REVIEW

DOI: 10.4274/nkmj.galenos.2025.48612
Nam Kem Med J 2026;14(1):103-107

Glial Fibrillary Acidic Protein as a Biomarker in Acute Ischemic Stroke: Diagnostic Value and Prognostic Implications

Akut İskemik İnmede Biyobelirteç Olarak Glial Fibriller Asidik Protein: Tanısal Değer ve Prognostik İpuçları

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ABSTRACT

Stroke is one of the leading causes of mortality and long-term disability worldwide, with ischemic stroke representing the most common subtype. Despite significant progress in neuroimaging and reperfusion therapies, rapid and accurate diagnosis remains a major challenge in clinical practice. Glial fibrillary acidic protein, an intermediate filament protein specific to astrocytes, has recently gained attention as a promising blood-based biomarker. Following astrocytic damage and disruption of the blood-brain barrier, glial fibrillary acidic protein is released into the extracellular space and enters the circulation. Although its levels rise rapidly in hemorrhagic stroke, the increase in ischemic stroke occurs more gradually, typically becoming measurable within the first 24 to 48 hours after the onset of symptoms. Clinical investigations have demonstrated that blood concentrations of glial fibrillary acidic protein are significantly higher in patients with ischemic stroke compared to healthy controls and that these elevations correlate with neurological severity, infarct volume, and functional outcomes. Thus, glial fibrillary acidic protein provides valuable prognostic insights and may support patient stratification. However, variability in cut-off values, differences in detection methods, and delayed kinetics remain important limitations. Future developments include the integration of glial fibrillary acidic protein into multimarker panels and the use of ultrasensitive point-of-care assays that may enable rapid decision-making in emergency settings. Overall, glial fibrillary acidic protein has the potential to serve as a complementary biomarker for diagnosis, prognosis, and monitoring in ischemic stroke, thereby contributing to improved patient care and individualized therapeutic strategies.

Keywords: Ischemic stroke, glial fibrillary acidic protein, biomarker, neuroinflammation, prognosis

ÖZ

İnme, dünya genelinde önde gelen ölüm ve kalıcı sakatlık nedenlerinden biridir ve en sık görülen alt tipi iskemik inmedir. Nörogörüntüleme yöntemleri ve reperfüzyon tedavilerindeki önemli ilerlemelere rağmen, hızlı ve doğru tanı klinik uygulamada hala büyük bir güçlük oluşturmaktadır. Astrositlere özgü bir ara filament proteini olan glial fibriller asidik protein, son yıllarda kan temelli umut verici bir biyobelirteç olarak öne çıkmıştır. Astrosit hasarı ve kan-beyin bariyerinin bozulmasını takiben glial fibriller asidik protein ekstrasellüler alana ve dolaşıma salınmaktadır. Hemorajik inmede düzeyler hızla yükselirken, iskemik inmede artış daha yavaş olmakta ve genellikle semptomların başlamasından sonraki ilk 24 ila 48 saat içerisinde ölçülebilir seviyelere ulaşmaktadır. Klinik araştırmalar, iskemik inme hastalarında kanda glial fibriller asidik protein düzeylerinin sağlıklı bireylere kıyasla belirgin şekilde yüksek olduğunu ve bu artışın nörolojik şiddet, enfarktüs hacmi ve fonksiyonel sonuçlarla ilişkili olduğunu göstermiştir. Bu nedenle glial fibriller asidik protein, prognostik öngörüler sunarak hasta sınıflamasına katkıda bulunabilir. Ancak, eşik değerlerdeki değişkenlik, ölçüm yöntemlerindeki farklılıklar ve gecikmiş kinetik önemli sınırlılıklar oluşturmaktadır. Gelecekte glial fibriller asidik proteinin multimarker panellere entegrasyonu ve ultrasensitif hızlı testlerin geliştirilmesi, acil servislerde daha etkin karar süreçlerini mümkün kılabilir. Genel olarak glial fibriller asidik protein, iskemik inmede tanı, prognoz ve izlem için tamamlayıcı bir biyobelirteç olma potansiyeline sahiptir.

Anahtar Kelimeler: İskemik inme, glial fibriller asidik protein, biyobelirteç, nöroenflamasyon, prognoz

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Received: 03.10.2025 **Accepted:** 23.12.2025 **Publication Date:** 04.03.2026

Cite this article as: Atasever Ü. Glial fibrillary acidic protein as a biomarker in acute ischemic stroke: diagnostic value and prognostic implications. Nam Kem Med J. 2026;14(1):103-107

INTRODUCTION

Stroke is recognized as one of the leading causes of mortality and morbidity worldwide. As of 2021, approximately 12 million new stroke cases were reported globally, highlighting the growing burden of stroke on healthcare systems each year. Currently, it is estimated that one in four individuals will experience at least one stroke during their lifetime[1,2].

