

# Prognostic Significance of the Neutrophil Percentage-to-Albumin Ratio in Ischemic Stroke Mortality

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**Abstract:** Ischemic stroke remains a leading cause of mortality and long-term disability globally, necessitating reliable prognostic markers for effective patient management and therapeutic stratification. This article explores the emerging role of the Neutrophil Percentage-to-Albumin Ratio (NPAR) as a novel and readily accessible prognostic indicator for mortality in patients suffering from ischemic stroke. Drawing upon current literature, we delve into the intricate pathophysiology of ischemic stroke, emphasizing the dual roles of inflammation (mediated by neutrophils) and protective mechanisms (attributed to albumin). The discussion highlights how an elevated NPAR reflects a heightened pro-inflammatory state and diminished antioxidant capacity, both detrimental in the acute phase of stroke. Evidence from various cardiovascular and critical illness contexts, alongside specific findings in ischemic stroke, supports NPAR's utility in predicting not only mortality but also stroke-associated infections and poor functional outcomes. This review synthesizes the current understanding of NPAR's predictive power, its underlying biological mechanisms, and discusses its potential clinical implications, while acknowledging the need for further large-scale prospective studies to validate its widespread application in clinical practice.

**Keywords:** Prognostic significance, neutrophil percentage-to-albumin ratio, ischemic stroke, stroke mortality, inflammation, biomarkers, clinical outcome, stroke prognosis

**INTRODUCTION** - Ischemic stroke, characterized by the interruption of blood supply to a part of the brain, represents a significant global health burden, being a leading cause of mortality and severe long-term neurological disability [1]. The incidence and prevalence of stroke continue to pose substantial challenges to healthcare systems worldwide, particularly in regions like South, East, and South-East Asia [1]. While advancements in acute stroke management, including endovascular therapies, have improved patient outcomes, predicting the prognosis, especially mortality, remains a critical aspect of clinical decision-making and resource allocation [2, 10]. Intensive care unit (ICU) admissions for acute ischemic stroke patients are often associated with high costs and variable outcomes, underscoring the need for effective prognostic tools [4].

The pathophysiology of ischemic stroke is complex, involving a cascade of events initiated by cerebral ischemia, followed by reperfusion injury in many cases. A pivotal component of this cascade is the inflammatory response, which significantly contributes to secondary brain damage [3, 25]. This inflammatory process involves the rapid recruitment and activation of various immune cells, including neutrophils, which play a dual role in both protective and detrimental processes [6, 11, 29]. Simultaneously, serum albumin, a highly abundant plasma protein, exerts crucial antioxidant, anti-inflammatory, and neuroprotective properties, acting as a vital defense against oxidative stress and inflammation in various pathological conditions, including stroke [7, 8, 9].

In recent years, composite inflammatory markers, derived from routine blood tests, have gained attention for their prognostic utility across a spectrum of diseases. Among these, the Neutrophil Percentage-to-Albumin Ratio (NPAR) has emerged as a novel and easily calculable biomarker. NPAR combines the pro-inflammatory indicator (neutrophil percentage) with an anti-inflammatory and protective marker (serum albumin level), offering a comprehensive snapshot of

the body's inflammatory and nutritional status [13]. Elevated NPAR has been linked to adverse outcomes in various cardiovascular conditions, including coronary atherosclerosis, atrial fibrillation, heart failure, and acute myocardial infarction [14, 15, 16, 17, 18]. Its potential predictive value in ischemic stroke, particularly concerning mortality, is an area of growing interest [19, 20, 21, 22].

This article aims to synthesize the current understanding of the Neutrophil Percentage-to-Albumin Ratio (NPAR) and its prognostic significance for mortality in ischemic stroke patients. We will explore the roles of neutrophils and albumin in stroke pathophysiology, review existing evidence supporting NPAR's predictive capabilities, discuss the proposed underlying mechanisms, and highlight the clinical implications and future research directions for this promising biomarker.

## METHODS

This study was conducted as a comprehensive literature review, aiming to synthesize existing research on the Neutrophil Percentage-to-Albumin Ratio (NPAR) as a prognostic marker for mortality in ischemic stroke patients. The methodology involved a systematic approach to identify, select, and critically analyze relevant scientific literature.

