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Anthropometric Adiposity and Systemic Inflammatory Markers and Response in Neurological Disease: An Exploratory Study of Cerebrovascular Ischaemic Events Associated to Carotid Atheromatous Processes

Andreea Nicoleta Tovârnac
Senior neurologist at Emergency county Hospital of Brăila, PhD student at „Dunărea de Jos" University of Galați, Romania. andreeatovarnac@yahoo.com

Eva-Maria Elkan*
Senior paediatric neurologist at Emergency County Hospital of Buzău, lecturer PhD at „Dunărea de Jos" University of Galați, Romania.
cojocarumariaeva@yahoo.com
https://orcid.org/0000-0003-2094-1089

Florin Tovârnac
Senior Gynecologist at Emergency County Hospital of Brăila, PhD student at University „Dunărea de Jos" University of Galați, Romania.
tovarnacf@yahoo.com

Diana Ionescu
Associate Professor PhD at „Titu Maiorescu" University of Bucharest, Romania.
diana.ionescu@prof.utm.ro

Lăcrămioara Ilie
Senior paediatrician at Târgu Bujor county Hospital, PhD student at University „Dunărea de Jos" University of Galați, Romania.
lacramioara.Ilie@ugal.ro
https://orcid.org/0000-0003-2285-9353

Carmen Laura Cristescu-Budală
Senior paediatrician at Emergency Clinical Hospital for Children „Saint John" PhD student at „Dunărea de Jos" University of Galați, Romania.
laura.cristescubudala@gmail.com
https://orcid.org/0009-0004-1577-1673

Gabriela Gurău
Professor habil. PhD at „Dunărea de Jos" University of Galați, Romania. gabriela.gurau@ugal.ro

** Corresponding author Eva Maria Elkan:e-mail:cojocarumariaeva@yahoo.com*

Abstract: *This exploratory study examined whether anthropometric adiposity and systemic inflammatory response represent convergent or partially distinct biological dimensions in neurological disease, and whether these characteristics differ according to cerebrovascular ischemic-event status. A retrospective observational analysis included 32 clinical monitoring observations from adults evaluated in a neurology department. Anthropometric indicators comprised body mass index (BMI), mid-upper arm circumference (MUAC), waist circumference, and waist-to-height ratio (WHtR). Inflammatory markers included erythrocyte sedimentation rate (ESR), leukocyte, neutrophil, lymphocyte counts, and neutrophil-to-lymphocyte ratio (NLR). Spearman correlations, Benjamini–Hochberg false-discovery-rate correction, Kruskal–Wallis and Mann–Whitney tests, Cliff's delta, and sensitivity analyses for extreme NLR values were applied. As results, excess body weight was common: 50.0% of observations were overweight and 37.5% obese. NLR was markedly right-skewed (median 3.20; IQR 2.35–4.57). None of the 20 anthropometric–inflammatory correlations remained significant after false-discovery-rate correction. BMI–NLR (ρ=-0.285, p=0.135) and waist circumference–NLR (ρ=-0.350, p=0.063) associations were inverse and nonsignificant. In contrast, strong within-domain correlations were observed, including BMI–WHtR (ρ=0.860) and leukocyte–neutrophil counts (ρ=0.901). Inflammatory markers did not differ significantly across BMI categories. Fourteen observations were classified as cerebrovascular ischemic events; neither anthropometric measures nor NLR significantly differentiated these from other neurological diseases. Sensitivity analyses excluding extreme NLR values did not materially change the findings. In conclusion, anthropometric adiposity and systemic inflammatory response showed strong internal but limited cross-domain coherence. In this small neurological cohort, NLR was not a direct surrogate of adiposity or an isolated discriminator of cerebrovascular ischemic events. These findings support multidimensional, context-dependent interpretation of adiposity and inflammation in neurological disease.*

Keywords: *obesity; anthropometric adiposity; systemic inflammation; neutrophil-to-lymphocyte ratio; ischemic stroke; cerebrovascular ischemic events.*

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1. Introduction

Obesity is increasingly understood not simply as an excess of body mass but as a heterogeneous metabolic and inflammatory condition in which the amount, distribution, and biological activity of adipose tissue may influence vascular and neurological health. Adipose tissue functions as an active endocrine and immunological organ, producing adipokines and inflammatory mediators capable of modifying insulin sensitivity, endothelial function, oxidative balance, and vascular homeostasis (Ruan & Lodish, 2004; Geng et al., 2023). This biological activity is particularly relevant to cerebrovascular disease because obesity frequently coexists with hypertension, dyslipidemia, insulin resistance, and other conditions that contribute to vascular injury. Large observational studies have consequently linked obesity and adiposity-related phenotypes to cerebrovascular disease and long-term neurological consequences (Jiang et al., 2010; Qin et al., 2015; Morys et al., 2021).

An important limitation of the conventional assessment of obesity is its frequent reliance on body mass index (BMI). Although BMI remains a practical and widely used indicator of overall body size, individuals with similar BMI values may differ considerably in body composition and regional fat distribution. Measures reflecting abdominal or central adiposity may therefore provide complementary information. Waist circumference, waist-to-height ratio (WHtR), and other body-shape or adiposity indices have increasingly been investigated alongside BMI because they may better capture metabolically relevant fat distribution in some populations. Consistent with this approach, studies examining body shape and fat depots have shown that adiposity-related measures do not necessarily provide equivalent information regarding cardiometabolic or cerebrovascular risk (Haberka et al., 2023; Meulmeester et al., 2023). In particular, waist circumference has shown predictive relevance for stroke in high cardiovascular-risk populations, supporting the evaluation of adiposity beyond BMI alone (Haberka et al., 2023).

The potential importance of fat distribution is biologically plausible because visceral adipose tissue has distinct inflammatory and metabolic properties. Experimental evidence indicates that visceral adipose tissue inflammation can contribute to systemic cytokine activation, blood-brain barrier alterations, and increased vulnerability to ischaemic cerebral injury (Shin et al., 2015). Similarly, experimental stroke models have shown that obesity modifies adipose tissue, metabolic, and inflammatory responses following cerebral ischaemia (Haley et al., 2017). These observations support a mechanistic connection between peripheral adipose tissue dysfunction and cerebral responses to ischaemic injury, although direct extrapolation from experimental models to clinically heterogeneous neurological populations remains difficult (Haley & Lawrence, 2016).

Systemic inflammation represents a second component of this relationship. Ischaemic brain injury triggers rapid interactions between the central nervous system and peripheral immune system, involving circulating leukocytes, neutrophils, lymphocytes, platelets, cytokines, and other inflammatory mediators. Neutrophils participate in early innate immune responses and may contribute to vascular and tissue injury, whereas lymphocyte dynamics reflect different components of adaptive immune regulation (Ansari & Gavins, 2021). Consequently, routinely available haematological measurements have increasingly been investigated as accessible indicators of systemic inflammatory activity in stroke and other neurological conditions. Recent literature continues to emphasise that post-stroke inflammation is systemic rather than exclusively cerebral and that its magnitude and temporal profile may be influenced by patients' metabolic characteristics (Abuzan et al., 2026).

Among these markers, the neutrophil-to-lymphocyte ratio (NLR) has attracted particular attention because it integrates two complementary leukocyte components into a simple index derived from routine complete blood counts. Elevated NLR has been associated with carotid atherosclerosis, vulnerable carotid plaque, stroke severity, complications, and adverse outcomes in several clinical settings (Li et al., 2021; Liu et al., 2025; Xu et al., 2025). Studies of acute ischaemic cerebrovascular disease have similarly examined NLR together with other cell-count-derived inflammatory ratios as potential prognostic or follow-up markers (Tekin et al., 2020). In critically ill

patients with cerebrovascular disease, higher NLR has also been associated with short-term mortality, further illustrating its potential relevance as a marker of systemic immune imbalance (Lin et al., 2024).

However, the relationship between NLR and adiposity is considerably less consistent than might be expected from the general inflammatory model of obesity. In a large study of patients with obesity and metabolic syndrome, Bahadir et al. (2015) found that leukocyte, neutrophil, and lymphocyte counts and high-sensitivity C-reactive protein were related to BMI, whereas NLR itself was neither significantly different across BMI categories nor correlated with BMI. These findings suggest that an increase in inflammatory cell counts accompanying obesity does not necessarily translate into a proportional increase in NLR.

Conversely, other studies have reported stronger relationships when adiposity is characterised more directly. Suárez-Cuenca et al. (2019) demonstrated that NLR was associated with high-sensitivity C-reactive protein and pro-inflammatory mediators and was also positively correlated with visceral adipose tissue area and adipocyte size in individuals with obesity. Li et al. (2020) subsequently showed that both NLR and the Chinese Visceral Adiposity Index were associated with carotid atherosclerosis and cardiovascular risk in older adults. Taken together, these findings suggest that the relationship between adiposity and systemic inflammation may depend on how adiposity is operationalised and may be more complex than a simple monotonic association between BMI and inflammatory markers.

This complexity becomes even more relevant in cerebrovascular disease. Obesity is an established contributor to vascular risk, yet its relationship with the biological response after ischaemic stroke is not straightforward. Studies addressing the so-called “obesity paradox” have reported that patients with obesity do not invariably experience worse post-stroke outcomes despite having a greater burden of conventional vascular risk factors. Rodríguez-Castro et al. (2019), for example, observed higher pro-inflammatory IL-6 concentrations in patients with obesity but no significant difference in functional outcome at three months, together with dynamic differences in pro- and anti-inflammatory mediators. These observations emphasise that obesity-related inflammation in stroke cannot be inferred solely from body size or from a single inflammatory pathway.

Human studies examining immune responses after stroke provide additional evidence of this heterogeneity. Ruhnau et al. (2023) reported BMI-related differences in post-stroke cytokine concentrations and leukocyte subpopulations, with higher BMI associated with changes consistent with a more pro-inflammatory immune phenotype. However, other investigations have identified nonlinear relationships between obesity-related variables and inflammatory mediators. Kim et al. (2023), for instance, reported U-shaped associations involving BMI, glycated albumin, and IL-10 in hyperacute ischaemic stroke. Such findings argue against assuming that increasing adiposity necessarily produces a uniform linear increase in systemic inflammatory activity.

The distinction between general and central adiposity may also be clinically relevant to cerebrovascular risk. Emerging composite approaches combining visceral adiposity and inflammatory information have been investigated for stroke-risk assessment (Xiao et al., 2025; Luwen et al., 2025). Prospective evidence has likewise examined the joint contribution of visceral adiposity indices and systemic inflammatory biomarkers to incident stroke (Xu et al., 2026). These approaches reflect a broader transition from treating obesity and inflammation as isolated risk factors toward evaluating whether their interaction provides additional information regarding vascular vulnerability.

Nevertheless, this developing literature remains fragmented. Some studies focus on BMI categories, others on visceral fat assessed by imaging or composite indices, while still others evaluate inflammatory biomarkers primarily as predictors of stroke severity or prognosis rather than as correlates of anthropometric adiposity. Moreover, much of the evidence concerns either metabolically characterised populations with obesity or cohorts restricted to confirmed ischaemic stroke. Relatively less attention has been directed toward the relationship between multiple simple

anthropometric indicators and routinely available systemic inflammatory markers within clinically heterogeneous neurological populations, where inflammatory activation may arise from several disease processes and where cerebrovascular ischaemia represents only one diagnostic component.

Three specific gaps therefore remain relevant. First, it is uncertain whether routinely available anthropometric measures representing different dimensions of adiposity — particularly BMI, mid-upper arm circumference (MUAC), waist circumference, and WHtR — show comparable relationships with systemic inflammatory markers. Second, although NLR is frequently used as a convenient inflammatory index, evidence linking NLR directly to anthropometric adiposity is inconsistent; positive associations reported with visceral adiposity contrast with studies showing little or no relationship with BMI (Bahadir et al., 2015; Suárez-Cuenca et al., 2019). Third, there is limited evidence establishing whether the adiposity-inflammation relationship observed in metabolic or cardiovascular cohorts can be identified within a mixed neurological population and whether either anthropometric or inflammatory characteristics distinguish patients with documented cerebrovascular ischaemic events from patients presenting with other neurological diseases.

