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Association between Systemic Immune-Inflammation Index, Body Composition, and Mortality among Older Adults

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ABSTRACT

Introduction: Chronic inflammation in older adults is associated with various age-related diseases and may contribute to functional decline and reduced quality of life. An imbalance in body composition, characterized by excess fat and inadequate muscle mass levels, has been identified as an underlying cause of inflammation. Numerous prognostic factors, such as indicators, have been employed to measure inflammation. The Systemic Immune-Inflammation Index (SII) is a reliable indicator of inflammation that correlates with mortality in numerous investigations substantially. This paper aims to examine the relationship between body composition, mortality, and SII.

Methods: This cross-sectional study was conducted on 60 years old or older adults using the Neyshabur Longitudinal Study on Ageing (NeLSA) data. SII scores were calculated using data from individual blood bank records. Data analysis involved analytical techniques such as correlation coefficient, logistic regression, and linear regression.

Results: A total of 3,534 individuals participated, of whom 1,858 were male. The median age of participants was 65.71. The study revealed a significant association between the percentage of body fat and SII ($p<0.001$). The overall mortality rate was 0.93 in 1000. Mortality was linked to SII after adjusting for confounding variables (OR=1.001, 95% CI=1.000 to 1.002, P=0.047).

Conclusion: SII only correlated with blood pressure and body fat. A weak correlation was observed between SII and hs-CRP, which was associated with overall mortality in older adults.

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Introduction

Aging is defined differently by various researchers, and older adults are defined as 50–80 years old. However, the World Health Organization (WHO) classifies people as elderly when they are 65 years or older [1]. The global aging population is increasing, with over 500 million people, or 8% of the world's population, over 65 [2, 3]. The National Institute on Aging and the United States National Institute of Health predict a quadrupling of adults over 80 by 2050 [4]. Around one-fifth of the population will be over 60 by the middle of this century [5]. This demographic transition affects individuals and groups globally, with 30% of individuals who are 80 or older living alone [6]. The aging population and labor unavailability cause economic challenges, with lower national income expansion, impacting elderly socioeconomic status through tax-transfer systems and the dissatisfaction of older workers due to delayed retirement [7]. Older adults often suffer from multifaceted diseases such as back and neck pain, inflammation, dementia, chronic obstructive pulmonary disease, diabetes, and osteoarthritis, which often require multiple drug treatments [8,

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9]. Older adults frequently face challenges in their independence due to physical and mental limitations, which cause feelings of isolation and a lack of social assistance [10]. The decline in the quality of life of older adults due to diseases necessitates public health to address healthcare needs and develop products and services tailored to the specific needs of older adults [11].

Significant changes in body composition, such as an increase in body fat percentage and a decrease in lean mass and bone density, are related to aging. These variations can affect the onset of chronic diseases and unfavorable health outcomes. Several factors, including hormone imbalance, ongoing inflammation, and metabolic changes, can influence the onset and progression of chronic diseases [12]. Inflammation is a defense mechanism against harmful assaults and is vital for healing injured tissue and eliminating harmful stimuli. Acute inflammation is related to innate immunity and the host's initial defense against chemicals and foreign invaders. New research has suggested its functions as a complex molecular system [13]. In contrast, chronic inflammation is related to various age-related diseases like cancer, diabetes, atherosclerosis, and hypertension.

Various techniques are used to measure inflammation. The overall inflammation status of the body can be measured using inflammation markers in addition to erythrocyte sedimentation rate (ESR) and high sensitivity C-reactive protein (hs-CRP). Inflammation indicators include neutrophil-to-lymphocyte ratios (NLR), platelet-to-lymphocyte ratios (PLR), and systemic immune-inflammation indexes (SII). SII is a quantitative, accessible, and affordable measurement of the systemic immune-inflammatory response in the human body. SII has been related to poor prognosis in malignant tumors and an increased risk of all-cause, cardiovascular, and cancer-related mortality [14, 15].

The association between aging and chronic diseases such as diabetes [16], cancer [17], and heart disease [15] has been the subject of several studies. Research has suggested that higher levels of oxidative stress and inflammation may be related to increased mortality in older adults. However, there is a lack of evidence and insufficient data regarding this relationship in older adults, which requires population-based studies.