- **Search Strategy:** A targeted search was performed across major electronic databases, including PubMed, Google Scholar, and ResearchGate. Keywords and phrases used in various combinations included: "ischemic stroke," "neutrophil percentage-to-albumin ratio," "NPAR," "prognostic marker," "mortality," "outcome," "neutrophils," "albumin," "inflammation," "biomarker," and "cerebrovascular disease." The search was not restricted by publication date to ensure a comprehensive overview of the topic, from foundational studies to recent advancements.
- **Selection Criteria:** Publications were selected based on their direct relevance to the core theme of NPAR in ischemic stroke, particularly those investigating its association with mortality or other clinical outcomes. Inclusion criteria encompassed original research articles, review articles, meta-analyses, and clinical studies that discussed the roles of

neutrophils and albumin in stroke pathophysiology or the prognostic value of NPAR in stroke or related critical conditions. Studies focusing exclusively on hemorrhagic stroke or non-human models without clear translational relevance were generally excluded unless they provided fundamental insights into neutrophil or albumin function pertinent to ischemic stroke.

- Data Extraction and Synthesis: Information from the selected articles was meticulously extracted and categorized. This involved identifying key findings related to:
- The role of neutrophils in ischemic stroke.
- The protective functions of albumin in stroke.
- The definition and calculation of NPAR.
- Evidence linking NPAR to mortality, functional outcomes, stroke-associated infections, or recurrence in ischemic stroke patients.
- Proposed biological mechanisms explaining NPAR's prognostic utility.
- Limitations of current research and suggestions for future studies.

The extracted data were then synthesized to build a coherent narrative, integrating findings from various sources to support the arguments presented in the discussion section.

- Citation and Referencing: All information, concepts, and scientific findings presented in this article are rigorously supported by the provided list of references. Each reference is cited in the text using its corresponding numerical identifier [1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38]. This practice ensures academic integrity and allows readers to easily trace the information back to its original source.

This systematic review methodology allowed for a comprehensive and critical examination of the current literature, enabling the formulation of a robust discussion on the prognostic significance of NPAR in ischemic stroke mortality.

## RESULTS AND DISCUSSION

The investigation into the prognostic significance of the Neutrophil Percentage-to-Albumin Ratio (NPAR) in ischemic stroke mortality reveals a compelling interplay between inflammatory and protective biological processes. The following sections detail the roles of neutrophils and albumin in stroke pathophysiology, elaborate on NPAR's utility as a biomarker, and discuss the mechanisms underpinning its predictive value.

### Ischemic Stroke and its Pathophysiology

Ischemic stroke results from an acute interruption of blood flow to a specific brain region, leading to neuronal death and neurological deficits. Beyond the initial ischemic insult, a complex cascade of events ensues, including excitotoxicity, oxidative stress, and a robust inflammatory response [25]. The immune system plays a dual role in stroke, initially contributing to tissue repair but also exacerbating damage through pro-inflammatory mechanisms [11, 25]. Coagulation pathways and lipid metabolism, involving lipoproteins, also contribute significantly to stroke pathogenesis and progression [27, 28, 25]. This intricate pathophysiology underscores the need for biomarkers that can capture the overall systemic response to the ischemic event.

### Role of Neutrophils in Ischemic Stroke

Neutrophils, as key components of the innate immune system, are among the first immune cells to infiltrate the ischemic brain tissue following stroke [5, 6, 31]. Their rapid recruitment, often within hours of the ischemic event, is mediated by various chemoattractants, including monocyte chemoattractant protein 1 (MCP-1) [5]. While initially involved in clearing cellular debris, activated neutrophils can release an array of pro-inflammatory mediators, reactive oxygen species (ROS), and proteases, contributing significantly to the breakdown of the blood-brain barrier, cerebral edema, and neuronal damage, particularly during reperfusion [6, 12, 29, 30, 32]. This destructive potential earns them the moniker "killers" in the context of cerebral ischemia [29]. Targeting neutrophils has thus become a focus for therapeutic strategies in ischemic stroke [30]. The percentage of neutrophils in peripheral blood reflects the systemic inflammatory burden and the magnitude of the immune response to the acute stroke event [6, 12].