The present study addresses these gaps through an integrated exploratory assessment rather than by treating obesity, inflammation, and neurological diagnosis as independent domains. Its novelty lies in the simultaneous evaluation of four complementary anthropometric indicators — BMI, MUAC, waist circumference, and WHtR — and five systemic inflammatory measures — erythrocyte sedimentation rate (ESR), total leukocyte count, neutrophil count, lymphocyte count, and NLR. This design permits comparison of overall and central anthropometric adiposity while also distinguishing individual leukocyte components from the derived NLR index.

A further contribution is the evaluation of these relationships within a clinically heterogeneous neurological cohort and the subsequent comparison of observations with documented cerebrovascular ischemic events (CIE; ischaemic stroke or transient ischaemic attack) against other neurological presentations. Rather than presupposing that greater adiposity should correspond to greater systemic inflammation, the study explicitly examines whether such a relationship is present, its direction and magnitude, and its robustness to influential NLR observations.

Accordingly, the principal research question is: *Do anthropometric indicators of adiposity show consistent associations with systemic inflammatory markers in patients with neurological disease, and do anthropometric or inflammatory profiles differ according to the presence of a cerebrovascular ischaemic event?*

The primary objective is to examine the associations of BMI, MUAC, waist circumference, and WHtR with ESR, leukocyte count, neutrophil count, lymphocyte count, and NLR. Secondary objectives are to characterise the internal correlation structure of the anthropometric and inflammatory domains; compare inflammatory profiles across BMI categories; assess the robustness of the principal anthropometric-NLR relationships to extreme NLR observations; characterise the diagnostic distribution of the cohort; and compare anthropometric and inflammatory characteristics between patients with documented CIE and those with other neurological diseases.

Given the exploratory nature and limited size of the cohort, the study is designed to estimate patterns, directions, and effect magnitudes rather than to establish causal relationships or definitive predictive thresholds. Rank-based correlation analyses and non-parametric between-group comparisons are therefore used, together with correction for multiple testing and sensitivity analyses for extreme NLR values. This analytical strategy is intended to assess whether the hypothesised adiposity-inflammation relationship is sufficiently coherent to be detectable across several complementary measures and whether it remains evident in the context of neurological disease.

The article describes the study population, anthropometric and laboratory measurements, diagnostic classification, and statistical strategy; presents the associations between adiposity and inflammatory markers, BMI-stratified inflammatory profiles, sensitivity analyses, and comparisons according to CIE status; and subsequently discusses the findings in relation to the apparently heterogeneous literature on obesity, systemic inflammation, and cerebrovascular disease.

2. Materials and Methods

2.1. Study design and clinical setting

This retrospective, exploratory observational study was conducted using clinical monitoring records from adult patients evaluated in the Neurology Department of the Emergency County Hospital of Brăila, Romania, during July 2026. The analysis was designed to investigate the relationship between anthropometric adiposity, systemic inflammatory markers, and cerebrovascular ischaemic events in a real-world neurological population.

The analytical dataset comprised 32 clinical monitoring encounters together for comparing stroke patients with nonstroke patients but with neurological involvement.. Repeated patient entries in the source database represented distinct monitoring visits and were therefore retained as separate clinical records, so we investigated 17 patients with stroke pathology comparing them with the nonstroke patients from the BMI point of view and regarding the inflammatory status . Patient names and other direct identifiers were not used in any statistical analysis. Each eligible record was treated as one clinical monitoring encounter for the purposes of the present exploratory analysis (Table 1).

Table 1. Definitions, reference ranges, and data availability for the anthropometric, inflammatory, haematological, and metabolic variables included in the analysis

Variable	Full name / definition	Reference range / clinical interpretation	Valid n	Missing
BMI	Body Mass Index (kg/m²)	<18.5: underweight; 18.5-24.9: normal weight; 25.0-29.9: overweight; 30.0-34.9: obesity class I; 35.0-39.9: obesity class II; ≥40.0: obesity class III	32	0
MUAC	Mid-Upper Arm Circumference (cm)	No single universal adult reference interval; interpretation depends on sex, age, nutritional status, and population-specific thresholds	32	0
Waist circumference	Waist Circumference (cm)	European populations: men <94 cm / women <80 cm: lower risk; men 94-101.9 cm / women 80-87.9 cm: increased cardiometabolic risk; men ≥102 cm / women ≥88 cm: substantially increased risk	32	0
Height	Body Height (cm)	No pathological reference interval; used for calculation of BMI and WHtR	32	0
Weight	Body Weight (kg)	No universal stand-alone normal interval; interpreted relative to height and body composition	32	0
Age	Age at Clinical Monitoring (years)	No normal/pathological reference interval; analysed as a continuous demographic variable	31	1
ESR	Erythrocyte Sedimentation Rate (mm/h)	Age- and sex-dependent; values above the laboratory reference interval may indicate an increased nonspecific inflammatory response	29	3
Haemoglobin	Haemoglobin Concentration (g/dL)	Approx. men: 13-17 g/dL; women: 12-16 g/dL; values below the reference interval suggest anemia	29	3
Leukocytes	Total Leukocyte Count (×10⁹/L)	Approx. 4.0-10.0 ×10⁹/L; <4.0: leukopenia; >10.0: leukocytosis	29	3
Neutrophils	Absolute Neutrophil Count (×10⁹/L)	Approx. 1.5-7.5 ×10⁹/L; values below range indicate neutropenia and values above range indicate neutrophilia	29	3
Lymphocytes	Absolute Lymphocyte Count (×10⁹/L)	Approx. 1.0-4.0 ×10⁹/L; values below range indicate lymphopenia and values above range indicate lymphocytosis	29	3
NLR	Neutrophil-to-Lymphocyte Ratio	No universally accepted clinical reference interval or universal diagnostic cut-off; analysed primarily as a continuous inflammatory marker	29	3
Total cholesterol	Total Cholesterol (mg/dL)	<200 mg/dL: desirable; 200-239 mg/dL: borderline high; ≥240 mg/dL: high	19	13

Variable	Full name / definition	Reference range / clinical interpretation	Valid n	Missing
LDL-C	Low-Density Lipoprotein Cholesterol (mg/dL)	Interpretation and therapeutic targets depend on individual cardiovascular risk; no single universal target applies to all patients	16	16
Triglycerides	Serum Triglycerides (mg/dL)	<150 mg/dL: normal; 150-199 mg/dL: borderline high; 200-499 mg/dL: high; ≥500 mg/dL: very high	21	11
Hip circumference	Hip Circumference (cm)	No universal stand-alone reference interval; principally interpreted in combination with waist circumference for calculation of WHR	13	19

Note: Reference intervals are provided for clinical interpretation. Laboratory-dependent haematological and biochemical measurements should be interpreted according to the reference intervals and units used by the analysing laboratory. NLR was treated as a continuous variable because no universally accepted diagnostic threshold exists.
Abbreviations: BMI, Body Mass Index; MUAC, Mid-Upper Arm Circumference; WHtR, Waist-to-Height Ratio; WHR, Waist-to-Hip Ratio; ESR, Erythrocyte Sedimentation Rate; NLR, Neutrophil-to-Lymphocyte Ratio; LDL-C, Low-Density Lipoprotein Cholesterol.

Source: Authors' own contribution

The study was conducted in accordance with applicable ethical standards. Ethical approval was reported under Ethics Committee decision no. 36/05.12.2023.

2.2. Eligibility criteria and study population

Records were eligible if they corresponded to adults aged ≥18 years who had undergone neurological evaluation or inpatient monitoring during the study period and for whom clinically relevant neurological and anthropometric information was available.

According to the study protocol, records relating exclusively to isolated vertigo or headache without relevant comorbidities or complications, severe protein-energy malnutrition, somatisation disorders, isolated panic attacks, stable non-progressive cerebral palsy, progressive neuromuscular diseases, treated multiple sclerosis under surveillance, isolated neuromotor developmental disorders, intellectual disability, or isolated autism spectrum disorders were not included to the analysis of the inflammatory status in this study, because of multiple pathophysiological mechanisms underlying in this conditions and so the comparative approach would be influenced by these mechanisms involved. .

Because the present study was exploratory and based on routinely collected clinical data, the number of observations available for individual analyses varied according to variable-specific missingness. No imputation of missing clinical or laboratory values was performed.

2.3. Anthropometric assessment and derived adiposity measures

Anthropometric assessment was based on the measurements documented in the clinical monitoring records and included body weight (kg), height (cm), body mass index (BMI, kg/m²), mid-upper arm circumference (MUAC, cm), waist circumference (cm), and, where available, hip circumference (cm). Rather than relying on a single measure of body size, the present analysis considered several complementary anthropometric indicators to characterise overall and central adiposity. This approach was selected because BMI primarily reflects body mass relative to height, whereas waist circumference and waist-to-height ratio (WHtR) provide additional information regarding central body-size distribution. MUAC was retained as a complementary peripheral anthropometric measure.

Body weight and height were available for all 32 clinical monitoring records. BMI values were also available for all records and were subjected to an internal consistency check by recalculating BMI from the recorded weight and height measurements. BMI was calculated as:

$$BMI\ (kg/m^2) = \frac{Body\ weight\ (kg)}{[Height\ (m)]^2} \quad (1)$$

The recalculated BMI values were compared with those originally recorded in the database. Only negligible differences attributable to numerical rounding were identified, supporting the

internal consistency of the anthropometric data. The recorded BMI values were therefore retained for the subsequent statistical analyses.

For descriptive and group-based analyses, BMI was interpreted according to conventional adult categories. Values below 18.5 kg/m² were considered underweight, values between 18.5 and 24.9 kg/m² normal weight, values between 25.0 and 29.9 kg/m² overweight, and values ≥30.0 kg/m² obesity. Obesity may be further classified as class I (30.0-34.9 kg/m²), class II (35.0-39.9 kg/m²), and class III (≥40.0 kg/m²). However, given the limited sample size of the present exploratory cohort, the obesity classes were combined into a single obesity category (BMI ≥30.0 kg/m²) for inferential group comparisons. Accordingly, the principal BMI categories used in comparative analyses were normal weight, overweight, and obesity. BMI was nevertheless retained as a continuous variable in correlation and regression analyses to avoid the loss of information associated with categorisation.

Waist circumference was analysed as a continuous measure of central adiposity. In addition, its clinical interpretation considered sex-specific thresholds commonly applied to European adult populations. Waist circumference values <94 cm in men and <80 cm in women were considered below the threshold for increased cardiometabolic risk; values of 94-101.9 cm in men and 80-87.9 cm in women indicated increased risk; and values ≥102 cm in men and ≥88 cm in women indicated substantially increased cardiometabolic risk. Because these thresholds are sex-specific, waist circumference was analysed primarily as a continuous variable, while threshold-based classification was used for complementary clinical characterisation.

To complement BMI and waist circumference, WHtR was derived for each clinical monitoring record for which waist circumference and height were available. WHtR was calculated as:

$$WHtR = \frac{Waist\ circumference\ (cm)}{Height\ (cm)} \quad (2)$$

Because both measurements were expressed in centimetres, WHtR is dimensionless. Waist circumference and height were available for all 32 records, allowing WHtR to be calculated without additional missing observations. WHtR was analysed primarily as a continuous indicator of central adiposity, thereby preserving the full variability of the measurement.

MUAC was available for all 32 clinical monitoring records and was expressed in centimetres. Unlike BMI, MUAC does not have a single universally applicable reference interval for the general adult population, because its interpretation may vary according to age, sex, nutritional status, and the population under investigation. Consequently, MUAC was not dichotomised according to an arbitrary threshold. It was retained as a continuous anthropometric variable and examined in relation to both other adiposity measures and systemic inflammatory markers.

