Brief Report

Comparative measurement of plasma soluble urokinase plasminogen activator receptor using a point-of-care device and a clinical analyzer in stroke patients

Areti Kourti*, Eirini Keskilidou, Alexandra Skoura, Athanasia Samara, Paraskevi Thanou, Paraskevi Karalazou, Katerina Thisiadou, and Kali Makedou

Laboratory of Biochemistry, AHEPA University Hospital, School of Medicine, Aristotle University of Thessaloniki, Thessaloniki, Central Macedonia 54124, Greece

Abstract

Background: Soluble urokinase plasminogen activator receptor (suPAR) reflects immune activation and chronic inflammatory responses and has been proposed as a prognostic biomarker in several acute conditions. **Objectives:** This study aims to evaluate the clinical performance of two analytical methods for plasma suPAR determination in patients with stroke: a point-of-care (POC) lateral-flow assay and an automated particle-enriched turbidimetric immunoassay performed on a biochemistry analyzer. **Methods:** Plasma samples from 27 patients with stroke and 14 age-matched healthy controls were analyzed with both methods. suPAR concentrations were significantly higher in stroke patients compared with controls for both assays. **Results:** A statistically significant difference between POC and analyzer measurements was observed only within the stroke group (p=0.024), and a strong positive correlation between the two methods was identified (r = 0.745, p<0.001). Receiver operating characteristic analysis demonstrated higher sensitivity for the POC method (96%) compared with the analyzer (82%), while the specificity was identical (57%). **Conclusion:** These findings suggest that suPAR measurement using a rapid POC platform provides reliable results and may support early risk stratification in patients with stroke in emergency department settings.

Keywords: Soluble urokinase plasminogen activator receptor, Point-of-care, Biochemistry analyzer, Stroke

1. Introduction

Soluble urokinase plasminogen activator receptor (suPAR) is the circulating form of the membrane-bound urokinase plasminogen activator receptor, a glycosylphosphatidylinositol-anchored glycoprotein expressed on various cell types, including immune cells (monocytes, neutrophils, and activated T cells), endothelial cells, fibroblasts, and podocytes. The soluble form (suPAR) is generated through proteolytic cleavage (e.g., by proteases or during inflammation), releasing a 20–50 kDa fragment into the circulation.[1,2] SuPAR levels reflect immune activation and chronic inflammation and have been implicated in various pathologies, including infections, cardiovascular disease, renal dysfunction, malignancies, and systemic inflammatory states.[3,4]

As a biomarker, suPAR has several attractive properties. It is relatively stable over time, less volatile than acute-phase reactants, such as C-reactive protein or interleukin 6, and shows minimal circadian variation or short-term fluctuation in response to transient stressors.[5,6] As a marker of systemic chronic inflammation, suPAR has been proposed to reflect a baseline immunological set point and is associated with morbidity and mortality across diverse disease states.[5-7] For instance, elevated suPAR in the general population predicts cardiovascular events, chronic kidney disease, and all-cause mortality.[4,8] Genetic and mechanistic studies further illuminate suPAR not just as a passive biomarker, but as an active participant in disease mechanisms. For example, high

***Corresponding author:**
Areti Kourti (aretikourti@auth.gr)

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suPAR levels have been shown to induce pro-inflammatory changes in vascular tissues, enhance monocyte chemotaxis, and promote atherogenesis.[9]

Recently, suPAR has been studied in the context of neurological disease. In acute brain injuries, such as ischemic stroke, subarachnoid hemorrhage, and traumatic brain injury, elevated suPAR levels in blood have been associated with poor outcomes, including high modified Rankin scale (mRS) scores at 90 days, suggesting that suPAR may serve as a prognostic biomarker in neurocritical illness.[10] In addition, in aneurysmal subarachnoid hemorrhage, elevated suPAR in cerebrospinal fluid correlates with delayed cerebral ischemia and neurological disability.[11]

Stroke, a leading cause of death and disability worldwide, is characterized by sudden disruption of cerebral blood flow, either due to arterial occlusion (ischemic) or vessel rupture (hemorrhagic), resulting in deprivation of oxygen and nutrients, and subsequent neuronal cell death.[12] According to the World Health Organization, stroke accounts for around 10% of global mortality, making it one of the top causes of death.[13] Traditional risk factors for stroke include hypertension, hyperlipidemia, smoking, obesity, and diabetes mellitus.[14] However, inflammation also plays a key pathophysiological role in stroke; systemic inflammatory responses either precede or follow the event, and influence both acute injury and recovery.[15,16] For instance, after a stroke, there is often a biphasic immune response characterized by early inflammation followed by immunosuppression, which causes predisposition to infections.[17] In this regard, suPAR may serve as a bridge biomarker, reflecting not only baseline immune activation, but also pathologically relevant inflammation in stroke.