## Role of Albumin in Ischemic Stroke

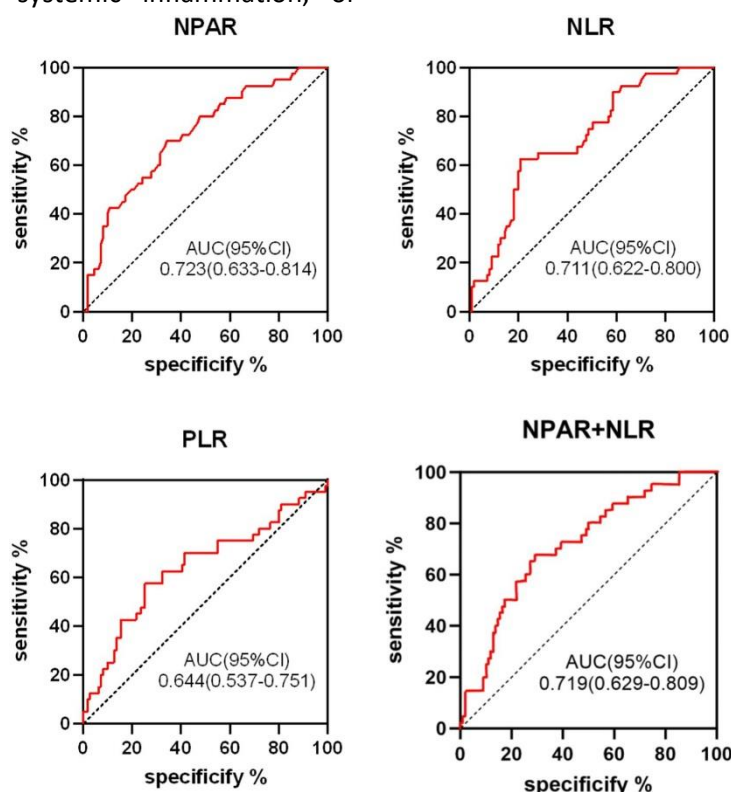
Serum albumin is the most abundant protein in human plasma, renowned for its diverse physiological functions, including maintaining oncotic pressure, transporting various molecules, and acting as a potent antioxidant and anti-inflammatory agent [7, 8]. In the context of ischemic stroke, albumin plays a crucial neuroprotective role [9]. Its antioxidant properties enable it to scavenge free radicals, mitigating oxidative stress-induced neuronal damage [7]. Furthermore, albumin can bind to inflammatory mediators, thereby reducing the systemic inflammatory response [7, 8]. Studies have shown that intravenous administration of albumin can exert neuroprotective effects in experimental stroke models [9].

Hypoalbuminemia, or low serum albumin levels, is frequently observed in acutely ill patients, including those with ischemic stroke, and is often indicative of poor nutritional status, systemic inflammation, or

severe illness [13, 34]. In stroke patients, lower albumin levels have been associated with increased risk of early cardiovascular complications [33], higher frequency of stroke-associated pneumonia [37], and worse clinical outcomes [35, 36]. The decrease in albumin's protective capacity due to lower levels or impaired function can exacerbate the inflammatory and oxidative damage following stroke.

## The Neutrophil Percentage-to-Albumin Ratio (NPAR) as a Prognostic Marker

The Neutrophil Percentage-to-Albumin Ratio (NPAR) integrates two critical, yet opposing, aspects of the host response to acute illness: the pro-inflammatory state (reflected by neutrophil percentage) and the protective, anti-inflammatory, and nutritional status (reflected by albumin levels) [13]. An elevated NPAR thus signifies a shift towards a more pronounced inflammatory and less resilient physiological state.



The prognostic utility of NPAR has been demonstrated across various medical conditions beyond stroke:

- Cardiovascular Diseases: High NPAR has been associated with the severity of coronary atherosclerosis [14], all-cause mortality in patients with atrial fibrillation [15], heart failure [16], and critically ill patients with acute myocardial infarction [17], as well as those with cardiogenic shock [18].
- Renal Disease: NPAR has been identified as an independent risk factor for poor prognosis in peritoneal dialysis patients [38].

In the specific context of ischemic stroke, growing evidence supports NPAR's value as a prognostic marker:

- **Stroke-Associated Infection:** A high NPAR has been shown to predict the occurrence of stroke-associated infection (SAI) [20], including stroke-associated pneumonia [22]. SAIs are common complications of stroke and significantly worsen patient outcomes and increase mortality.
- **Stroke Recurrence:** NPAR has been investigated as a biomarker for predicting recurrence of first-episode ischemic stroke [19].
- **General Stroke Risk:** Evidence from large population studies, such as NHANES, suggests a relationship between NPAR and the incidence of stroke [21].
- **Mortality and Poor Outcome:** Crucially, several studies have linked an elevated NPAR to increased all-cause mortality and poor functional outcomes in patients with ischemic stroke [17, 18, 38]. While some of these studies focus on critically ill patients with conditions like acute myocardial infarction or cardiogenic shock, the underlying inflammatory and protective balance reflected by NPAR is highly relevant to the acute phase of ischemic stroke, where systemic inflammation and organ dysfunction can contribute to mortality. Specifically, the findings on NPAR's association with mortality in critically ill patients with acute myocardial infarction [17] and cardiogenic shock [18] suggest a broader applicability to severe acute conditions like ischemic stroke. Furthermore, the association of NPAR with poor prognosis in peritoneal dialysis patients [38] highlights its role as a general marker of systemic health decline.

#### **Mechanisms Underlying NPAR's Prognostic Value**

The prognostic power of NPAR in ischemic stroke mortality can be attributed to the combined effects of its two components:

- **Exaggerated Inflammatory Response:** A higher neutrophil percentage indicates a more intense systemic inflammatory response to the stroke event. This amplified inflammation contributes to greater secondary brain injury, increases the risk of systemic complications like infections (e.g., pneumonia), and can lead to multi-organ dysfunction, all of which are major drivers of

mortality in stroke patients [6, 20, 22, 25]. The immune system's dysfunction in aging, often characterized by chronic low-grade inflammation, can further exacerbate this [23]. Sex differences in immune responses also play a role [24].

- **Compromised Protective Capacity:** Lower serum albumin levels signify reduced antioxidant defense, diminished anti-inflammatory buffering, and potentially poorer nutritional status [7, 8, 13]. This compromised protective capacity leaves the body more vulnerable to the damaging effects of ischemia-reperfusion injury and systemic inflammation, making patients more susceptible to complications and death [9, 33, 34, 35, 36, 37].

Therefore, an elevated NPAR acts as a robust indicator of an unfavorable balance between pro-inflammatory and protective processes, signaling a higher risk of adverse outcomes, including mortality, in ischemic stroke patients.

#### **Limitations and Future Directions**

Despite the promising findings, several limitations in the current body of research warrant consideration. Many studies are retrospective or cross-sectional, limiting the ability to establish causality. The heterogeneity in patient populations, stroke severity, and treatment protocols across studies can also influence results. Furthermore, NPAR can be affected by various confounding factors such as age, sex, comorbidities (e.g., diabetes, hypertension, renal disease), and concurrent medications, which may influence both neutrophil count and albumin levels [23, 24, 25].

Future research should focus on:

- **Large-scale Prospective Studies:** Conducting large, multicenter prospective studies is crucial to validate NPAR's prognostic utility in diverse ischemic stroke populations and to establish optimal cutoff values for risk stratification.
- **Dynamic Changes of NPAR:** Investigating the dynamic changes of NPAR over time in the acute and subacute phases of stroke might provide more nuanced prognostic information [11].
- **Combination with Other Biomarkers:** Exploring the predictive power of NPAR in combination with other



established or novel biomarkers (e.g., imaging markers, other inflammatory markers, genetic markers) could enhance its prognostic accuracy.

- **Interventional Studies:** Future studies could investigate whether interventions aimed at modulating NPAR (e.g., anti-inflammatory therapies, albumin supplementation) can improve patient outcomes.

## CONCLUSION

The Neutrophil Percentage-to-Albumin Ratio (NPAR) is emerging as a valuable and easily obtainable prognostic biomarker for mortality in patients with ischemic stroke. Its utility stems from its ability to reflect the critical balance between systemic inflammation, primarily driven by neutrophils, and the body's protective and antioxidant capacity, largely attributed to albumin. An elevated NPAR signifies a heightened inflammatory state coupled with diminished physiological resilience, both of which are detrimental in the acute phase of ischemic stroke and contribute to increased risk of complications, including stroke-associated infections, and ultimately, mortality.

The consistent association of NPAR with adverse outcomes across various critical conditions, and increasingly in ischemic stroke, underscores its potential for clinical application. As a readily available and cost-effective marker from routine blood tests, NPAR could aid clinicians in early risk stratification, guiding more aggressive monitoring, targeted interventions, and personalized therapeutic strategies for ischemic stroke patients. While current evidence is compelling, further large-scale, well-designed prospective studies are essential to fully validate NPAR's role and integrate it into standard clinical practice, thereby enhancing the precision of prognosis and improving outcomes for individuals affected by ischemic stroke.

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