Hip circumference was recorded for only a subset of the analytical dataset (13 of 32 records). Because of this substantial degree of missingness, hip circumference was included in the descriptive assessment of available anthropometric information but was not considered a principal exposure variable. For the same reason, waist-to-hip ratio (WHR), although potentially informative for the characterisation of body-fat distribution, was not included in the principal inferential analyses. This decision was taken to avoid restricting the main analyses to a substantially smaller and potentially non-representative subset of the cohort.

The principal anthropometric indicators used in the inferential analyses were therefore BMI, MUAC, waist circumference, and WHtR. These measures were analysed as complementary rather than interchangeable indicators. BMI represented overall weight relative to height, waist circumference and WHtR were used to characterise central adiposity, and MUAC provided an additional peripheral anthropometric dimension.

This multidimensional anthropometric approach was particularly relevant to the exploratory objective of the study, which was not to assume a priori that a single measure of adiposity adequately represented the biological relationship between body composition and systemic

inflammation. Instead, each anthropometric indicator was independently evaluated in relation to the inflammatory profile and cerebrovascular ischaemic-event status. This analytical strategy enabled assessment of whether different manifestations of anthropometric adiposity showed convergent or divergent relationships with systemic inflammatory markers.

2.4. Inflammatory and haematological variables

The systemic inflammatory and haematological profile was characterised using routinely available laboratory parameters recorded during clinical monitoring. The principal variables included erythrocyte sedimentation rate (ESR), total leukocyte count, absolute neutrophil count, absolute lymphocyte count, and the neutrophil-to-lymphocyte ratio (NLR). Haemoglobin concentration was additionally included to characterise the haematological status of the study population. Together, these parameters provided complementary information regarding systemic inflammatory activity and hematological status rather than being interpreted as interchangeable indicators of a single biological process.

ESR was considered a nonspecific marker of systemic inflammatory activity and was analysed as a continuous variable. Because ESR reference values may vary according to age, sex, and laboratory-specific methodology, the primary statistical analyses were based on the measured continuous values rather than on a universal dichotomous normal/abnormal classification. This approach preserved the variability of ESR within the cohort and avoided introducing a threshold that could not be uniformly applied to all clinical records.

Total leukocyte count, absolute neutrophil count, and absolute lymphocyte count were evaluated both as individual haematological parameters and as components of the systemic inflammatory profile. Leukocyte count provides an overall measure of circulating white blood cells, whereas neutrophil and lymphocyte counts represent distinct cellular components of the inflammatory and immune response. Their separate inclusion was particularly important because changes in the balance between circulating neutrophils and lymphocytes may not be adequately represented by the total leukocyte count alone.

The neutrophil-to-lymphocyte ratio was used as a derived inflammatory index integrating the relative contribution of these two leukocyte populations. NLR was calculated for each record with available neutrophil and lymphocyte measurements using the following expression:

$$NLR = \frac{Absolute\ neutrophil\ count}{Absolute\ lymphocyte\ count} \quad (3)$$

Because the numerator and denominator were expressed using the same concentration units, NLR was treated as a dimensionless ratio.

The source database contained both the individual neutrophil and lymphocyte measurements and previously recorded NLR values. As part of the data-quality assessment, NLR was therefore independently recalculated for every record for which both components were available. The recalculated values were compared with the NLR values recorded in the original dataset. Only negligible discrepancies attributable to numerical rounding were identified, supporting the internal consistency of the derived inflammatory measure. NLR was available for 29 of the 32 clinical monitoring records, while three records lacked the laboratory information required for its calculation. Missing NLR values were not imputed.

NLR was analysed primarily as a continuous variable. This decision was particularly important because no single universally accepted NLR threshold can distinguish normal from pathological systemic inflammation across different neurological conditions and clinical contexts. Consequently, the colour-coded categories used in the initial descriptive version of the study, such as “correct,” “acceptable,” “alarm,” or “bad” NLR values, were not retained in the revised analytical framework. No unvalidated clinical cut-off was imposed on the NLR distribution.

The continuous treatment of NLR also allowed the analysis to account for the substantial interindividual variability observed in the neurological population. Because acute and chronic neurological disorders may be accompanied by heterogeneous inflammatory responses, the magnitude of NLR was interpreted as an indicator of relative systemic inflammatory activity rather

than as a disease-specific diagnostic marker. Accordingly, NLR was not considered a priori to provide evidence of cerebrovascular ischaemia or to distinguish ischaemic from non-ischaemic neurological disease independently of the broader clinical context.

The distribution of NLR was formally evaluated before inferential testing. Given its marked positive skewness and the presence of high values in a limited number of clinical records, rank-based statistical procedures were prioritised for unadjusted analyses involving NLR. Spearman rank correlation was therefore used to evaluate its association with the principal anthropometric adiposity indicators. Sensitivity analyses were additionally performed to determine whether extreme NLR observations materially influenced the magnitude or direction of the observed anthropometric-inflammatory associations.

Haemoglobin concentration was analysed as a continuous hematological variable and was used primarily for characterisation of the study population. Its interpretation considered sex-specific adult reference intervals, while recognising that laboratory-specific reference values may differ. Haemoglobin was not considered a direct inflammatory marker and was therefore distinguished conceptually from ESR, leukocyte count, neutrophil count, lymphocyte count, and NLR in the principal inflammatory analyses.

The primary inflammatory domain used for inferential analysis therefore comprised ESR, total leukocyte count, absolute neutrophil count, absolute lymphocyte count, and NLR. These variables were examined both individually and jointly through correlation analysis. This approach allowed assessment of whether the inflammatory markers demonstrated internal convergence and, importantly, whether this inflammatory domain showed corresponding associations with the anthropometric adiposity domain represented by BMI, MUAC, waist circumference, and WHtR.

The analytical framework did not assume a priori that increased anthropometric adiposity would necessarily correspond to a proportionally increased systemic inflammatory response. Instead, the relationships between the two biological domains were empirically evaluated. This distinction was central to the exploratory design of the study and enabled anthropometric and inflammatory characteristics to be investigated as potentially complementary biological dimensions within a heterogeneous neurological population.

2.5. Metabolic and cardiovascular variables

Metabolic and cardiovascular characteristics were evaluated as complementary components of the clinical phenotype because both adiposity and systemic inflammation may coexist with established cardiometabolic risk factors. The available information was therefore reviewed to characterise the metabolic and vascular background of the neurological cohort and to distinguish these characteristics from the principal anthropometric and inflammatory domains investigated in the study.

The metabolic laboratory profile included total cholesterol, low-density lipoprotein cholesterol (LDL-C), and triglycerides. Total cholesterol was available for 19 of the 32 clinical monitoring records, LDL-C for 16 records, and triglycerides for 21 records. Because these measurements were not available uniformly across the analytical dataset, lipid parameters were retained for descriptive and exploratory analyses based on available observations, and missing values were not imputed.

Total cholesterol and triglycerides were analysed as continuous variables to preserve the information contained in their original measurements. For complementary clinical interpretation, total cholesterol values were considered desirable when <200 mg/dL, borderline high between 200 and 239 mg/dL, and high when ≥240 mg/dL. Triglyceride concentrations were interpreted as normal when <150 mg/dL, borderline high between 150 and 199 mg/dL, high between 200 and 499 mg/dL, and very high when ≥500 mg/dL. LDL-C was analysed primarily as a continuous variable because its clinical interpretation and recommended therapeutic targets depend on the individual patient's overall cardiovascular-risk category rather than on a single universal threshold.

Information regarding diabetes mellitus and arterial blood pressure was also retained from the clinical records as part of the cardiometabolic characterisation of the cohort. These variables were considered clinically relevant because they may contribute to the vascular-risk background of neurological patients and may influence the interpretation of associations involving adiposity, systemic inflammation, and cerebrovascular disease.

Diabetes mellitus was identified from the documented clinical history or diagnostic information available in the source records. It was treated as a categorical clinical characteristic when the presence of diabetes was explicitly documented. No assumption of absence was made solely on the basis of an unrecorded diagnosis. The available dataset did not provide a sufficiently complete standardised measure of long-term glycaemic control, such as glycated haemoglobin (HbA1c), for incorporation into the principal inferential analyses.

Systolic and diastolic blood pressure measurements were considered cardiovascular characteristics of the clinical monitoring encounters. Blood pressure information was interpreted in the context of the recorded clinical assessment rather than being treated as a direct measure of chronic hypertensive burden. A measurement obtained during a neurological clinical encounter may reflect both the patient's underlying cardiovascular status and the acute clinical context; consequently, individual blood pressure measurements were not considered equivalent to a diagnosis of chronic arterial hypertension.

The metabolic and cardiovascular variables were therefore used primarily to describe the broader cardiometabolic context of the study population and, where data completeness permitted, to support exploratory analyses. They were not incorporated indiscriminately into a single multivariable model. This decision reflected both the limited number of clinical monitoring records and the variable-specific amount of missing information, particularly for lipid measurements.

Accordingly, the principal inferential framework remained focused on the relationship between the predefined anthropometric domain — BMI, MUAC, waist circumference, and WHtR — and the systemic inflammatory domain — ESR, total leukocyte count, absolute neutrophil count, absolute lymphocyte count, and NLR. Metabolic and cardiovascular characteristics were used to contextualise these relationships and to evaluate whether the observed anthropometric and inflammatory patterns occurred within a broader cardiometabolic risk profile.

This analytical distinction was intended to avoid overfitting while retaining clinically relevant information. Given the exploratory design and limited sample size, no high-dimensional cardiovascular risk prediction model was developed, and missing metabolic measurements were not imputed. Where metabolic or cardiovascular variables were included in exploratory comparisons, the number of available observations was reported explicitly.

2.6. Classification of neurological diagnoses and cerebrovascular events

Neurological diagnoses were derived from the diagnostic information documented in the clinical monitoring records. Because the source dataset included patients with a heterogeneous spectrum of neurological disorders, diagnostic information was systematically reviewed and recoded before statistical analysis to permit clinically interpretable comparisons while preserving the diagnostic information available in the original records.

For analytical purposes, cerebrovascular presentations were classified according to the documented type of event into five categories: ischaemic stroke, transient ischemic attack (TIA), haemorrhagic stroke, stroke of unspecified mechanism, and other neurological disease. This classification was performed before comparative statistical analyses and was based exclusively on the diagnostic and treatment information available in the source dataset.

Ischaemic stroke was assigned when the clinical record explicitly documented an ischaemic cerebrovascular event or when the documented cerebrovascular presentation was accompanied by an acute reperfusion intervention specifically compatible with ischaemic stroke management, such as intravenous thrombolysis and/or mechanical thrombectomy, in the absence of documentation indicating a primary haemorrhagic stroke.

Transient ischaemic attack (TIA) was retained as a separate diagnostic category when explicitly documented in the clinical record. TIA was not considered equivalent to established ischaemic stroke. However, because both conditions represent ischaemic cerebrovascular presentations, ischaemic stroke and TIA were combined for a predefined composite analytical category termed cerebrovascular ischemic event (CIE) when the objective was to compare ischaemic cerebrovascular presentations with other neurological disorders.

Accordingly, the composite variable was defined as:

$$CIE = documented\ ischemic\ stroke\ or\ documented\ TIA \quad (4)$$

Records classified as haemorrhagic stroke, stroke of unspecified mechanism, or other neurological disease were not coded as confirmed CIEs in the primary definition.

Haemorrhagic stroke was assigned when the diagnostic record explicitly identified a primary haemorrhagic cerebrovascular event. These observations were maintained as a distinct category because the underlying vascular mechanism differs fundamentally from that of ischaemic stroke. Primary haemorrhagic strokes were therefore not included in the principal CIE group.