Chronic inflammation has many causes and mechanisms, and while lifestyle changes can help prevent these disorders, understanding the causes and mechanisms is crucial as healthy aging becomes more prevalent in developed and developing countries. This study was conducted on Iranian older adults because of the correlation between body composition and inflammation, the SII index's importance in predicting mortality, and the limited number of studies examining SII, body composition, and mortality among the general elderly population.

Materials & Methods

Study Population

Neyshabur Longitudinal Study on Ageing (NeLSA) is a large-scale and population-based study conducted in Neyshabour, Iran, among individuals between 50 and 94 years old [18]. NeLSA is intended to include 7460 subjects and assess various aspects of aging. Data collected in the NeLSA include demographic information, including sex, age, income, education level, and all causes of mortality and smoking, collected through interviews, blood tests, and comprehensive questionnaires, including physical activity.

Study design

This cross-sectional study used the NeLSA database. The Mashhad University of Medical Sciences Ethics Committee approved each procedure involving human subjects (IR.UMS.MEDICAL.REC.1401.325).

The inclusion criteria for NeLSA and the current study were as follows: (i) Having Iranian nationality, (ii) being older than sixty years, and (iii) willingness to participate in the cohort by signing the informed consent form. The exclusion criteria were those without fully documented information.

Study Measurements

Laboratory Measurements

The blood biochemistry data were the complete blood count (CBC) and serum high sensitivity-CRP (hs-CRP), which were measured in the registration & enrollment phase of NeLSA [18].

Body Composition Measurements

Body composition, including anthropometric and bioelectric impedance (BIA), were recorded. The BIA data were recorded using the InBody 770, BIOSPACE KOREA connected to a BSM. The BIA variables were total body water (TBW), percent

body fat (PBF), fat-free mass (FFM), skeletal muscle mass (SMM), visceral fat level (VFL), and visceral fat area (VFA).

Socioeconomic and Lifestyle

The participants' level of physical activity was estimated using the Physical Activity Scale for older adults (PASE), which has been appropriately confirmed in previous Iranian research [19]. Socioeconomic status indications included educational levels categorized as illiterate, less than a high school diploma, high school diploma, and university degree. The income adequacy of the respondent was assessed based on their financial status. Smoking status was determined using a self-report.

Past Medical History

A physician performed clinical examinations, took histories, and double-checked participants' medical records. The current study included chronic diseases, including diabetes, heart disease, and hypertension (HTN).

Mortality

All-cause mortality was obtained from the cohort records from the initiation of the study till the time the data were collected.

Systematic Inflammation Index (SII)

The total blood count results from tests were used to determine SII. In addition, platelet count (PC), neutrophil count (NC), and lymphocyte count (LC) were measured in 1000 cells/ml. Based on previous research, the SII was computed as PC * (NC/LC) [17, 20].

Statistical Analysis

The Statistical Package for Social Sciences (SPSS) software version 26 was used for data analysis. The Kolmogorov-Smirnov test was used to evaluate the normality of the data. Continuous variables were presented using Cromedian and interquartile range (IQR), while categorical variables were presented as percentages and frequencies. The Spearman correlation coefficient was used to evaluate the correlation between variables and identify the confounders. The relationship between the study variables and SII was examined using linear regression after adjusting for age, sex, educational attainment, smoking, income, and other medical conditions. Univariate models were performed first, and variables with p-values less than 0.1 were included in the final model. The multivariate regression analysis was performed using backward elimination. The logistic regression analysis assessed the relationship between mortality and study variables. A $P<0.05$ was considered significant in all statistical analyses.

Results

The current study was conducted on 3534 participants, including 1676 females (47.4%) and 1858 males (52.6%). The median age of the included participants was 65.71(62.17-71.83) years. Table 1 summarizes the anthropometric measurements of the participants. The education level of the majority of the participants was less than a high school diploma (46.1%).

The participants' median SII was 284.42 (203.74, 392.76), and their median serum hs-CRP was 0.2 (0.1, 0.5) mg/dl, respectively.