Despite this potential, point-of-care (POC) measurement of suPAR in acute stroke settings has not been well studied. Emergency departments (EDs) and stroke units could benefit from rapid biomarkers that help stratify patients by risk, guide early management, and predict outcomes. During the COVID-19 pandemic, suPAR gained traction as a triage biomarker, especially in settings with high patient volumes.[18] Several hospitals used automated assays on biochemistry analyzers, but POC devices (such as lateral flow tests) are now available, offering the promise of rapid turnaround, decentralized testing, and real-time decision-making.

Thus, this study aims to evaluate the clinical performance of two different suPAR quantification methods in the plasma of stroke patients: (i) A POC lateral flow-based assay, and (ii) A conventional immunological method (particle-enhanced turbidimetric immunoassay [PETIA]) performed on an automated biochemistry analyser. Our objective is to assess agreement, correlation, and discrimination between the methods, and explore the potential benefit of using a POC device in EDs for early risk stratification of stroke patients.

2. Methods

2.1. Population and study design

This was a retrospective observational study. Plasma samples were obtained from 27 patients (13 males, 14 females; mean age 69 ± 15 years) who presented with stroke to the ED of AHEPA University Hospital, Thessaloniki, Greece. Patients were selected based on a confirmed clinical diagnosis of stroke (ischemic or hemorrhagic), made by neurologists, using standard imaging (computed tomography/magnetic resonance imaging). Samples were collected in tubes containing ethylenediaminetetraacetic acid as part of routine care, then processed to obtain plasma. The aliquoted samples were stored at −80°C for 1 month until analysis and were thawed only at the time of analysis.

A control group of age-matched 14 healthy individuals (4 males, 10 females) was recruited voluntarily. Controls had no known history of cerebrovascular disease, systemic inflammatory or autoimmune disease, recent infection, or malignancy. All participants (or their legal representatives) provided informed consent, and all procedures of the study were in accordance with those demanded by the Ethics Committee of Aristotle University of Thessaloniki.

2.2. Methods of suPAR measurement

2.2.1. POC method

The first method used for suPAR quantification was the suPARnostic QT assay (ViroGates, Denmark), designed for POC use. This test is based on lateral flow immunoassay technology, where the test cassette contains a nitrocellulose strip with two immobilized antibody zones, and a running buffer is applied. After a plasma sample is introduced, it migrates through capillary action and binds to detection antibodies bonded to colored particles. The test result is interpreted using the LF QT FLEX reader (QIAGEN, Germany), a portable colorimetric reader, which reports suPAR concentration in ng/mL, over a dynamic range of about 2–15 ng/mL, according to manufacturer specifications.

This POC system has been widely used in acute settings, such as triage during infectious outbreaks, due to its speed, simplicity, and minimal laboratory infrastructure requirements.

2.2.2. Automated analyzer method

The second method was the suPARnostic Turbilatex PETIA assay, performed on a Cobas e801 (Roche Diagnostics,

Germany) within the Cobas 8000 analytical system. Two dedicated reagents (latex particles coated with anti-suPAR antibodies, derived from mouse/rat) are loaded into a cassette in the analyzer. The analyzer automatically dilutes latex particles, forming immune complexes that agglutinate. The increase in turbidity is then measured spectrophotometrically at 570–590 nm, and the signal is translated into suPAR concentration (ng/mL) based on calibration curves.

The PETIA-based method is suited for high-throughput laboratories, offering good precision, reproducibility, and an extended measuring range.