A distinction was also made between primary haemorrhagic stroke and haemorrhagic transformation of an ischaemic stroke. When the available diagnostic information indicated that haemorrhagic transformation occurred in the context of an underlying ischaemic cerebrovascular event, the record remained classified according to the primary ischaemic mechanism. This prevented secondary haemorrhagic transformation from being incorrectly interpreted as a primary haemorrhagic stroke.

For the primary comparative analysis, the cerebrovascular ischaemic-event (CIE) group was restricted to observations with a documented ischaemic mechanism and documented TIA. Strokes of undetermined type were retained as a separate diagnostic category and were excluded from the primary CIE versus other-neurological-disease comparison to avoid potential aetiological misclassification.

The category other neurological disease comprised clinical monitoring encounters without a documented cerebrovascular ischaemic or haemorrhagic event. This group represented the heterogeneous non-cerebrovascular neurological population available in the source dataset and was used as the comparison group in analyses specifically examining the anthropometric and inflammatory characteristics associated with documented ischaemic cerebrovascular presentations.

The principal cerebrovascular comparison was therefore designed to contrast clinical monitoring encounters with a clearly documented CIE against encounters classified as other neurological disease. Primary haemorrhagic strokes and strokes of unspecified mechanism were not included in this primary two-group comparison because their inclusion could obscure mechanistically distinct cerebrovascular conditions or introduce diagnostic misclassification.

Because the study was retrospective and based on routinely documented clinical information, the diagnostic classification was restricted to the information available in the source records. Variables such as vascular territory, infarct volume, detailed neuroimaging characteristics, and standardised timing from symptom onset were not reconstructed when they were not explicitly available in the analytical dataset. Likewise, the study did not retrospectively infer stroke mechanism from information that was absent from the source records.

2.7. Data quality control and missing-data management

Prior to statistical analysis, the analytical dataset underwent a structured quality-assurance procedure aimed at verifying data completeness, internal consistency, numerical plausibility, and concordance between directly recorded and derived variables. The verification process was performed before inferential analyses and included assessment of variable formats, identification of missing observations, evaluation of implausible numerical entries, and cross-validation of variables that could be independently reconstructed from their component measurements. Anthropometric and laboratory variables were converted to standardised numerical formats while preserving their

original measurement units. Values that were not accompanied by a valid quantitative measurement were treated as missing rather than assigned a numerical value. The number of available observations was determined separately for each variable because data completeness differed across anthropometric, haematological, inflammatory, and metabolic measurements.

Internal consistency was assessed for the two principal derived variables for which their constituent measurements were available. BMI was independently recalculated from recorded body weight and height, and NLR was independently recalculated from absolute neutrophil and lymphocyte counts. Recalculated values were compared with those recorded in the source dataset. The observed differences were limited to numerical rounding and did not indicate systematic discrepancies; consequently, the validated recorded values were retained for analysis.

Following data-quality assessment, 32 eligible observations were retained in the final analytical dataset. Data preparation was completed prior to statistical analysis, and all subsequent analyses were performed using a de-identified dataset containing only the variables required for the study objectives. Data completeness was evaluated at the variable level and was explicitly documented before statistical modelling. Anthropometric measurements forming the principal adiposity domain were complete, whereas inflammatory, haematological, and particularly metabolic variables showed varying degrees of missingness. Consequently, the effective sample size was allowed to vary across analyses according to the availability of the variables required for each statistical procedure. The number of observations contributing to each principal analysis was reported together with the corresponding result.

Missing data were managed according to the availability of each variable, without statistical imputation. Analyses were conducted using the observed data available for the variables involved in each specific assessment; consequently, the analytical sample size varied across statistical procedures. The number of observations included in each analysis was reported explicitly to ensure transparency regarding data completeness.

No statistical imputation was performed. Given the limited sample size, the exploratory character of the study, and the variable-specific pattern of missingness, model-based or multiple imputation would have required assumptions that could not be adequately supported by the available dataset. The primary analyses therefore remained based on observed data, with the effective sample size reported transparently for each analysis.

Variables with substantial missingness were not automatically discarded from the study. Instead, their analytical role was determined according to data availability and clinical relevance. Variables with adequate coverage were eligible for the principal inferential analyses, whereas variables available only in a restricted subset of observations were retained primarily for descriptive or secondary exploratory purposes. In particular, incomplete lipid measurements and hip circumference were not used as mandatory covariates in the principal models, thereby avoiding unnecessary reduction of the analytical sample.

This data-management strategy was designed to maximise the use of observed clinical information while maintaining transparency regarding data completeness and avoiding unsupported reconstruction of unavailable measurements.

2.8. Statistical analysis

Statistical analyses were performed in Python using established scientific-computing and statistical libraries. All statistical tests were two-sided, with a nominal significance level of $p < 0.05$. Given the exploratory design and limited sample size, statistical inference was based on the joint consideration of effect estimates, 95% confidence intervals (CIs), nominal p-values, and multiplicity-adjusted results. Statistical significance was therefore not considered in isolation when interpreting the magnitude or clinical relevance of observed associations.

Continuous variables were characterised using measures of central tendency and dispersion. Variables with approximately symmetric distributions were summarised as mean ± standard deviation (SD), whereas variables with asymmetric distributions were reported as median and

interquartile range (IQR). Minimum and maximum values were additionally examined to characterise the observed range and identify potentially influential extreme observations. Categorical variables were summarised as absolute frequencies and percentages. Because data availability differed across variables, the number of observations contributing to each analysis was reported explicitly.

Distributional characteristics were assessed by graphical inspection in conjunction with the Shapiro-Wilk test. The selection of parametric or non-parametric procedures was guided by distributional characteristics, sample size, and the measurement properties of the variables. Rank-based methods were preferentially used when the assumptions underlying parametric inference were not adequately supported.

The principal association analysis evaluated relationships between the anthropometric adiposity domain, comprising BMI, MUAC, waist circumference, and WHtR, and the systemic inflammatory domain, comprising ESR, total leukocyte count, absolute neutrophil count, absolute lymphocyte count, and NLR. Associations between continuous variables were quantified using Spearman's rank correlation coefficient (ρ). Each correlation was calculated using observations with available measurements for both variables under consideration. Correlation coefficients were interpreted according to their direction, magnitude, and statistical uncertainty.

Because multiple associations were examined within the anthropometric-inflammatory correlation framework, the Benjamini-Hochberg procedure was applied to control the false discovery rate (FDR). Nominal and FDR-adjusted p-values were reported to distinguish exploratory associations from those remaining statistically supported after correction for multiple comparisons.

Differences in systemic inflammatory markers across BMI categories — normal weight, overweight, and obesity-were evaluated using the Kruskal — Wallis test. For comparisons involving two independent clinical groups, the Mann-Whitney U test was used. Cliff's delta (δ) was additionally calculated for two-group comparisons as a non-parametric measure of effect size, providing an estimate of the magnitude and direction of group differences independently of sample-size-dependent significance testing.

Secondary analyses evaluated anthropometric and inflammatory differences between observations classified as cerebrovascular ischaemic events (CIE) and those classified as other neurological diseases, according to the diagnostic framework described above. The principal CIE definition comprised documented ischaemic stroke and TIA. Primary haemorrhagic stroke and stroke of undetermined type were not included in this primary two-group comparison, thereby preserving aetiological specificity.

The robustness of the anthropometric-NLR associations was evaluated through sensitivity analyses addressing potentially influential extreme NLR values. Spearman correlations were repeated after exclusion of the single highest NLR observation and, separately, after exclusion of extreme NLR values identified using the conservative Tukey criterion:

$$Extreme\ NLR\ value\colon\ NLR\ >\ Q3\ +\ 3\times IQR \qquad (5)$$

where Q3 represents the third quartile and IQR the interquartile range. These sensitivity analyses were used to assess whether the direction and magnitude of the principal associations were materially altered by extreme inflammatory observations.

Given the exploratory design and limited sample size, multivariable analyses were intentionally restricted to parsimonious, clinically interpretable models. No automated or data-driven variable-selection procedures were applied. This strategy was adopted to reduce model instability and overfitting while preserving direct interpretation of the relationships among anthropometric adiposity, systemic inflammatory response, and cerebrovascular ischaemic events.

3. Results

The analytical strategy was organised to characterise the study population, evaluate the internal structure of the anthropometric and inflammatory domains, quantify cross-domain

associations, compare inflammatory profiles across adiposity categories, and examine differences according to cerebrovascular ischaemic-event status. Descriptive statistics were first used to establish the distributional characteristics and completeness of the principal variables. These results informed the choice of subsequent non-parametric and adjusted analyses and provided the basis for interpreting the magnitude and robustness of the observed associations.

3.1. Characteristics of the Analytical Cohort

The final analytical dataset comprised 32 eligible observations. Data completeness was highest for the anthropometric domain: BMI, MUAC, waist circumference, body weight, and height were available for all 32 observations. Age was available for 31 observations. The main inflammatory and haematological variables, including ESR, total leukocyte count, neutrophil count, lymphocyte count, and NLR, were available for 29 observations. This difference in data availability was reflected in the effective sample size of the subsequent analyses.

The analysed population was predominantly older, with a mean age of 69.10 ± 10.52 years and a median age of 70.0 years [IQR: 62.0-75.5]. The observed age range extended from 42 to 88 years, indicating a broad adult neurological population with a concentration in older age groups.

Anthropometric assessment showed a high burden of excess body weight. Mean BMI was 28.91 ± 3.61 kg/m², while the median BMI was 29.66 kg/m² [IQR: 26.95-30.93], with values ranging from 21.37 to 39.06 kg/m². The distribution of BMI did not deviate significantly from normality according to the Shapiro-Wilk test ($p = 0.139$). MUAC showed a mean value of 17.03 ± 2.43 cm and a median of 18.0 cm [IQR: 15.0-19.0], with an observed range of 12-22 cm. Its distribution was also close to normality (Shapiro-Wilk $p = 0.067$).

Waist circumference showed greater asymmetry. The mean waist circumference was 82.38 ± 10.42 cm, while the median was 82.0 cm [IQR: 79.75-88.00], with values ranging from 57 to 120 cm. The Shapiro-Wilk test indicated a significant deviation from normality ($p < 0.001$). A similar pattern was observed for WHtR, which had a mean value of 0.487 ± 0.065 and a median of 0.484 [IQR: 0.465-0.513], with values between 0.365 and 0.750. WHtR also showed a significant departure from normality ($p < 0.001$).

The inflammatory and haematological profile showed greater dispersion and stronger distributional asymmetry than the principal anthropometric variables. Mean ESR was 18.24 ± 22.48 mm/h, whereas the median was 11.0 mm/h [IQR: 8.0-17.0], with a wide range of 6-123 mm/h. Total leukocyte count had a mean of 9.04 ± 2.74 ×10⁹/L and a median of 8.17 ×10⁹/L [IQR: 7.29-10.44]. Absolute neutrophil count showed a mean of 6.47 ± 2.92 ×10⁹/L and a median of 5.78 ×10⁹/L [IQR: 4.47-7.44]. Both leukocyte and neutrophil distributions deviated significantly from normality.

Absolute lymphocyte count showed substantial variability, with a mean of 2.22 ± 2.61 ×10⁹/L and a median of 1.76 ×10⁹/L [IQR: 1.24-2.44]. Values ranged from 0.25 to 15.25 ×10⁹/L, and the distribution was markedly non-normal ($p < 0.001$).

NLR exhibited the most pronounced variability among the principal inflammatory measures. The mean NLR was 5.67 ± 8.21, while the median was substantially lower at 3.20 [IQR: 2.35-4.57], reflecting the influence of a limited number of markedly elevated values. NLR ranged from 0.81 to 37.82 and showed a highly non-normal distribution (Shapiro-Wilk $p < 0.001$). This marked difference between mean and median, together with the broad range, indicated substantial heterogeneity in the systemic inflammatory response across the analysed observations (Table 2).

