot exploratory study, a formal power-based sample size calculation was not performed. A total sample size of 20 participants was selected as an estimated 10% of the projected sample size for a future large-scale definitive study. This approach is consistent with recommendations for pilot biomedical research, where pilot samples are used primarily to assess feasibility, generate preliminary estimates, and guide subsequent power calculations [23,46–48].

Blood Pressure Measurement

Blood pressure was measured using a calibrated analogue mercury sphygmomanometer with participants seated and at rest for at least five minutes. Two measurements were taken at each visit, and the average was recorded. Hypertension was confirmed based on systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg, measured on at least two separate occasions, in accordance with JNC 8 and WHO/ISH guidelines [19,20].

Sample Collection and Processing

Venous blood samples (5 mL) were collected from each participant using a new disposable sterile syringe and transferred into plain sample bottles. The samples were centrifuged at 2,000 rpm for 10 minutes using a bench centrifuge (Eppendorf 5702, Germany). Serum was carefully separated using an adjustable micropipette (Eppendorf Research Plus, Germany) and transferred into labelled serum storage bottles. The serum samples were stored at 4°C in a laboratory refrigerator (Haier HYC-610, China) and analysed within the recommended storage period to preserve sample integrity [24].

Determination of Serum Albumin

Serum albumin concentration was determined using the bromocresol green (BCG) colorimetric method. Separate test tubes were prepared for test samples, controls, standards, and reagent blanks. A volume of 3.0 mL (3,000 µL) of bromocresol green reagent (Randox Laboratories, UK) was dispensed into each tube. Subsequently, 0.01 mL (10 µL) of serum sample, control, or standard was added to the appropriate tube using a precision micropipette.

The reaction mixtures were vortexed immediately using a vortex mixer (IKA MS 3 Basic, Germany) and incubated at room temperature (20–27°C) for 5 minutes. Absorbance was measured at a wavelength of 640 nm using a UV–visible spectrophotometer (Thermo Scientific Evolution 201, USA) against the reagent blank [25,26].

Serum albumin concentration was calculated using the formula:

$$\text{Concentration of Albumin} = \frac{\text{Absorbance of Standard} \times \text{Concentration of Standard}}{\text{Absorbance of Test}}$$

Determination of Serum C-Reactive Protein

Serum C-reactive protein (CRP) concentration was measured using a sandwich immunoenzymometric assay (ELISA) based on high-affinity monoclonal antibodies specific for human CRP. The assay utilised streptavidin-coated microplate wells, allowing immobilisation through the interaction between streptavidin and a biotinylated monoclonal anti-CRP antibody.

Upon addition of biotinylated antibody, enzyme-labelled antibody, and serum-containing antigen, a soluble antibody–antigen–antibody sandwich complex was formed without competition or steric hindrance [27,28].

Reagents and Equipment

The assay kit components included biotin-labelled monoclonal anti-CRP antibody with horseradish peroxidase (HRP), streptavidin-coated 96-well microplates, serum diluent, wash buffer concentrate, tetramethylbenzidine (TMB) substrate solution, hydrogen peroxide substrate, and stop solution (1N hydrochloric acid). All reagents were stored at 2–8°C before use.

Absorbance was read using a microplate reader (BioTek Epoch, USA) at 450 nm. Sample handling was performed using multichannel micropipettes (Eppendorf Research Plus, Germany), and incubations were carried out at room temperature (20–27°C).

Assay Procedure

All reagents, controls, and serum samples were allowed to equilibrate to room temperature prior to analysis. A volume of 25 µL of serum sample, control, or reference was dispensed into the debeforesignated microplate wells, followed by the addition of 100 µL of CRP enzyme conjugate. The plate was gently mixed and incubated for 15 minutes at room temperature.

Following incubation, the contents of the wells were discarded, and each well was washed three times with 350 µL of diluted wash buffer using an automated plate washer. Subsequently, 100 µL of substrate solution was added to each well and incubated for 15 minutes at room temperature in the dark. The enzymatic reaction was terminated by adding 50 µL of stop solution, and absorbance was read immediately at 450 nm using the microplate reader [29].

The CRP ELISA kit used was an in vitro diagnostic assay validated for the quantitative measurement of human CRP in serum samples [30].

Calculation of C-Reactive Protein–to–Albumin Ratio

The C-reactive protein–to–albumin ratio (CAR) was calculated for each participant by dividing the measured serum CRP concentration by the corresponding serum albumin concentration. CAR was used as a composite biomarker reflecting systemic inflammation and nutritional status. [31]

Statistical Analysis

Data analysis was performed using IBM SPSS Statistics version 26. Continuous variables were summarised using means and standard deviations or medians as appropriate, while categorical variables were presented as frequencies and percentages. Due to the small sample size and non-normal distribution of CAR values, non-parametric statistical tests were employed. The association between CAR and stroke status was assessed using Kendall's Tau-b correlation coefficient. Statistical significance was set at a p-value of <0.05.