2.3. Statistical analysis

All statistical analyses were carried out using the Statistical Package for Social Sciences software v.25 (IBM, United States of America). First, the Kolmogorov–Smirnov test was applied to assess the normality of the distribution of suPAR values in each method and group. Given non-normal distributions, non-parametric tests were employed. Mann–Whitney U test was used to compare suPAR concentrations between stroke patients and healthy controls for each method. Spearman's rank correlation coefficient (ρ) was used to assess the correlation between POC and analyzer suPAR measurements in the stroke group (and in controls).

In addition, receiver operating characteristic (ROC) curve analyses were performed for each assay to evaluate their diagnostic or discriminative performance in distinguishing stroke patients from controls. From ROC curves, we computed the area under the curve (AUC), optimal cut-off values (Youden's index), sensitivity, and specificity. Finally, a Bland–Altman analysis was performed to assess the agreement between the two different measurement techniques. A p-value (two-tailed) of 0.05 was set as the threshold for statistical significance.

3. Results

3.1. Plasma suPAR concentrations in study and control groups

Plasma suPAR levels were significantly higher in patients with stroke compared with healthy controls for both analytical methods (Table 1). The POC assay consistently produced higher readings than the automated analyzer, particularly in patients with elevated suPAR levels. A significant difference between the two assays was observed only in the stroke group, whereas measurements in the control group were comparable.

3.2. Correlation between POC and analyzer measurements

In the stroke cohort, measurements obtained using the POC method showed a strong positive correlation with those from the automated analyzer (r = 0.745, p<0.001). As shown in Figure 1, agreement between the two methods was generally good, although divergence increased at higher concentrations.

Table 1. Values of plasma soluble urokinase plasminogen activator receptor

Method	Median (Interquartile range)		p
	Study group (n=27)	Controls (n=14)	
Point-of-care	7.2 (4.2–15.0)	3.95 (3.2–6.6)	<0.001*
Analyzer	5.5 (3.2–17.5)	3.8 (2.2–7.5)	0.012*
p	0.024*	0.713	

Note: *Statistically significant difference determined at p<0.05.

3.3. Diagnostic performance

Receiver operating characteristic curves analysis demonstrated that the POC method achieved higher sensitivity (96%) for identifying stroke cases compared with the automated analyzer (82%), while both assays exhibited identical specificity (57%). The asymptotic 95% confidence intervals for the two methods were 0.824–1.000 for the POC and 0.580–0.902 for the analyzer method (Figure 2). These findings suggest that the POC method may offer enhanced discriminative ability in emergency settings.

3.4. Bland–Altman analysis

The results of the Bland–Altman analysis are shown in Figure 3. There was a mean difference (bias, POC–analyzer) of +1.16 ng/mL and a standard deviation of differences of about 3.0 ng/mL (95% limits of agreement from –4.72 to +7.04) (Figure 3). The positive bias indicates that the POC method tends to yield higher suPAR concentrations compared to the analyzer. The wide limits of agreement suggest a substantial variability between the methods at the individual patient level and an increasing disagreement in higher concentration ranges.

4. Discussion

4.1. Interpretation of results

suPAR has emerged as an increasingly valuable biomarker across a wide range of acute and chronic inflammatory conditions, and recent evidence supports its expanding use in emergency and critical care settings. In the context of stroke, where early identification of high-risk patients is essential for timely intervention and allocation of resources, the ability to rapidly measure suPAR at the bedside presents an important clinical opportunity. Although computed tomography and magnetic resonance imaging remain the gold standards for diagnosing stroke, biomarkers may provide pathophysiological insights and prognostic information, particularly when imaging is delayed or when patients present atypically. This is especially

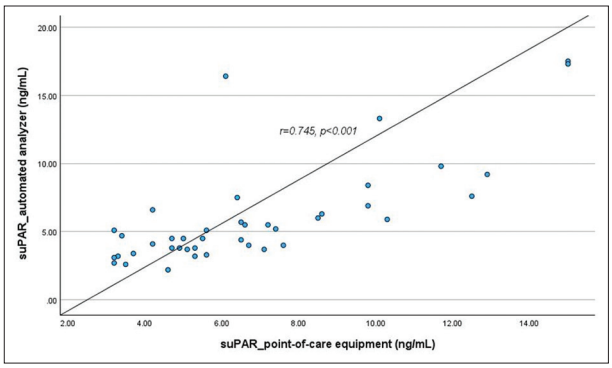

Figure 1. Scatterplot showing the correlation between the results of the measurements of plasma suPAR using two methods, POC and automated analyzer, in the study group
Abbreviations: POC: Point-of-care; suPAR: Soluble urokinase plasminogen activator receptor.