Table 2. Descriptive characteristics and distributional assessment of the principal anthropometric and inflammatory variables

Variable	n	Mean ± SD	Median [IQR]	Range	Shapiro-Wilk p
Age, years	31	69.10 ± 10.52	70.00 [62.00-75.50]	42-88	0.811
BMI, kg/m²	32	28.91 ± 3.61	29.66 [26.95-30.93]	21.37-39.06	0.139
MUAC, cm	32	17.03 ± 2.43	18.00 [15.00-19.00]	12-22	0.067
Waist circumference, cm	32	82.38 ± 10.42	82.00 [79.75-88.00]	57-120	<0.001
WHtR	32	0.487 ± 0.065	0.484 [0.465-0.513]	0.365-0.750	<0.001

ESR, mm/h	29	18.24 ± 22.48	11.00 [8.00-17.00]	6-123	<0.001
Leukocytes, ×10⁹/L	29	9.04 ± 2.74	8.17 [7.29-10.44]	5.86-16.17	0.001
Neutrophils, ×10⁹/L	29	6.47 ± 2.92	5.78 [4.47-7.44]	2.99-14.37	<0.001
Lymphocytes, ×10⁹/L	29	2.22 ± 2.61	1.76 [1.24-2.44]	0.25-15.25	<0.001
NLR	29	5.67 ± 8.21	3.20 [2.35-4.57]	0.81-37.82	<0.001

Source: Authors' calculation using Python 3.14.7 version

BMI classification further illustrated the prevalence of excess body weight within the cohort. Four observations (12.5%) were classified as normal weight, 16 (50.0%) as overweight, and 12 (37.5%) as obese. Accordingly, 28 of 32 observations (87.5%) had a BMI ≥25 kg/m², indicating that excess body weight was highly prevalent in the analyzed neurological population.

The distributional assessment also demonstrated that the anthropometric and inflammatory variables did not share a uniform statistical profile. Age, BMI, and MUAC were approximately normally distributed, whereas waist circumference, WHtR, and all principal inflammatory variables showed significant departures from normality. These findings supported the use of rank-based procedures for the principal unadjusted association and group-comparison analyses reported in the following sections.

Figure 1 summarises the distribution of BMI categories in the analytical cohort and visually emphasises the predominance of overweight and obesity.

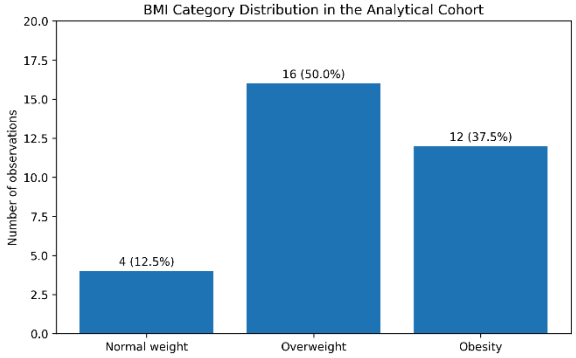

Figure 1. Distribution of BMI categories in the analytical cohort.

Source: Authors` contribution using Python 3.14.7 version

Figure 1 confirms the predominance of excess body weight within the analytical cohort. Half of the observations were classified as overweight, while more than one-third were classified as obese, leaving only a small proportion within the normal-weight range. Overall, 87.5% of the analysed observations had a BMI ≥25 kg/m². This distribution indicates that increased adiposity was highly prevalent in the neurological population included in the study and provides the descriptive context for the subsequent analyses examining whether higher anthropometric adiposity was accompanied by a distinct systemic inflammatory profile.

3.2. Associations between anthropometric adiposity and systemic inflammatory markers

The principal cross-domain analysis evaluated 20 pairwise associations between four anthropometric adiposity indicators-BMI, MUAC, waist circumference, and WHtR-and five systemic inflammatory markers-ESR, total leukocyte count, absolute neutrophil count, absolute lymphocyte count, and NLR. Spearman rank correlations were used because several inflammatory variables exhibited non-normal distributions. Across the full set of anthropometric-inflammatory comparisons, the observed correlation coefficients were generally weak and showed no uniform directional pattern. Some associations were positive, whereas others were inverse, indicating that greater anthropometric adiposity was not consistently accompanied by a higher systemic inflammatory profile.

BMI showed no meaningful association with ESR (ρ = -0.115, p = 0.551), leukocyte count (ρ = 0.085, p = 0.659), or neutrophil count (ρ = -0.002, p = 0.992). The relationships with lymphocyte count and NLR were somewhat larger in magnitude but remained statistically non-significant. BMI was positively associated with lymphocyte count (ρ = 0.285, p = 0.134) and inversely associated with NLR (ρ = -0.285, p = 0.135). Thus, the observed BMI-NLR association did not support a pattern of increasing NLR with increasing BMI. MUAC demonstrated the weakest overall relationship with the inflammatory domain. Correlation coefficients ranged from -0.114 to 0.071 across ESR, leukocytes, neutrophils, lymphocytes, and NLR, and all nominal p-values were >0.55. The MUAC-NLR association was negligible (ρ = -0.091, p = 0.637), indicating little evidence of a monotonic relationship between peripheral anthropometric size and NLR in the analysed cohort.

Waist circumference showed the largest cross-domain coefficients. Its association with lymphocyte count was positive and of moderate magnitude (ρ = 0.362, p = 0.053), whereas its association with NLR was inverse (ρ = -0.350, p = 0.063). Waist circumference was also inversely associated with ESR (ρ = -0.248, p = 0.195) and neutrophil count (ρ = -0.167, p = 0.387). Although the waist-lymphocyte and waist-NLR relationships approached nominal significance, neither met the conventional $p < 0.05$ threshold.

WHtR showed a similar absence of robust association with the inflammatory domain. The largest coefficient involved ESR and was inverse (ρ = -0.267, p = 0.162). Associations with leukocyte count (ρ = -0.034, p = 0.860), neutrophil count (ρ = -0.055, p = 0.776), lymphocyte count (ρ = 0.111, p = 0.565), and NLR (ρ = -0.130, p = 0.501) were weak.

Because 20 anthropometric-inflammatory associations were examined, nominal p-values were adjusted using the Benjamini-Hochberg procedure. None of the cross-domain associations remained statistically significant after false-discovery-rate correction. The smallest FDR-adjusted p-value was 0.626, observed for both the waist circumference-lymphocyte and waist circumference-NLR relationships (Table 3).

Table 3. Spearman associations between anthropometric adiposity and systemic inflammatory markers

Anthropometric marker	Inflammatory marker	n	Spearman ρ	Nominal p	FDR-adjusted p
BMI	ESR	29	-0.115	0.551	0.910
BMI	Leukocytes	29	0.085	0.659	0.910
BMI	Neutrophils	29	-0.002	0.992	0.992
BMI	Lymphocytes	29	0.285	0.134	0.646
BMI	NLR	29	-0.285	0.135	0.646
MUAC	ESR	29	0.006	0.974	0.992
MUAC	Leukocytes	29	-0.069	0.722	0.910
MUAC	Neutrophils	29	-0.114	0.556	0.910
MUAC	Lymphocytes	29	0.071	0.712	0.910
MUAC	NLR	29	-0.091	0.637	0.910
Waist circumference	ESR	29	-0.248	0.195	0.651
Waist circumference	Leukocytes	29	-0.067	0.728	0.910
Waist circumference	Neutrophils	29	-0.167	0.387	0.910
Waist circumference	Lymphocytes	29	0.362	0.053	0.626
Waist circumference	NLR	29	-0.350	0.063	0.626
WHtR	ESR	29	-0.267	0.162	0.646
WHtR	Leukocytes	29	-0.034	0.860	0.956
WHtR	Neutrophils	29	-0.055	0.776	0.913
WHtR	Lymphocytes	29	0.111	0.565	0.910
WHtR	NLR	29	-0.130	0.501	0.910

Source: Authors' calculation using Python 3.14.7 version

The overall pattern therefore showed limited cross-domain convergence. The anthropometric indicators did not display a consistent positive association with systemic inflammatory markers, and the observed effect sizes were predominantly small (Figure 2).

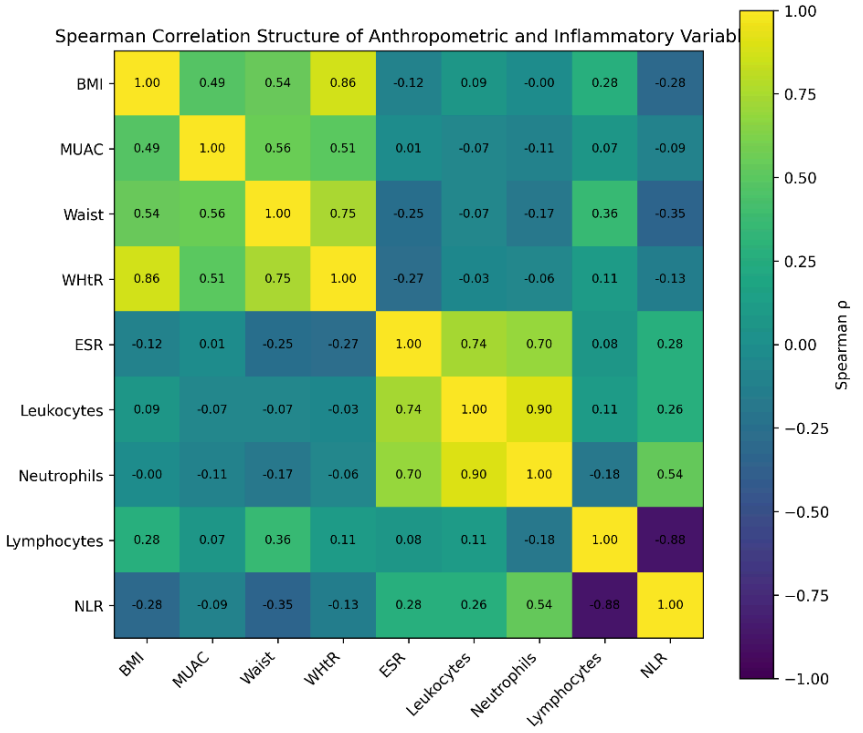

Figure 2. Spearman correlation matrix.
Source: Authors` contribution using Python 3.14.7 version

Figure 2 provides a visual synthesis of the correlation structure across the anthropometric and inflammatory variables. The anthropometric measures formed a relatively coherent cluster, with the strongest positive association observed between BMI and WHtR ($\rho = 0.860$), followed by waist circumference and WHtR ($\rho = 0.749$). Moderate positive correlations were also evident between MUAC and waist circumference ($\rho = 0.561$), BMI and waist circumference ($\rho = 0.542$), and BMI and MUAC ($\rho = 0.491$). These relationships indicate substantial internal consistency among the anthropometric indicators, while also showing that the individual measures capture related but not identical aspects of body-size and adiposity distribution.

A similarly structured pattern was observed within the inflammatory domain. Total leukocyte count and absolute neutrophil count showed the strongest positive correlation ($\rho = 0.901$), while ESR correlated positively with both leukocytes ($\rho = 0.741$) and neutrophils ($\rho = 0.703$). NLR was positively associated with neutrophil count ($\rho = 0.536$) and showed a strong inverse association with lymphocyte count ($\rho = -0.877$), consistent with its mathematical construction from these two leukocyte components.

In contrast, the cells linking the anthropometric and inflammatory domains were characterised predominantly by weak coefficients and inconsistent directions. The largest cross-domain relationships were observed between waist circumference and lymphocyte count ($\rho = 0.362$) and between waist circumference and NLR ($\rho = -0.350$), but neither association remained statistically significant after correction for multiple comparisons. BMI, MUAC, and WHtR likewise showed no consistent correlation pattern with ESR, leukocyte count, neutrophil count, lymphocyte count, or NLR.

The heatmap therefore illustrates a clear contrast between strong within-domain coherence and limited cross-domain correspondence. Anthropometric indicators correlated substantially with other measures of anthropometric adiposity, and inflammatory markers showed strong internal

relationships, whereas the correlations connecting the two domains were comparatively weak. This pattern supports the interpretation of anthropometric adiposity and systemic inflammatory response as related clinical characteristics that may nevertheless represent partially distinct biological dimensions in this neurological cohort.