Table 2 presents the relationship between SII and study variables. The relationship was evaluated using five different adjusted models. SII was positively associated with HTN in the crude model (Model 1) (β=28.673, 95% CI=8.516 to 48.830, P=0.005). No significant relationship was found between SII and the body composition variables (PBF, VFL, and VFA), sex, or education. In Model 4 (adjusted for VFA, education, and VFL). SII was significantly related to HTN (β=28.809, 95% CI=8.696 to 48.923, P=0.005) and PBF (β=1.92, 95% CI=0.994 to 2.86, P<0.001). Furthermore, SII continued to be positively related to both PBF (β=1.922, 95% CI=0.987 to 2.857, P=0.00) and HTN (β=28.81, 95% CI=8.690 to 48.933, P=0.005) after adjustment for VFA, education, VFL, and sex.

Table 1. Body composition measurements of the study participants.

Variables	Median (Q_1-Q_3)
PBF	37.6 (30.3, 44.3)
FFM	42.4 (37.5, 49.5)
SMM	23 (20, 27.3)
TBW	31.3 (27.6, 36.6)
VFL	13 (9, 17)
VFA	135.6 (94.65, 174.25)

For non-normal distribution variables, the median and first and third quartiles were used. PBF, Percent Body Fat; FFM, Fat-Free Mass; SMM, Skeletal Muscle Mass; TBW, Total Body Water; VFL, Visceral Fat Level; VFA, Visceral Fat Area.

Table 2. Relationship between SII and study variables.

		Sex	PBF	VFL	VFA	HTN	Education
Model 1	**β (95%CI low-up)**	14.34 (-3.877, 32.56)	1.55 (-1.02, 4.13)	1.41 (-28.81, 31.64)	-0.68 (-3.12, 2.98)	28.67 (8.51, 48.83)	-0.43 (-11.52, 10.66)
	P-value	0.12	0.23	0.92	0.96	0.005*	0.93
Model 2	**β (95%CI low-up)**	14.34 (-3.87, 32.56)	1.54 (-1.01, 4.10)	0.74 (-3.89, 5.38)	-	28.68 (8.53, 48.83)	-0.43 (-11.52, 10.66)
	P-value	0.12	0.23	0.75		0.005*	0.93
Model 3	**β (95%CI low-up)**	14.53 (-2.99,32.97)	1.54 (-1.00,4.10)	0.744 (-3.89,5.38)	-	28.71 (8.58,48.84)	-
	P-value	0.10	0.23	0.75		0.005*	
Model 4	**β (95%CI low-up)**	14.60 (-2.91,32.13)	1.92 (0.994,2.86)	-	-	28.80 (8.69,48.92)	-
	P-value	0.10	<0.001*			0.005*	
Model 5	**β (95%CI low-up)**	-	1.92 (0.987,2.85)	-	-	28.81 (8.69,48.93)	-
	P-value		<0.001*			0.005*	

SII, Dependent variable. A significant difference is shown by P < 0.05

Model 1: Unadjusted model. ***Model 2:*** VFA was adjusted. ***Model 3:*** VFA and education were adjusted. ***Model 4:*** VFA, education, and VFL were adjusted. ***Model 5:*** VFA, education, VFL, and sex were adjusted.

PBF, Percent Body Fat; VFL, Visceral Fat Level; VFA, Visceral Fat Area; HTN, Hypertension; CI, confidence Interval

* Significant relationship at α=0.05.

Figure 1 illustrates the relationship between the SII and hs-CRP. There was a significant weekly positive relationship between SII and hs-CRP among the study participants (r=0.076, p=0.001). The mortality rate was 0.93 in 1000 (328 from 3534 persons). The crude model had a significant relationship between mortality and SII (OR=1.001, 95% CI=1.000 to 1.001, p=0.016). The relationship between SII and mortality remained significant after adjustment for income, education, sex, diabetes, heart disease, HTN, and heart disease (OR=1.001, 95% CI=1.000 to 1.002, P=0.047).