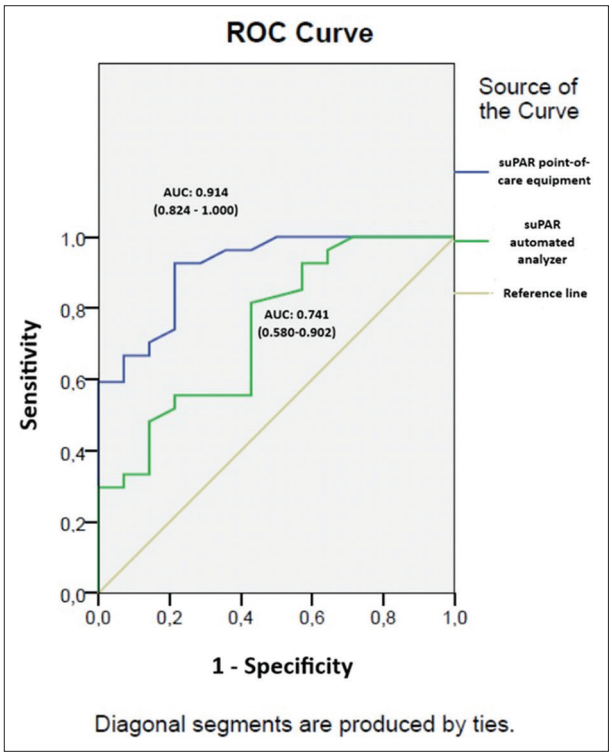

Figure 2. ROC curves of the results of the two methods, POC and analyzer
Note: Diagonal segments are produced by ties.
Abbreviations: AUC: Area under the curve; POC: Point-of-care; ROC: Receiver operating characteristics.

relevant given that inflammation is now recognized as a central component of both ischemic and hemorrhagic stroke pathophysiology. Experimental and clinical studies demonstrate that the inflammatory cascade following cerebrovascular injury contributes to blood–brain barrier disruption, neuronal death, vascular remodeling, and systemic immune activation.[10,11,12] suPAR, which reflects chronic immune activation rather than acute-phase response, may therefore capture the underlying inflammatory milieu that predisposes individuals to more severe cerebrovascular disease or poorer outcomes.

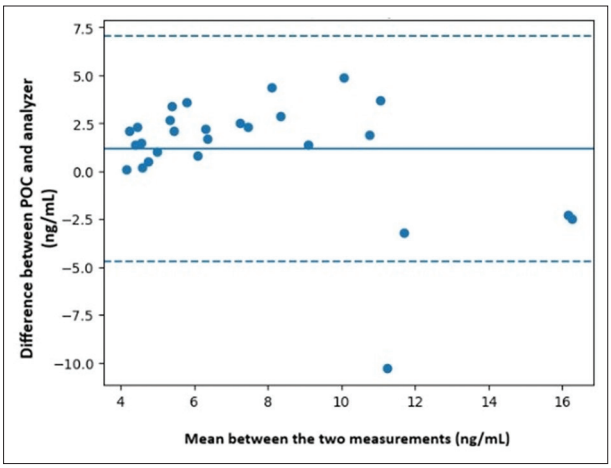

Figure 3. Bland-Altman plot for the comparison of the measurement of suPAR with a POC method and an automated analyzer
Abbreviations: POC: Point-of-care; suPAR: Soluble urokinase plasminogen activator receptor.

Our study demonstrates that in plasma samples from patients presenting with stroke, suPAR levels measured by a POC lateral flow assay (suPARnostic QT) correlated strongly with values obtained using a standard automated immunoassay (suPARnostic Turbilatex PETIA). This finding suggests good concordance in relative terms between the two methods, although absolute values differed. Importantly, the POC assay tended to overestimate suPAR at higher concentrations, consistent with the statistically significant difference in our stroke cohort.