Acute stroke is characterized by the sudden onset of a focal neurological deficit affecting a specific vascular territory of the brain, retina, or spinal cord. Clinically, it is classified into two main subtypes: hemorrhagic and ischemic stroke (IS). Among these, IS accounts for approximately 85% of all stroke cases, making it the most prevalent form[3]. Acute ischemic stroke (AIS) typically occurs as a result of the occlusion of a cerebral vessel lumen due to either a thrombus or an embolus, leading to an interruption of regional blood flow[4]. Thrombotic strokes are most commonly associated with large-vessel atherosclerotic obstructions, whereas embolic strokes usually occur when clots formed in the cardiac chambers migrate distally into the cerebral vasculature. Lacunar infarcts, on the other hand, develop as a consequence of small-vessel disease, characterized by occlusion of penetrating arteries in subcortical regions with poor collateral circulation[5].

Pathophysiology of Acute Ischemic Stroke

Under normal physiological conditions, cerebral blood flow is maintained constant by autoregulatory mechanisms involving vessel diameter regulation and vasodilators such as nitric oxide[6]. This system operates within a mean arterial pressure range of 60-150 mmHg; however, in pathological conditions such as stroke, autoregulation is impaired, and brain tissue responds passively to decreases in perfusion pressure. This decline in perfusion pressure first results in the cessation of protein synthesis, followed by reduced glucose utilization, activation of anaerobic metabolism, and ultimately neuronal dysfunction[7]. Within the ischemic territory, an infarct core forms in areas supplied by a single artery, surrounded by partially perfused but still viable tissue maintained through collateral circulation, termed the “ischemic penumbra.” Preservation of this region requires early recanalization, which underscores the principle that “time is brain” in IS therapy[7,8].

The cellular damage following IS is not limited to apoptosis but results from the interplay of multiple regulated cell death mechanisms, including ferroptosis, necroptosis, pyroptosis, parthanatos, phagoptosis, and autophagy. These pathways are driven by shared upstream processes such as oxidative stress, metabolic failure, inflammatory signaling, and immune activation, which collectively contribute to irreversible tissue injury within both the infarct core and the ischemic penumbra. Although these mechanisms have distinct molecular features, they converge in amplifying cellular dysfunction and structural damage within the ischemic brain[9-13].

Importantly, these regulated cell death pathways do not exclusively affect neurons but also involve glial cells within the ischemic microenvironment, particularly astrocytes, which play a central role in maintaining metabolic support and blood-brain barrier integrity. The extent and pattern of astroglial involvement may therefore reflect the severity and progression of ischemic injury[12,13].

Diagnosis and Treatment

AIS is a neurological emergency characterized by the sudden onset of focal neurological deficits and requires prompt recognition and intervention. Common clinical manifestations include unilateral weakness, speech and visual disturbances, sensory deficits, imbalance, nausea, and sudden severe headache. Although classical symptoms occur with similar frequency in both sexes, atypical presentations may be more frequent in women, contributing to diagnostic challenges[14,15].

Due to the heterogeneity of clinical presentation, early and accurate diagnosis is a critical determinant of effective stroke management. Timely identification of AIS directly influences treatment eligibility and clinical outcomes, as delays in diagnosis are associated with increased neurological damage and poorer prognosis. Therefore, rapid diagnostic evaluation remains a cornerstone of modern stroke care[16,17].

The primary goal of AIS treatment is the preservation of at-risk brain tissue through timely reperfusion. Treatment strategies and patient management depend largely on early recognition, appropriate triage, and prompt initiation of therapy. Delays in the pre-hospital and early in-hospital phases significantly reduce the effectiveness of therapeutic interventions and negatively affect functional recovery[18-20].

Despite advances in acute stroke management, variability in clinical course and outcomes remains substantial. Early prognostic assessment is essential for guiding treatment decisions, optimizing patient selection, and predicting neurological recovery. In this context, there is a growing need for reliable biomarkers that can support early diagnosis, reflect the extent of brain injury, and provide prognostic information, particularly in the hyperacute phase of AIS[14,21-24].