The absence of robust cross-domain associations contrasted with the stronger within-domain correlations identified among the anthropometric measures themselves and among the inflammatory markers. This distinction is examined in the following section and provides a clearer representation of the internal structure of the two biological domains.

3.3. Internal correlation structure of the anthropometric and inflammatory domains

In contrast to the weak cross-domain associations, substantial internal correlations were observed within both the anthropometric and inflammatory domains.

Within the anthropometric domain, BMI showed a strong positive correlation with WHtR ($\rho = 0.860$, $p < 0.001$) and moderate correlations with waist circumference ($\rho = 0.542$, $p = 0.001$) and MUAC ($\rho = 0.491$, $p = 0.004$). Waist circumference was strongly associated with WHtR ($\rho = 0.749$, $p < 0.001$) and moderately associated with MUAC ($\rho = 0.561$, $p < 0.001$). MUAC was also positively correlated with WHtR ($\rho = 0.506$, $p = 0.003$).

A similarly coherent structure was observed within the inflammatory domain. Leukocyte and neutrophil counts were very strongly correlated ($\rho = 0.901$, $p < 0.001$). ESR correlated positively with both leukocyte count ($\rho = 0.741$, $p < 0.001$) and neutrophil count ($\rho = 0.703$, $p < 0.001$). NLR was positively associated with neutrophil count ($\rho = 0.536$, $p = 0.003$) and strongly inversely associated with lymphocyte count ($\rho = -0.877$, $p < 0.001$).

The correlation structure therefore demonstrated substantial convergence among measurements belonging to the same biological domain, whereas correlations between anthropometric and inflammatory domains were comparatively weak.

3.4. Systemic inflammatory profile across BMI categories

To further examine whether differences in overall adiposity were accompanied by differences in systemic inflammatory status, the principal inflammatory markers were compared across the three predefined BMI categories: normal weight, overweight, and obesity. This categorical analysis complemented the continuous correlation framework by assessing whether clinically interpretable levels of general adiposity were associated with distinguishable inflammatory profiles.

No statistically significant between-group differences were identified for any of the five inflammatory markers examined (Table 4). ESR showed the greatest numerical difference across BMI categories, with a median value of 24.00 mm/h in the normal-weight group compared with 10.00 mm/h in both the overweight and obese groups; however, the overall difference did not reach statistical significance (Kruskal-Wallis $p = 0.250$). Given the small number of observations in the normal-weight category, this numerical difference should be interpreted cautiously.

Total leukocyte counts were remarkably similar across BMI categories, with median values of 8.10×10^9/L in the normal-weight group, 8.18×10^9/L in the overweight group, and 7.99×10^9/L in the obese group ($p = 0.990$). Absolute neutrophil counts likewise showed no systematic variation according to BMI status, with corresponding medians of 5.80, 6.48, and 5.50×10^9/L ($p = 0.861$).

A different numerical pattern was observed for lymphocyte count and NLR. Median lymphocyte count increased from 1.39×10^9/L in the normal-weight group to 1.53×10^9/L among overweight observations and 2.39×10^9/L among obese observations, although the overall difference was not statistically significant ($p = 0.399$). Conversely, median NLR decreased across the same BMI categories, from 4.37 in the normal-weight group to 3.29 in the overweight group and 2.52 in the obese group. This apparent gradient was also not statistically supported by the Kruskal-Wallis test ($p = 0.243$).

Importantly, the direction of the observed NLR pattern did not indicate progressively greater systemic inflammatory activity with increasing BMI. Instead, the highest median NLR occurred in the normal-weight category and the lowest in the obesity category. In the context of the small and unequally distributed BMI groups, particularly the limited number of normal-weight observations, these values should be regarded as descriptive rather than evidence of an inverse biological relationship between BMI and inflammation (Table 4).

Table 4. Inflammatory characteristics according to BMI category

Marker	Normal weight	Overweight	Obesity	Kruskal-Wallis p
ESR, median	24.00	10.00	10.00	0.250
Leukocytes, median	8.10	8.18	7.99	0.990
Neutrophils, median	5.80	6.48	5.50	0.861
Lymphocytes, median	1.39	1.53	2.39	0.399
NLR, median	4.37	3.29	2.52	0.243

Source: Authors' calculation using Python 3.14.7 version

After accounting for the multiple inflammatory outcomes examined, none of the between-category comparisons provided evidence of an association between BMI classification and systemic inflammatory status. These categorical findings were therefore concordant with the preceding continuous-variable analyses: although excess body weight was highly prevalent in the cohort, increasing anthropometric adiposity was not accompanied by a consistently higher inflammatory profile.

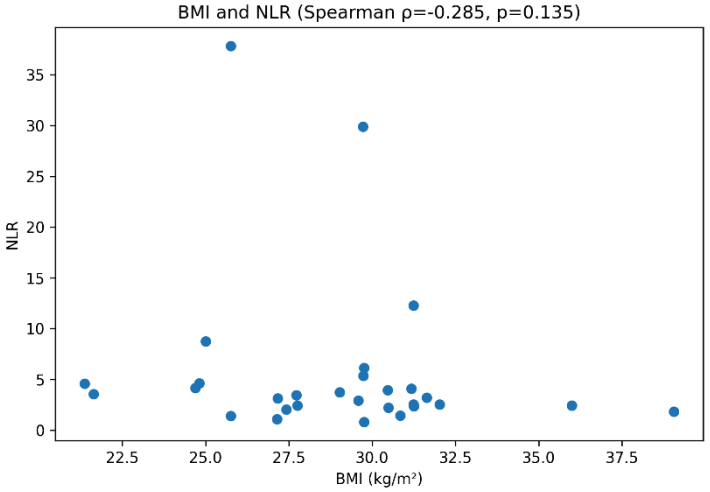

Figure 3. Distribution of neutrophil-to-lymphocyte ratio according to BMI category.
Source: Authors' contribution using Python 3.14.7 version

Figure 3 illustrates the distributional heterogeneity of NLR within the three BMI categories. Although the median NLR decreased numerically from the normal-weight to the overweight and obese groups, substantial within-group variability and overlap were evident. The distribution was additionally influenced by several markedly elevated NLR observations, particularly outside the central range of the data. Consequently, the apparent ordering of group medians did not translate into statistically distinguishable distributions (Kruskal-Wallis p = 0.243). The visual pattern therefore reinforces the absence of a clear BMI-dependent inflammatory gradient in this cohort and supports the interpretation of BMI-defined adiposity and NLR-defined systemic inflammatory response as non-equivalent clinical dimensions.

3.5. Robustness of NLR Associations and Clinical Comparison According to Cerebrovascular Ischaemic-Event Status

Given the marked right-skewness of the NLR distribution and the presence of several unusually high values, additional analyses were performed to determine whether the principal

anthropometric-inflammatory associations were disproportionately influenced by extreme observations. This assessment was particularly relevant because NLR ranged from 0.81 to 37.82 in the analytical sample, whereas its median was 3.20 [IQR: 2.35-4.57], indicating that a limited number of high values contributed substantially to the overall variability of this marker.

Using the conservative criterion of Q3 + 3 × IQR, with Q3 = 4.57 and IQR = 2.22, values exceeding 11.23 were considered extreme for sensitivity-analysis purposes. Three observations met this criterion, with NLR values of approximately 12.28, 29.88, and 37.82. The principal Spearman analyses were therefore repeated both after exclusion of all observations above this threshold and after exclusion of only the single highest NLR value.

The direction of the principal anthropometric-NLR associations remained stable across these alternative analytical specifications. In the complete dataset, BMI showed an inverse but non-significant association with NLR (ρ = -0.285, p = 0.135), while the corresponding association for waist circumference was somewhat stronger (ρ = -0.350, p = 0.063). After exclusion of observations with NLR >11.23, the coefficients remained inverse for both BMI (ρ = -0.334, p = 0.095) and waist circumference (ρ = -0.363, p = 0.068). Similarly, exclusion of only the highest NLR observation produced coefficients of ρ = -0.232 (p = 0.235) for BMI and ρ = -0.288 (p = 0.138) for waist circumference. Thus, neither approach to extreme-value exclusion produced a positive anthropometric-NLR association or materially changed the overall pattern observed in the primary analysis.

The distribution underlying the strongest anthropometric association with NLR is illustrated in Figure 4. Waist circumference was selected for graphical presentation because it showed the largest absolute Spearman coefficient with NLR among the four anthropometric indicators evaluated.

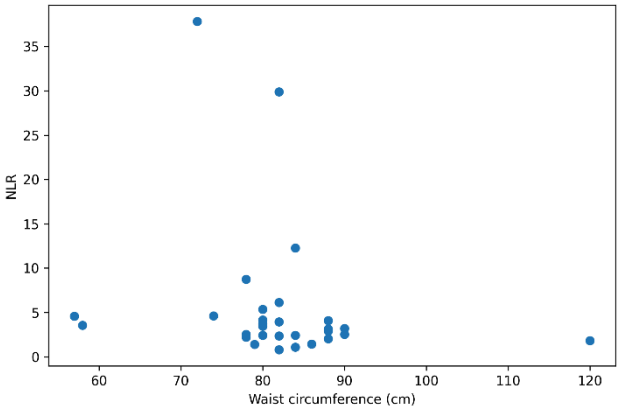

Figure 4. Association between waist circumference and neutrophil-to-lymphocyte ratio in the analytical cohort.

Source: Authors' contribution using Python 3.14.7.

Figure 4 illustrates the substantial heterogeneity of NLR across the observed range of waist circumference. Most observations were concentrated at relatively low NLR values, whereas a small number displayed markedly elevated ratios. Importantly, the highest NLR observations occurred at intermediate rather than at the largest waist circumferences, while the observation with the greatest waist circumference showed a comparatively low NLR value. The scatterplot therefore does not indicate a consistent monotonic increase in NLR with increasing central adiposity. Instead, the visual pattern is compatible with the moderate inverse rank correlation observed in the complete sample (ρ = -0.350, p = 0.063) and with the stability of its direction following exclusion of extreme NLR values. Accordingly, the sensitivity analyses indicate that the absence of a positive waist circumference-NLR relationship was not attributable solely to a small number of extreme inflammatory observations.

As a complementary analysis, the principal anthropometric–NLR relationships were further examined after adjustment for age and sex. Separate parsimonious models were fitted for each

standardised anthropometric indicator, with natural-log-transformed NLR as the dependent variable. Adjustment did not materially alter the pattern observed in the unadjusted analyses. None of the anthropometric indicators showed a statistically significant independent association with log-transformed NLR: BMI (β = -0.153; 95% CI: -0.402 to 0.096; p = 0.228), MUAC (β = -0.001; 95% CI: -0.495 to 0.493; p = 0.997), waist circumference (β = -0.217; 95% CI: -0.455 to 0.022; p = 0.075), and WHtR (β = -0.161; 95% CI: -0.434 to 0.113; p = 0.249). Waist circumference yielded the largest inverse adjusted coefficient, consistent in direction with the unadjusted Spearman association (ρ = -0.350; p = 0.063), although the confidence interval included zero. These complementary models therefore provided no evidence that the absence of a positive anthropometric–NLR association was explained by differences in age or sex. Given the limited sample size, the adjusted estimates were interpreted as supportive rather than confirmatory.

A separate component of the analysis examined whether anthropometric and inflammatory characteristics differed according to the presence of a documented cerebrovascular ischaemic event. Diagnostic classification identified 12 observations with explicitly or procedurally supported ischaemic stroke and two with transient ischaemic attack (TIA), resulting in 14 observations included in the primary cerebrovascular ischaemic-event (CIE) group. Four additional observations were classified as stroke of undetermined type, two as primary haemorrhagic stroke, and 12 as other neurological diseases. To preserve aetiological specificity and reduce potential misclassification, the four unspecified strokes and two haemorrhagic strokes were excluded from the primary CIE versus other-neurological-disease comparison.