From a clinical perspective, the POC device demonstrated slightly superior discriminative performance in ROC analysis, showing a more favorable sensitivity–specificity trade-off compared with the automated analyzer. This finding is highly relevant in EDs and triage settings, where rapid decision-making is crucial and where POC testing may facilitate early risk stratification.

The elevated suPAR levels in stroke patients compared with controls further support the hypothesis that inflammation plays a critical role in the pathophysiology of stroke. Elevated suPAR levels reflect both pre-existing chronic inflammatory state and acute immune activation in response to ischemic or hemorrhagic injury. These observations align with prior research. For example, Śmiłowska *et al*.[8] reported that suPAR concentrations strongly correlated with clinical severity in the early days after stroke. Another longitudinal study found that suPAR measured on day 3 post-stroke correlated with mRS at 24 months, demonstrating prognostic value for long-term disability, though not necessarily for mortality.[2] In addition, in a prospective cohort of patients with acute neuronal injury (ischemic stroke, subarachnoid hemorrhage, traumatic brain injury), higher suPAR levels measured around five to six days post-injury predicted unfavorable outcomes

(AUC ~ 0.66), and combining suPAR with interleukin 1β improved prognostic performance (AUC ~ 0.77).[10]

The comparison between the POC and automated analyzer methods revealed good correlation, supporting the technical reliability of the POC platform for clinical use. Notably, the POC device exhibited higher sensitivity for identifying stroke cases, suggesting that it may detect subtle elevations in suPAR that are clinically meaningful. This is consistent with prior studies in infectious diseases and COVID-19, where POC suPAR demonstrated strong concordance with enzyme-linked immunoassay-based assays and reliably identified patients at risk of respiratory deterioration or need for intensive care.[9,17] Although the slight overestimation observed at higher concentrations warrants attention, this does not diminish the clinical value of the POC method for early risk stratification. Instead, it highlights the need to interpret POC values with awareness of method-specific bias, particularly when suPAR levels fall near clinically relevant decision thresholds.

In terms of clinical workflow, the availability of a bedside suPAR test could help streamline triage decisions in emergency settings. Stroke patients often present with heterogeneous symptoms and comorbidities, and therapeutic windows for reperfusion therapies remain narrow. Biomarkers capable of predicting severity, prognosis, or the likelihood of complications, such as hemorrhagic transformation, could therefore complement clinical assessment. Although no biomarker has yet been universally adopted for this purpose, suPAR's stability, lack of circadian variation, and resistance to rapid fluctuations associated with acute stress make it a promising candidate.[7] These features contrast with other inflammatory markers, such as C-reactive protein and procalcitonin, which may fluctuate rapidly or lack specificity for chronic vascular inflammation.

Despite these strengths, our study also highlights the need for further research. The small sample size and single-center design limit generalizability. As this is only a preliminary study, additional studies should evaluate the clinical utility of suPAR in predicting functional outcomes, stroke recurrence, or complications across diverse populations and stroke subtypes. Longitudinal studies examining suPAR kinetics following stroke may also be informative, as persistent elevation after hospital admission may indicate ongoing immune dysregulation or increased risk of deterioration. There remains a need to establish clinically validated cut-off values for stroke severity and prognosis, ideally stratified by measurement method. Future work should also explore the combined use of suPAR with other biomarkers, such as neurofilament light chain, glial fibrillary acidic protein, or inflammatory cytokine panels, to determine whether multimarker strategies further improve diagnostic or prognostic accuracy.

Overall, our findings show that suPAR measurement, whether performed on a high-throughput automated analyzer or a rapid bedside device, has the potential to contribute meaningfully to the evaluation of patients with stroke. The POC assay's analytical performance, high sensitivity, and ease of use strengthen the case for its incorporation into emergency care pathways. With further validation in larger cohorts, suPAR could become a valuable component of risk-stratification tools for stroke, aiding timely decision-making and improving patient outcomes.