Glial Fibrillary Acidic Protein

Glial fibrillary acidic protein (GFAP) is a unique intermediate filament protein of astrocytes in the central nervous system (CNS), playing a critical role in maintaining the structural integrity of these cells, providing mechanical resilience, and sustaining homeostasis within neural tissue. Astrocytes constitute approximately 30-40% of CNS cells and, beyond their role in the

blood-brain barrier (BBB), they engage in extensive interactions with neurons and other glial cells. They are central to fundamental processes such as synaptic transmission, ion balance, and metabolic support. Structurally, GFAP is composed of three main domains: an N-terminal head region, a central rod domain, and a C-terminal tail domain, all of which are functionally important for filament assembly and stabilization[25,26]. GFAP is expressed in mature astrocytes located in both gray and white matter, in the cerebellum, subventricular and subgranular zones, and in Müller cells of the retina. Moreover, GFAP expression can also be observed in Schwann cells, enteric glia, and hepatic stellate cells. To date, ten distinct isoforms of GFAP have been identified in the nervous system, with GFAPα being the most common and extensively studied variant[26]. GFAP is not merely a structural protein but also serves as an important biomarker reflecting glial cell responses. Its levels rise markedly in conditions characterized by impaired gliovascular integrity, disrupted glymphatic clearance, or astrocytic injury[27]. For instance, in post-mortem brain tissue of individuals with CADASIL, a hereditary small-vessel disease, an accumulation of GFAP-positive clasmatodendritic astrocytes was observed in perivascular regions of the deep white matter, indicating disruption of the gliovascular unit and significant impairment of glymphatic function[28].

The recognition of GFAP as a clinically relevant biomarker has been enabled by advances in highly sensitive detection methods in biological fluids such as cerebrospinal fluid (CSF), vitreous humor, and amniotic fluid. Although conventional assays like enzyme-inked immunosorbent assay lack the sensitivity to detect its low plasma concentrations, elevated GFAP levels can nonetheless be observed in conditions such as traumatic brain injury and neuromyelitis optica[26,29]. More recently, ultrasensitive platforms, particularly Simoa, have allowed for reliable quantification of GFAP in both healthy individuals and neurological disorders, with portable devices even enabling results within minutes[30]. The mechanisms by which GFAP enters the circulation under pathological conditions remain incompletely understood, but current evidence indicates a multifactorial process. Proposed pathways include bulk flow through arachnoid villi, the glymphatic system and cervical lymphatic drainage, as well as bidirectional exchange across the BBB and the blood-CSF barrier[26,27,31]. In addition to pre-analytical factors, the "hook effect" caused by protein aggregation represents a technical limitation, potentially compromising assay reliability. Remarkably, GFAP has been shown to remain stable within aggregate structures for extraordinary durations, as evidenced by the "Heslington Brain" specimen, which preserved GFAP integrity for thousands of years under exceptional conditions. In living tissues, however, such aggregate accumulation has been associated with fatal neurological disorders, including Alexander disease[32].

In conclusion, GFAP has emerged as a versatile biomarker not only for elucidating astrocyte biology but also for its growing significance in the diagnosis, prognosis, and monitoring of neurological diseases. Owing to its structural features at the molecular level and its association with the glymphatic system, GFAP provides a valuable parameter for assessing astrocytic responses in both clinical and research contexts.

Glial Fibrillary Acidic Protein in Acute Ischemic Stroke

GFAP is an intermediate filament protein exclusively expressed in astrocytes within the CNS. During acute AIS, disruption of astrocytic integrity and the BBB leads to the release of GFAP into the extracellular space and subsequently into the circulation. This process renders GFAP a promising biomarker reflecting astroglial injury in AIS. While GFAP rises rapidly and to high levels in hemorrhagic stroke, its increase in AIS is more gradual, typically reaching measurable levels within the first 24-48 hours after symptom onset[33,34]. Several clinical studies have demonstrated that GFAP levels in AIS are significantly elevated compared to healthy controls and strongly correlate with clinical severity and outcome measures. Ferrari et al.[35] reported that serum GFAP concentrations peaked within the first 24 hours and showed significant associations with National Institutes of Health Stroke scale (NIHSS) scores, modified Rankin scale and three-month functional outcomes. Similarly, Amalia[34] observed a positive correlation between GFAP levels and NIHSS scores, with markedly higher values in patients with large vessel occlusion compared to small vessel occlusion. These findings indicate that GFAP not only reflects acute astroglial injury but also provides prognostic insights into stroke severity and recovery potential. GFAP has also been linked to infarct volume and neurovascular status in AIS. Wunderlich et al.[36] reported that elevated serum GFAP levels were associated with larger infarct volumes and impaired neurovascular integrity. Furthermore, the multicenter BE FAST India study by Kalra et al.[37] demonstrated that GFAP, at defined cut-off values (0.33-0.57 µg/L), exhibited high diagnostic accuracy and could support clinical decision-making particularly in the early phase. In addition, recent evidence suggests that GFAP can be evaluated not only in its soluble protein form but also in association with circulating immune cells. van den Bossche et al.[38] showed that the proportion of circulating $GFAP^{+}CD16^{+}$ monocytes significantly increased within the first 2-8 hours after AIS and correlated with infarct volume. This cellular approach represents an innovative method that may capture lesion size earlier and more precisely than soluble GFAP. It should be noted that reported GFAP cut-off values may vary depending on the analytical assay and measurement platform used. Key studies evaluating GFAP for early stroke differentiation and prognostic assessment are summarized in Table 1. Taken together, these findings demonstrate that GFAP possesses both diagnostic and prognostic potential in AIS. GFAP reflects astroglial damage, correlates with neurological deficit and infarct burden, and predicts functional recovery. Although its slower rise compared