The resulting principal clinical comparison therefore involved 14 observations with confirmed CIE and 12 with other neurological diseases. No statistically significant between-group differences were identified for the anthropometric indicators examined. Median BMI was 30.13 kg/m² in the CIE group compared with 28.40 kg/m² in the other neurological disease group (p = 0.237; Cliff's δ = 0.280). Although the direction of the effect estimate indicated somewhat higher BMI values in the CIE group, its magnitude was modest and the distributions were not statistically distinguishable. Median MUAC was identical at 18.00 cm in both groups (p = 0.979; δ = -0.012). Waist circumference also showed no evidence of group separation, with median values of 80.00 cm and 83.00 cm, respectively (p = 0.552; δ = -0.143), while WHtR was nearly identical between groups (p = 0.938; δ = -0.024).

A similar pattern was observed for the systemic inflammatory markers included in the comparison. Among observations with available inflammatory measurements, median NLR was 2.93 in the CIE group and 3.93 in the other neurological disease group. Despite the numerically higher median in the latter group, the difference was not statistically significant (p = 0.247), with Cliff's δ = -0.287 indicating only modest distributional separation. Median ESR was 12.00 mm/h among CIE observations and 10.00 mm/h among those with other neurological diseases, with little evidence of between-group differentiation (p = 0.861; δ = 0.049). The principal comparative results are summarised in Table 5.

Table 5. Principal anthropometric and inflammatory characteristics according to cerebrovascular ischaemic-event status

Variable	CIE	Other neurological disease	p	Cliff's δ
BMI, median, kg/m²	30.13	28.40	0.237	0.280
MUAC, median, cm	18.00	18.00	0.979	-0.012
Waist circumference, median, cm	80.00	83.00	0.552	-0.143
WHtR, median	0.484	0.486	0.938	-0.024
NLR, median	2.93	3.93	0.247	-0.287
ESR, median, mm/h	12.00	10.00	0.861	0.049

Note: Between-group comparisons were performed using the Mann-Whitney U test. Cliff's δ is reported as a non-parametric measure of effect size; positive values indicate a tendency toward higher values in the CIE group and negative values towards higher values in the other neurological disease group.

Source: Authors' calculation using Python 3.14.7 version

The distribution of NLR in the two diagnostic groups provides additional information beyond the comparison of group medians and is presented in Figure 5.

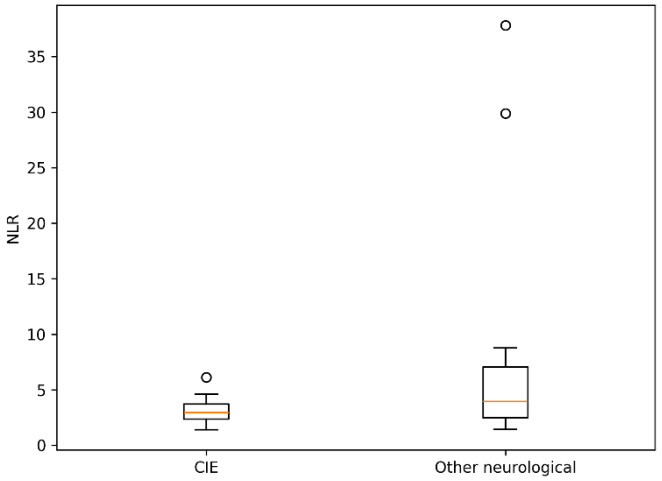

Figure 5. Distribution of neutrophil-to-lymphocyte ratio according to cerebrovascular ischemic-event status.

Source: Authors' contribution using Python 3.14.7.

As shown in Figure 5, the central portions of the NLR distributions overlapped substantially between the CIE and other neurological disease groups. The CIE group displayed a relatively compact distribution, with most observations concentrated within a narrow range and only one moderately elevated value. In contrast, the other neurological disease group showed considerably greater dispersion and contained the two highest NLR observations, approaching values of 30 and 38. This pronounced upper-tail variability explains why the arithmetic distribution of NLR appears markedly different between groups despite the absence of statistically supported separation when assessed using the rank-based Mann-Whitney test.

The graphical pattern is also consistent with the estimated Cliff's δ of -0.287, which suggests a modest tendency towards higher NLR values in the other neurological disease group rather than in the CIE group. However, the substantial overlap between the central distributions and the non-significant between-group comparison (p = 0.247) indicate that NLR did not clearly distinguish confirmed ischaemic cerebrovascular events from other neurological presentations in this sample.

Taken together, the sensitivity and diagnostic-group analyses reinforce the principal findings obtained from the primary correlation framework. The inverse associations of BMI and waist circumference with NLR were not reversed by removal of extreme NLR observations, indicating that the primary cross-domain findings were not dependent on a small number of highly elevated inflammatory values. At the same time, neither anthropometric measures nor NLR demonstrated clear distributional separation between confirmed CIE and other neurological diseases. The results therefore support a pattern of substantial individual heterogeneity in systemic inflammatory response, without evidence in this dataset of a consistent positive relationship between greater anthropometric adiposity and NLR or of a distinct NLR distribution characterising CIE.

4. Discussion

The present study explored the relationship between anthropometric adiposity, systemic inflammatory response, and cerebrovascular ischaemic events in a heterogeneous neurological population. The principal finding was not a simple positive adiposity–inflammation gradient, but rather a relative dissociation between these two domains. Although excess body weight was highly prevalent in the cohort, BMI, MUAC, waist circumference, and WHtR showed predominantly weak and inconsistent associations with ESR, leukocyte count, neutrophil count, lymphocyte count, and

NLR. None of the cross-domain correlations remained statistically significant after correction for multiple comparisons. These findings suggest that anthropometric adiposity and routinely measured systemic inflammatory activity should not be interpreted as interchangeable components of the same clinical phenotype.

This result requires interpretation within the established biology of obesity. Adipose tissue is metabolically and immunologically active, and its expansion and dysfunction can alter the secretion of adipokines and inflammatory cytokines, contributing to insulin resistance, endothelial dysfunction, and vascular disease (Ruan & Lodish, 2004; Geng et al., 2023). Visceral adiposity may be particularly relevant because its inflammatory activity has been associated with vascular and cerebral consequences in both experimental and clinical research (Shin et al., 2015; Suárez-Cuenca et al., 2019). Nevertheless, the biological presence of obesity-related inflammation does not imply that routinely measured circulating leukocyte indices must increase proportionally with BMI or other anthropometric measurements.

The present findings concerning NLR are particularly informative in this regard. Previous clinical studies have produced heterogeneous results concerning the relationship between obesity and NLR. Bahadir et al. (2015), in a substantially larger population, found that leukocyte and neutrophil counts and hs-CRP increased with BMI, whereas NLR neither differed significantly between BMI categories nor correlated with BMI. This distinction suggests that individual inflammatory components may respond to adiposity without producing a corresponding linear change in their ratio. Conversely, Suárez-Cuenca et al. (2019) reported positive relationships between NLR and inflammatory mediators, visceral adipose tissue, adipocyte area, and carotid intima-media thickness in individuals with obesity. Thus, the association between anthropometric adiposity and NLR appears to depend on population characteristics, the adiposity measure considered, and the biological context in which NLR is assessed.

This heterogeneity provides an appropriate context for the present results. BMI was inversely, rather than positively, associated with NLR, although the relationship was not statistically significant. Waist circumference showed a somewhat stronger inverse relationship with NLR, while MUAC and WHtR demonstrated only weak associations. These results should therefore not be interpreted as evidence against the inflammatory biology of obesity. Rather, they indicate that NLR may not function as a direct linear surrogate of anthropometric adiposity in a heterogeneous neurological population. Similar complexity has been observed in ischaemic stroke, where obesity may influence several inflammatory pathways simultaneously rather than producing a uniform increase in individual circulating biomarkers (Rodríguez-Castro et al., 2019; Ruhnau et al., 2023).

The correlation structure observed in the present study reinforces this interpretation. Anthropometric indicators were strongly or moderately interrelated, including a strong relationship between BMI and WHtR and substantial associations among BMI, waist circumference, MUAC, and WHtR. The inflammatory domain also displayed considerable internal coherence, particularly through the strong leukocyte–neutrophil relationship and the expected positive association of NLR with neutrophil count and inverse association with lymphocyte count. In contrast, correlations linking anthropometric and inflammatory variables were considerably weaker and inconsistent in direction.

This distinction between strong within-domain relationships and weak cross-domain correspondence may be more informative than any individual correlation coefficient. BMI, waist circumference, WHtR, and MUAC characterise related but non-identical aspects of body size and adiposity, whereas leukocyte-derived indices characterise a dynamic systemic immune state. Evidence from population studies likewise indicates that different measures of adiposity do not necessarily provide equivalent information regarding cardiometabolic and cerebrovascular risk (Meulmeester et al., 2023; Morys et al., 2021). Accordingly, anthropometric and inflammatory measures should be considered complementary rather than redundant markers.

The categorical BMI analysis produced a consistent pattern. Median NLR decreased numerically from the normal-weight group to the overweight and obesity groups, but the difference

was not statistically significant. ESR, leukocyte count, neutrophil count, and lymphocyte count likewise showed no significant differences across BMI categories. This finding is particularly compatible with the results reported by Bahadir et al. (2015), who observed no significant NLR differences across BMI groups despite evidence of obesity-related changes in other inflammatory markers. Therefore, the numerical decline in NLR observed in the present cohort should not be interpreted as evidence of an anti-inflammatory effect of increasing adiposity. The small number of normal-weight observations and substantial within-group variability preclude such an interpretation.

The divergence between NLR and anthropometric adiposity may partly reflect differences in biological timescale. BMI, waist circumference, WHtR, and MUAC represent relatively stable characteristics of body size and fat distribution, whereas circulating neutrophils and lymphocytes may change rapidly in response to acute disease, physiological stress, infection, medication, and tissue injury. This distinction is especially important in neurological patients because cerebral ischemia itself initiates a complex systemic inflammatory response involving leukocyte activation, platelet–neutrophil interactions, cytokine signalling, and subsequent immune modulation (Ansari & Gavins, 2021; Abuzan et al., 2026). Consequently, a single NLR measurement may reflect the immediate neurological and systemic state more strongly than the chronic low-grade inflammatory burden associated with adiposity.

The distribution of NLR in the present cohort supports this interpretation. NLR was markedly right-skewed and included several extreme observations. However, sensitivity analyses showed that these values did not account for the direction of the adiposity–NLR relationships. After exclusion of the extreme NLR observations, the associations of both BMI and waist circumference with NLR remained inverse. Therefore, the principal pattern cannot readily be attributed to a small number of patients with unusually high inflammatory values.

Waist circumference deserves particular attention because it produced the largest cross-domain coefficients in the study. Central adiposity is generally considered more metabolically informative than BMI alone, and measures reflecting visceral fat distribution have been associated with inflammation, atherosclerotic burden, and cerebrovascular risk (Li et al., 2020; Suárez-Cuenca et al., 2019; Xiao et al., 2025). Suárez-Cuenca et al. (2019), for example, demonstrated relationships between NLR and directly assessed visceral adipose tissue as well as carotid intima-media thickness. The present study did not reproduce such a positive association using waist circumference as an anthropometric proxy. Instead, waist circumference was positively associated with lymphocyte count and inversely associated with NLR, although neither relationship remained statistically supported after multiple-comparison correction.

This apparent discrepancy may partly reflect differences between anthropometric proxies and direct measurements of visceral adipose tissue. Waist circumference approximates central adiposity but cannot distinguish visceral from subcutaneous abdominal fat or quantify the biological activity of adipose tissue. More sophisticated indices combining adiposity and inflammatory or metabolic information have consequently been proposed for cerebrovascular risk assessment (Li et al., 2020; Xiao et al., 2025). The current findings therefore reinforce the need to distinguish simple anthropometric central adiposity from metabolically and immunologically active visceral adiposity.