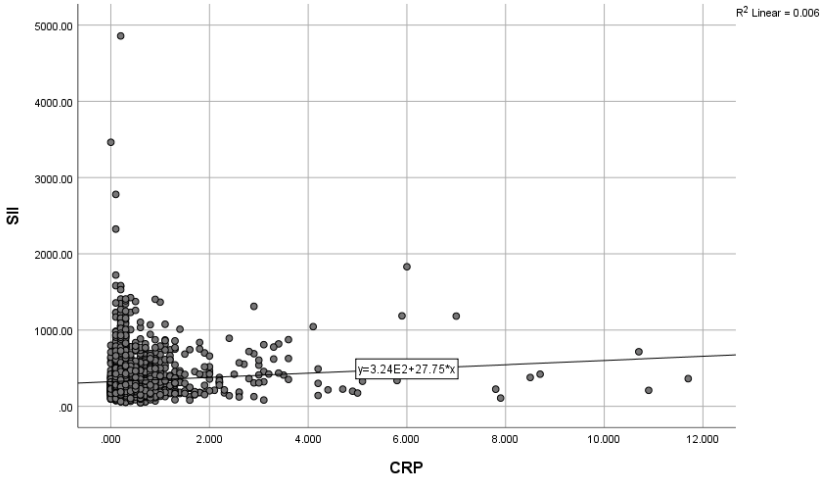

Figure 1. Relationship between high sensitivity C-reactive protein (hs-CRP) and systemic immune inflammation index (SII). A significant difference is shown by P < 0.05.

Discussion

The current study investigated the relationship between the immune systemic inflammation index (SII) and body composition among older adults. The higher body fat percentage and hypertension were associated with increased SII. The study also indicated that increased SII was related to an increased mortality risk, even after adjusting for confounders.

The direct association between SII and PBF was in line with the findings of the previous studies. Funghetto et al. reported that PBF categorization based on dual-energy X-ray absorptiometry (DEXA) and biochemical tests accurately predicted obesity, systemic inflammation, and atherogenic lipid profiles in older women than BMI [21]. According to another cross-sectional study using data from the National Health Survey on 8-18-year-old children and adolescents by

Singer et al. (2023), increased SII was associated with an increased likelihood of losing muscle mass, suggesting a strong association between inflammation and childhood obesity [22]. Eren et al. studied the association between childhood obesity and inflammatory mediators in children aged 6-16 and reported that increased SII was associated with increased body fat, waist circumference, and BMI [23]. In contrast to the current study's findings, Hestiantoro et al. reported that PBF was an accurate measure for assessing inflammation associated with body fat mass among women with polycystic ovarian syndrome (PCOS) [24]. This difference might be attributed to the difference in the study population between Hestiantoro et al. (only women with PCOS) and the present study (elderly men and women).

Different mechanisms have been proposed for the association between fat mass and inflammation among obese individuals. The low-grade chronic inflammation due to the increased adipose tissue might be, to some extent, due to the release of inflammatory mediators from adipose tissue. The buildup of aberrant or excess fat associated with obesity causes adipose tissue to emit inflammatory mediators, including CRP, tumor necrosis factor-alpha (TNF-α), and interleukin 6 (IL-6). The decrease in the synthesis of the anti-inflammatory adiponectin exacerbates the pro-inflammatory state and oxidative stress. The liver produces and secures CRP in response to increased IL-6, indicative of inflammation. Additionally, white adipose tissue (WAT) can cause inflammation in obesity through immune cell-adipocyte interactions, hypoxia, and increased adipocyte death [25]. The relationship between inflammation and body fat is primarily explained by the release of inflammatory mediators and the intricate interactions between immune cells and adipocytes [26]. Research has also been conducted on adipokines, including adiponectin, in the inflammatory processes associated with obesity. Adipose tissue secretes adipokines, and the dysregulation of adipokine synthesis can lead to inflammation and metabolic dysfunction in obesity. For example, reduced production of adiponectin and an anti-inflammatory adipokine might lead to a pro-inflammatory condition in obese individuals [27].