Table 1. Key studies evaluating GFAP for early stroke differentiation and prognosis

Study (references)	Study design/population	Time window from symptom onset	Correlations	Main findings
Ferrari et al.[35]	Prospective observational study, AIS patients	≤24 h	NIHSS, mRS, 3-month outcome	GFAP levels correlated with stroke severity and functional outcome
Amalia[34]	Observational cohort, AIS patients	≤24-48 h	NIHSS, vessel occlusion	Higher GFAP levels observed in large vessel occlusion compared to small vessel disease
Wunderlich et al.[36]	Cohort study, AIS patients	≤72 h	Infarct volume, neurovascular integrity	Elevated GFAP associated with larger infarct volume and impaired neurovascular status
Kalra et al.[37]	Multicenter prospective study	≤6 h	NIHSS, clinical diagnosis	GFAP showed high diagnostic accuracy and supported early clinical decision-making
van den Bossche et al.[38]	Observational study, AIS patients	2-8 h	Infarct volume	Circulating GFAP-positive monocytes increased early and correlated with lesion size

AIS: Acute ischemic stroke, GFAP: Glial fibrillary acidic protein, NIHSS: National Institutes of Health Stroke scale, mRS: Modified Rankin scale

to hemorrhagic stroke limits its utility in very early diagnosis, GFAP holds substantial clinical relevance in assessing disease severity, predicting outcomes, and serving as a complementary component of multiparametric biomarker panels in AIS.

Future Perspectives and Current Challenges

GFAP has emerged as a promising biomarker in AIS, yet important challenges remain before it can be translated into routine clinical practice. Variability in reported cut-off values, differences in assay platforms, and the absence of standardized protocols continue to hinder comparability across studies. Furthermore, the relatively delayed rise of GFAP in IS limits its usefulness as a very early diagnostic marker. Clinical interpretation may also be complicated by comorbid neurological conditions or prior cerebrovascular events. In particular, chronic neurodegenerative diseases, traumatic brain injury, or other conditions associated with astroglial damage may elevate baseline GFAP levels and thereby confound stroke-related measurements. Looking ahead, technological advances such as rapid point-of-care assays could enable bedside testing and shorten decision-making times in emergency settings. Integrating GFAP into multimarker panels may provide higher diagnostic and prognostic accuracy than using it alone. In addition, innovative cellular approaches, including the assessment of GFAP-positive monocytes, offer new perspectives for estimating lesion burden and monitoring tissue injury in the hyperacute phase. These developments highlight the potential of GFAP to complement existing diagnostic and prognostic strategies in acute AIS.

CONCLUSION

GFAP reflects astrocytic injury and offers valuable insights into disease severity, infarct volume, and functional outcome in acute IS. While its temporal dynamics restrict its role in ultra-early diagnosis, GFAP remains highly relevant for prognostic assessment and patient stratification. Importantly, this review provides a unified framework by integrating the early diagnostic utility of GFAP with its prognostic relevance across the acute and recovery phases of IS. By bridging early stroke differentiation with longitudinal outcome assessment, this perspective highlights GFAP as a versatile biomarker that extends beyond single-purpose applications. With further methodological refinement and integration into multimarker strategies, GFAP is likely to become an important complementary tool in the modern management of acute IS.

Footnote

Financial Disclosure: The author declared that this study received no financial support.

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