Ethical Considerations

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Ethical approval was obtained from the Research and Ethics Committees of Ifebi Medical Center and Regina Specialist Hospital, Awka. Written

informed consent was obtained from all participants before enrolment [28].

RESULTS

Sociodemographic Characteristics of Study Participants

A total of 20 participants were enrolled in this pilot study, comprising 10 hypertensive patients and 10 apparently healthy normotensive controls.

The hypertensive group had a significantly higher mean age compared with controls (61.0 ± 9.7 years vs 30.7 ± 6.1 years). Among hypertensive participants, 70% were male (n = 7) and 30% were female (n = 3), while the control group consisted of 40% males (n = 4) and 60% females (n = 6).

Baseline demographic characteristics are summarised in Table 1 below.

Table 1: Sociodemographic Characteristics of Study Participants

Characteristics	Hypertensive Patients (Cases, n = 10)	Controls (n = 10)
Age (years)	61 ± 9.7 (46–75)	30.7 ± 6.1 (21–42)
Sex – Male	7 (70%)	4 (40%)
Sex – Female	3 (30%)	6 (60%)

Notes: Values are presented as mean ± SD (range) for age and number (%) for sex.

Serum Albumin, C-Reactive Protein Levels and C-Reactive Protein to Albumin Ratio (CAR)

Mean serum albumin concentration was markedly lower among hypertensive patients compared with controls. Albumin values in hypertensive patients ranged from 13.6 to 36.2 g/L, while control participants demonstrated higher albumin concentrations ranging from 34 to 54 g/L. Conversely, serum C-reactive protein (CRP) levels were higher among hypertensive patients, with values ranging from 3.0 to 6.8 µg/mL, compared with 0.9 to 3.2 µg/mL in the control group, indicating a heightened inflammatory state among hypertensive subjects (P = 0.004).

The C-reactive protein to albumin ratio (CAR) was consistently higher in hypertensive patients compared with normotensive controls (P = 0.023). Among hypertensive participants, CAR values ranged from 1.077 to 4.264, whereas control participants had markedly lower CAR values ranging from 0.265 to 0.842.

These findings demonstrate a clear separation in inflammatory burden between hypertensive patients and controls, as illustrated in Tables 2 and 3 below.

Table 2: Blood Pressure, Stroke Status, Serum Albumin, CRP, and CAR in Hypertensive Patients (Cases)

Patient ID	Sex	Age (yrs)	Blood Pressure (mmHg)	Stroke Status	Albumin (g/L)	CRP (µg/mL)	CAR (×10)
H1	M	68	180/90	Negative	22.1	3.0	1.357
H2	M	60	170/70	Negative	23.6	6.8	2.881
H3	F	58	140/90	Negative	13.6	5.8	4.264
H4	M	46	180/100	Negative	36.2	3.9	1.077
H5	M	55	160/100	Negative	29.3	3.8	1.296
H6	M	60	160/100	Negative	23.0	4.7	2.043
H7	F	65	140/80	Positive	10.5	4.9	4.667
H8	M	75	150/90	Positive	12.0	4.8	4.000
H9	F	74	140/100	Positive	12.5	5.5	4.400
H10	M	49	190/100	Negative	31.8	2.5	0.785

Table 3: Blood Pressure, Stroke Status, Serum Albumin, CRP, and CAR in Controls

Patient ID	Sex	Age (yrs)	Blood Pressure (mmHg)	Stroke Status	Albumin (g/L)	CRP (µg/mL)	CAR (×10)
C1	F	25	120/80	Negative	36	1.2	0.333
C2	F	27	130/70	Negative	34	0.9	0.265
C3	F	35	140/90	Negative	38	3.2	0.842
C4	F	28	130/87	Negative	41	2.9	0.707
C5	M	36	120/90	Negative	52	3.0	0.577
C6	M	42	120/80	Negative	45	2.8	0.622
C7	F	31	130/90	Negative	39	2.5	0.641
C8	M	29	120/80	Negative	54	1.5	0.277
C9	M	33	120/80	Negative	38	1.8	0.474
C10	F	21	120/90	Negative	48	1.6	0.333

Association Between CAR and Stroke Status

Correlation analysis using Kendall's tau-b revealed a strong positive association between CAR and stroke status ($\tau b = 0.618$, $p = 0.001$), indicating that higher CAR values were significantly associated with the presence of stroke.

Additionally, A significant association was observed between sex and CAR among hypertensive participants (Kendall's $\tau b = -0.683$, $p = 0.017$), with female hypertensive participants demonstrating notably higher CAR values (median = 4.40; range: 4.264–4.667) compared with their male counterparts (median = 1.36; range: 0.785–4.000). This difference was corroborated by Mann–Whitney U analysis (U = 0.000, p = 0.017).

Table 4: Association Between CAR, Stroke Status, and Sex

Variable 1	Variable 2	Kendall's Tau-b	p-value
Stroke Status	CAR	0.618	0.001
Sex	CAR	–0.683	0.017

Notes: Positive Tau-b indicates a direct association, negative indicates an inverse association.