Based on the findings, hypertension was associated with increased SII, similar to the findings of a prospective population-based cohort study conducted in China that indicated a positive correlation between hypertension and increased SII. The cohort study discovered that those with higher baseline SII levels had a higher risk of developing hypertension throughout the follow-up period [28]. As this was a cross-sectional study, the priority of the events could not be determined. However, the significant association between hypertension and increased inflammation in the current study could align with the findings of the mentioned cohort study.

Higher SII index and hypertension may be associated with intricate interactions between inflammation, immunological response, and cardiovascular health. Low-grade chronic inflammation that elevated SII levels can indicate can result in endothelial dysfunction, oxidative stress, and arterial stiffness. This inflammatory milieu can lead to elevated blood pressure by upsetting the balance of vasoactive chemicals, including nitric oxide bioavailability, and generating pro-inflammatory cytokines. Furthermore, inflammatory mediators and immune cells may directly affect myocardial remodeling and contribute to hypertension [29].

The current study found a direct but weak relationship between SII and CRP. Similarly, Ömür et al. showed that SII and CRP were positively correlated, particularly in individuals with persistent atrial fibrillation (AF) [28]. In contrast, Ustundag et al. reported no correlation between the CRP value and SII [30]. Various factors, including variations in study populations, sample sizes, research methodologies, and confounding variables, may have caused these contradictory results.

The current study showed a significant relationship between increased SII and mortality. Studies have demonstrated a nonlinear link between the SII and all-cause mortality and a correlation between the SII and cardiovascular, cardio-cerebrovascular, and all-cause mortality in the general population. Cardiovascular and cardiocerebrovascular mortality is strongly correlated with increased SII, and a twofold increase in SII was related to a 42% increase in mortality while reducing SII to half was associated with a 15% reduction in mortality. Furthermore, a linear relationship has been identified between the SII and insulin resistance and inflection [15].

Several variables, including age, gender, and comorbidities, mediate the relationship between SII and mortality. Given the nonlinear relationship between SII and mortality, SII may have varying effects on different forms of mortality [16]. Cao et al. reported a J- or U-shaped pattern, indicating that high or low SII may be associated with an increased risk of mortality due to cancer, cardiovascular disease, and all-cause mortality [31]. Wang et al. found that the SII was associated with the lowest mortality risk at a specific cut-off, and the association was nonlinear [15]. Similarly, another study reported a cut-off for SII concerning the risk of insulin resistance among individuals with abdominal obesity [32]. Similarly, a substantially strong correlation was reported between SII and cardiovascular mortality, indicating that SII and cardiovascular mortality may be more tightly related [15]. The results suggested that the association between SII and mortality is complex and may be affected by several variables, including demographics, gender, and some medical issues. Consequently, more investigation is required to comprehend the intricate connection between SII and mortality.

Strength and limitations

This study is bolstered by its extensive sample size and the inclusion of multiple confounding factors. However, the study's limitations include its cross-sectional design and the absence of a clear understanding of the causality of the SII and mortality or other conditions.

Conclusion

Based on the results, the Systemic Inflammation Index (SII) was associated with percentage body fat (PBF) and hypertension. Additionally, there was a weak correlation between SII and high-sensitivity C-reactive protein (hs-CRP), which was linked to overall mortality in older individuals. The body fat percentage can be a suitable predictive variable for the SII inflammatory index; therefore, controlling body fat may help improve inflammation in older adults. However, further prospective and large-scale studies are warranted to establish the results.

Declarations

Conflict of Interest

The authors declare no conflicts of interest.

Author Contributions

MM, PK, SD, JJ, SMA, SRM, ST, AGH, and AJ designed the study and were involved in the manuscript's data collection, analysis, and drafting. MS was involved in the study's design and critically reviewed the manuscript. All authors read and approved the final manuscript.

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Ethical Considerations

The Mashhad University of Medical Sciences Ethics Committee authorized the procedures performed under its ethical guidelines. Before the data collection, the participants also signed an informed consent form, and Neyshabur Longitudinal Study on ageing protocols were adhered to for data confidentiality and anonymization.

Code of Ethics

IR.MUMS.MEDICAL.REC.1401.325

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