The cerebrovascular analysis provides an additional perspective. The primary comparison restricted cerebrovascular ischaemic events (CIE) to documented ischaemic stroke and transient ischaemic attack, thereby avoiding the biological heterogeneity that would result from combining ischaemic and haemorrhagic cerebrovascular conditions. BMI was numerically higher in the CIE group than in patients with other neurological diseases, but the difference was not statistically significant. MUAC, waist circumference, and WHtR similarly failed to differentiate the two diagnostic groups.

These findings do not contradict the established relationship between adiposity and long-term cerebrovascular risk. Large observational studies have associated obesity and measures of body-fat distribution with cerebrovascular disease, while contemporary work increasingly emphasizes visceral adiposity and metabolic phenotype rather than BMI alone (Qin et al., 2015;

Haberka et al., 2023; Xiao et al., 2025). Instead, the present results indicate that anthropometric measurements obtained in a small clinical neurological cohort do not necessarily discriminate patients currently presenting with ischaemic cerebrovascular events from those with other neurological disorders.

The interpretation of NLR requires a similar distinction between risk, diagnosis, and prognosis. NLR has been extensively investigated as an inflammatory marker in cerebrovascular disease. In acute ischaemic stroke, higher NLR has been associated with carotid plaque vulnerability and atherosclerotic instability (Li et al., 2021), while other studies have related elevated NLR and related systemic inflammatory indices to greater disease severity and adverse clinical outcomes (Lin et al., 2024; Liu et al., 2025; Xu et al., 2025). These findings support the clinical relevance of NLR in cerebrovascular disease but do not establish it as a disease-specific diagnostic marker.

This distinction is directly relevant to the present study. Median NLR was lower in the CIE group than in the other-neurological-disease group, and the between-group difference was not statistically significant. Most previous stroke studies have evaluated NLR within populations already diagnosed with cerebrovascular disease and have asked whether inflammatory status is associated with severity, plaque vulnerability, mortality, or functional outcome (Li et al., 2021; Lin et al., 2024; Liu et al., 2025). The present analysis addressed a different question: whether NLR differentiates documented ischemic cerebrovascular presentations from a heterogeneous group of other neurological disorders. The absence of such differentiation is therefore not inconsistent with the prognostic literature.

Indeed, the substantial overlap in NLR distributions between the diagnostic groups illustrates the limited specificity of this marker. NLR represents the balance between circulating neutrophils and lymphocytes and may be altered by numerous inflammatory, infectious, metabolic, vascular, and stress-related processes. In the neurological setting, inflammatory responses may also vary according to disease type, severity, timing, and treatment. Consequently, NLR is more appropriately regarded as a context-dependent marker of systemic inflammatory status than as an isolated discriminator of cerebrovascular ischemia (Ansari & Gavins, 2021; Abuzan et al., 2026).

The study also failed to identify a simple sequential relationship in which greater adiposity was accompanied by higher NLR and subsequently by a greater prevalence of CIE. Such a pathway is biologically plausible because obesity, chronic inflammation, endothelial dysfunction, atherosclerosis, and cerebrovascular disease are interconnected processes (Ruan & Lodish, 2004; Morys et al., 2021; Haberka et al., 2023). However, the empirical findings did not support a simple linear representation of this pathway. BMI and waist circumference showed inverse rather than positive associations with NLR, inflammatory markers did not differ significantly across BMI categories, and neither anthropometric measures nor NLR clearly distinguished CIE from other neurological diseases.

A more plausible interpretation is that adiposity, systemic inflammation, and cerebrovascular disease interact across different biological timescales. Anthropometric adiposity reflects cumulative exposure developing over months or years, while leukocyte-derived inflammatory indices may vary over hours or days. Cerebrovascular ischemia can itself modify systemic immune activity, further complicating cross-sectional relationships between pre-existing adiposity and inflammatory markers (Haley & Lawrence, 2016; Haley et al., 2017; Ruhnau et al., 2023). Therefore, measurements obtained during neurological evaluation may simultaneously capture chronic metabolic burden and acute disease-related inflammatory responses.

These findings have practical implications for clinical assessment. BMI, waist circumference, WHtR, and MUAC are inexpensive and rapidly obtainable measures that characterise different aspects of anthropometric status. NLR is similarly accessible because it can be calculated from routine complete blood counts. Their clinical usefulness does not depend on their being strongly correlated. The weak cross-domain relationships observed here suggest that anthropometric and inflammatory measures may provide non-redundant information.

Anthropometric indicators may characterise longer-term metabolic and vascular-risk burden, whereas NLR may provide information regarding a more immediate systemic inflammatory state.

Accordingly, neither domain should be interpreted in isolation. Assessment integrating anthropometry, routine inflammatory indices, metabolic risk factors, neurological severity, and neuroimaging may provide a more clinically meaningful characterization of cerebrovascular risk and disease status. This multidimensional perspective is consistent with emerging approaches that combine visceral adiposity and inflammatory information to improve stroke-risk assessment rather than relying on a single anthropometric or inflammatory indicator (Li et al., 2020; Xiao et al., 2025).

Several methodological features strengthen the present exploratory analysis. Multiple anthropometric indicators were considered rather than BMI alone, permitting evaluation of overall body size, central adiposity, and peripheral anthropometry. BMI and NLR were independently recalculated from their constituent variables as part of data-quality control. Rank-based methods were used for non-normal distributions, multiple cross-domain tests were controlled for false discovery, effect sizes accompanied group comparisons, and sensitivity analyses explicitly examined the influence of extreme NLR observations. In addition, missing values were not imputed, and the diagnostic classification distinguished documented ischemic events from haemorrhagic and aetiologically undetermined strokes.

Nevertheless, the findings should be interpreted within several limitations. The principal limitation is the small sample size. The analytical dataset contained 32 clinical monitoring observations, while the principal inflammatory analyses included 29 observations because of missing laboratory measurements. Statistical power was therefore limited, particularly for subgroup comparisons, and the normal-weight BMI category was small. Consequently, the absence of statistical significance cannot be interpreted as evidence of biological equivalence or proof that no underlying relationship exists.

The retrospective and single-center design represents a further limitation. The comparison group consisted of patients with heterogeneous neurological disorders rather than healthy controls, and these conditions may themselves influence systemic inflammatory activity. This characteristic is clinically realistic but makes the comparison fundamentally different from studies contrasting patients with obesity or stroke against healthy populations. The present findings should therefore be interpreted as reflecting inflammatory differentiation within a neurological clinical population.

Temporal information was also insufficiently standardised. NLR and other leukocyte-derived markers may vary according to the interval between symptom onset, hospital presentation, treatment, infection, physiological stress, and blood collection. Previous stroke research demonstrates that immune responses evolve dynamically during the early post-stroke period and may also differ according to BMI (Ruhnau et al., 2023). Detailed neuroimaging characteristics, infarct volume, vascular territory, and standardised timing from symptom onset were not consistently available and could not therefore be incorporated into the analysis.

The metabolic profile was similarly incomplete. Total cholesterol, LDL-C, triglycerides, diabetes status, and other cardiometabolic variables were not uniformly available and could not be incorporated as mandatory covariates without substantially reducing the effective sample size. This is relevant because adiposity may influence cerebrovascular disease through multiple intermediate pathways, including dyslipidemia, hypertension, insulin resistance, and systemic inflammation (Morys et al., 2021; Meulmeester et al., 2023). Residual confounding by metabolic status, medication, infection, smoking, and other vascular-risk factors therefore remains possible.

Finally, anthropometric measures are proxies rather than direct measurements of adipose-tissue quantity, distribution, or biological activity. BMI cannot distinguish adipose from lean mass, MUAC incorporates several tissue compartments, and waist circumference and WHtR approximate rather than directly quantify visceral fat. This limitation is particularly relevant given evidence that directly assessed visceral adiposity may relate to inflammatory activity and vascular

pathology even when simpler anthropometric measures provide incomplete information (Suárez-Cuenca et al., 2019; Xiao et al., 2025).

Future research should therefore move beyond simple bivariate adiposity–NLR relationships. Larger prospective cohorts should incorporate standardised timing of blood sampling, repeated inflammatory measurements, stroke severity measures such as NIHSS, functional outcomes, detailed neuroimaging, reperfusion treatment, infection status, diabetes and glycaemic control, lipid profiles, medication exposure, smoking status, and more direct measures of body composition. Longitudinal approaches would be particularly valuable for distinguishing chronic adiposity-related inflammatory burden from the acute systemic immune response associated with cerebrovascular injury.

Overall, the present study should be regarded as hypothesis-generating rather than confirmatory. Its principal contribution is the observation that, within a real-world neurological cohort characterised by a high prevalence of overweight and obesity, anthropometric adiposity and routinely measured systemic inflammatory activity did not behave as interchangeable biological constructs. The strong internal coherence of the anthropometric and inflammatory domains, combined with weak cross-domain relationships, stable sensitivity findings, and the absence of clear discrimination of CIE, supports a multidimensional interpretation of the relationship among adiposity, inflammation, and cerebrovascular disease.

5. Conclusions

This exploratory study indicates that anthropometric adiposity and systemic inflammatory response should not be interpreted as interchangeable dimensions in neurological disease. Although excess body weight was highly prevalent in the analysed cohort, with 87.5% of observations classified as overweight or obese, BMI, MUAC, waist circumference, and WHtR did not show a consistent positive relationship with ESR, leukocyte count, neutrophil count, lymphocyte count, or NLR. None of the anthropometric-inflammatory associations remained statistically significant after false-discovery-rate correction.

The correlation structure showed stronger coherence within each biological domain than between them. Anthropometric indicators correlated substantially with one another, while inflammatory markers also displayed strong internal relationships, particularly among leukocytes, neutrophils, lymphocytes, ESR, and NLR. In contrast, cross-domain associations were comparatively weak and inconsistent in direction. This pattern supports the interpretation that anthropometric adiposity and systemic inflammatory activity capture complementary, but only partially overlapping, aspects of the clinical phenotype.

The categorical BMI analysis led to the same conclusion. Despite the high prevalence of overweight and obesity, inflammatory markers did not differ significantly across normal-weight, overweight, and obese groups. Median NLR decreased numerically across these categories, but the difference was not statistically significant and should not be interpreted as evidence of an inverse biological relationship between adiposity and inflammation.

The robustness analyses further showed that the principal findings were not driven by a small number of extreme NLR values. Excluding the highest NLR observation or all values above the conservative Q3 + 3 × IQR threshold did not change the direction of the BMI-NLR or waist circumference-NLR associations.

Similarly, neither the anthropometric indicators nor the principal inflammatory markers clearly differentiated documented cerebrovascular ischaemic events from other neurological diseases in the primary exploratory comparison. NLR showed substantial within-group heterogeneity and considerable overlap between diagnostic groups, indicating that it should not be interpreted as an isolated discriminator of cerebrovascular ischaemia in this cohort.

Taken together, these findings suggest that simple anthropometric measurements and routine inflammatory indices provide non-redundant clinical information rather than representing sequential markers of a single adiposity-inflammation-ischaemia pathway. In neurological patients, adiposity

appears to reflect a more stable anthropometric and cardiometabolic dimension, whereas NLR and related haematological markers reflect a more dynamic systemic inflammatory state. Their clinical interpretation should therefore remain contextual and integrated with neurological diagnosis, vascular-risk factors, laboratory data, and disease severity.

Given the exploratory design and limited sample size, these results should be regarded as hypothesis-generating. Larger prospective studies with standardised timing of blood sampling, repeated inflammatory measurements, detailed stroke phenotyping, neuroimaging, metabolic profiling, medication exposure, smoking status, and validated neurological outcome measures are needed to clarify how chronic adiposity-related risk interacts with acute systemic inflammation and cerebrovascular disease.

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