DISCUSSION

This pilot study evaluated the C-reactive protein to albumin ratio (CAR) as an inflammatory marker among hypertensive patients, with particular emphasis on its association with stroke status.

The findings demonstrate that hypertensive patients had lower serum albumin levels, higher CRP concentrations, and consequently significantly elevated CAR values compared with apparently healthy normotensive controls. Furthermore, CAR showed a strong positive association with stroke, suggesting its potential utility as a prognostic inflammatory marker in hypertensive individuals.

Inflammatory Burden and Hypertension

Chronic low-grade inflammation plays a central role in the pathogenesis of hypertension and its vascular complications. Elevated CRP reflects systemic inflammation and endothelial dysfunction, while hypoalbuminaemia is increasingly recognised as a negative acute-phase response associated with poor clinical outcomes. The observed increase in CRP and reduction in albumin among hypertensive patients in this study are consistent with previous reports linking inflammatory dysregulation to hypertension-related target organ damage [32–34].

CAR as an Integrated Inflammatory Marker

The C-reactive protein to albumin ratio integrates both positive and negative acute-phase reactants, providing a more stable and comprehensive indicator of systemic inflammation than either parameter alone. In this study, CAR was markedly higher among hypertensive patients, particularly those with stroke, highlighting its potential role as a sensitive marker of inflammatory burden. Similar findings have been reported in cardiovascular and cerebrovascular diseases, where elevated CAR was associated with disease severity and adverse outcomes [35–37].

Association Between CAR and Stroke

A key finding of this study is the strong positive association between CAR and stroke status. Stroke is increasingly recognised as an inflammatory-mediated vascular event, particularly in individuals with long-standing hypertension. Elevated CAR may reflect enhanced cytokine-driven inflammation, endothelial injury, and impaired vascular integrity, all of which contribute to cerebrovascular events. Previous studies have demonstrated that higher CAR values are associated with increased stroke severity, poorer functional outcomes, and higher mortality rates, lending support to the present findings [38–40].

Sex Differences in CAR

An exploratory analysis suggested that female hypertensive participants had higher CAR values than males, a finding that reached statistical significance despite the small sample ($U = 0.000$, $p = 0.017$). This observation may reflect the disproportionate representation of female participants among those with stroke and severely low albumin levels within the hypertensive group, rather than a true sex-specific difference in inflammatory burden. Sex-based variations in inflammatory biomarkers have been reported in cardiovascular and cerebrovascular populations, with some evidence suggesting that hormonal factors and distinct vascular ageing patterns may modulate CRP and albumin levels differently in men and women [41,42].

Clinical Implications

CAR is derived from routinely available laboratory parameters, making it a cost-effective, accessible, and practical biomarker, particularly in resource-limited settings such as sub-Saharan Africa. Its potential use as a prognostic tool in hypertensive patients may aid in early risk stratification, improved monitoring, and timely intervention to prevent stroke and other cardiovascular complications.

Study Limitations

This study has several limitations. The small sample size and cross-sectional design limit the generalisability of the findings and preclude causal inference. The observed age difference between hypertensive patients and controls may also have influenced inflammatory markers. Additionally, stroke severity scores and long-term outcomes were not assessed. Also, with only three female hypertensive participants, the statistical power for this comparison is severely limited, and the observed association is highly susceptible to confounding by stroke status and disease severity. This finding should not be interpreted as a reliable indicator of sex differences in systemic inflammation among hypertensive patients. Future studies with larger, sex-balanced samples are required to adequately examine this relationship. Nevertheless, as a pilot study, the findings provide valuable preliminary evidence to justify larger, longitudinal studies.

Future Directions

Future research with larger sample sizes, age-matched controls, and longitudinal follow-up is needed to validate the prognostic utility of CAR in hypertensive populations. Incorporation of stroke severity indices and functional outcomes would further strengthen the clinical relevance of CAR.

Conclusion

This pilot study demonstrates that the C-reactive protein to albumin ratio (CAR) is significantly elevated among hypertensive patients compared with apparently healthy normotensive individuals and shows a strong association with stroke status. The observed increase in CAR reflects the combined effect of heightened systemic inflammation and reduced serum albumin levels in hypertensive patients, particularly those with cerebrovascular complications. Given that CAR is derived from routinely available laboratory parameters, it represents a simple, cost-effective, and accessible inflammatory marker that may have prognostic value in the assessment of hypertensive patients. Its potential utility is especially relevant in resource-limited settings, where advanced inflammatory profiling may not be readily available.

However, due to the small sample size and cross-sectional design, these findings should be interpreted with caution. Larger, longitudinal studies are required to validate the prognostic role of CAR and to establish its usefulness in predicting stroke risk and outcomes among hypertensive populations.

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Conflict Of Interest Statement

The authors declare no conflict of Interest

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