4.2. Biological plausibility and mechanisms

The role of suPAR in stroke may not be purely passive; elevated levels may contribute directly or indirectly to pathogenesis. uPAR and suPAR are known to mediate cell adhesion, migration, and signaling through interactions with integrins (e.g., β3 integrin), vitronectin, and other co-receptors.[1,4] The binding of suPAR to integrins may promote vascular inflammation, endothelial dysfunction, leukocyte chemotaxis, and pro-atherogenic signaling, thereby contributing to plaque instability and vascular events.[9] Epidemiological cohort data show that high suPAR is associated with increased incidence of carotid plaques, ischemic stroke, and coronary artery disease.[17]

Genetic studies further support a causal relationship: variants in the *PLAUR* gene, which encodes uPAR and leads to higher circulating suPAR levels, are associated with increased risk of atherosclerosis and cardiovascular disease. Animal models with suPAR overexpression display vascular and inflammatory changes consistent with disease promotion.[9] Therefore, suPAR may be more than a biomarker; it may be a mediator of vascular pathology, including cerebrovascular disease.

In addition, suPAR's stability over time and limited responsiveness to acute fluctuations make it a robust indicator of a patient's baseline inflammatory milieu, often termed systemic chronic subclinical inflammation.[5] A review of suPAR as a systemic chronic subclinical inflammation biomarker identified multiple characteristics aligning suPAR with chronic immune activation, such as upregulation by immune activation, pro-inflammatory functions, correlation with established inflammatory markers (e.g., C-reactive protein, interleukin 6), prediction of morbidity and mortality across diseases, independence from short-term perturbations, and responsiveness to anti-inflammatory interventions.[5]

Stroke triggers complex immune dynamics. The acute phase involves activation of inflammatory cells, cytokine release, and blood–brain barrier disruption; later phases may involve stroke-induced immunosuppression, increasing susceptibility to infection and secondary complications.[17]

suPAR, produced by activated immune cells (e.g., monocytes, T cells), integrates these processes over time and may offer a composite signal of both pre-existing risk and ongoing injury responses.

4.3. Clinical implications

Our findings have several potential clinical implications:

(i) *Rapid triage and risk stratification:* A POC suPAR test could be deployed in the ED or stroke unit to help identify high-risk patients early. Elevated suPAR on admission may flag individuals at risk of poorer outcomes, complications, or the need for more intensive monitoring

(ii) Prognostic use: Although our study did not include long-term follow-up, prior work shows that suPAR correlates with functional outcomes (e.g., mRS) months to years after stroke.[2,10] Serial suPAR measurements (POC or laboratory-based) could potentially become part of risk models to predict recovery trajectory

(iii) Therapeutic decision support: Given that suPAR is linked to underlying vascular inflammation and may be mechanistically involved in pathogenesis, interventions targeting suPAR or its upstream drivers could be explored. Future interventional studies may test whether suPAR-guided therapies (e.g., inflammatory or immunomodulatory treatments) could improve outcomes

(iv) Resource allocation and health-economic benefits: POC testing may reduce turnaround times, streamline ED workflow, and enable real-time decision-making, which is critical in stroke care. If suPAR-guided triage improves the efficacy of resource use (admission to high dependency units only for high-risk patients), it could have cost-effective implications.

4.4. Future directions

Based on our findings, several avenues for future research are warranted:

(i) Prospective longitudinal studies with larger cohorts that can incorporate both POC and analyzer-based suPAR measurements at multiple time points (e.g., admission, 24 h, 72 h, day 7, etc.), along with clinical variables (National Institutes of Health Stroke Scale), imaging, complications (e.g., infection), and long-term outcomes (mRS and mortality)

(ii) Stratify analyses by stroke subtype (ischemic vs. hemorrhagic), severity, treatment (thrombolysis, thrombectomy), and comorbidities to assess how suPAR behaves across clinical contexts

(iii) Interventional studies aiming to determine whether suPAR-guided management, including early immunomodulation or more intensive monitoring, improves outcomes

(iv) Analytical and assay-development studies to refine POC performance, extend dynamic range, improve accuracy at high concentrations, and cross-validate against reference enzyme-linked immunoassay platforms

(v) Mechanistic studies (*in vitro* and *in vivo*) to elucidate how suPAR contributes to vascular injury, blood–brain barrier disruption, leukocyte infiltration, and neuronal injury, for instance, assessing whether suPAR directly affects endothelial permeability, microglial activation, or neuronal apoptosis.

5. Conclusion

The findings of the present study support the growing body of evidence that suPAR is a promising biomarker for evaluating inflammatory activity and potential disease severity in patients with stroke. By directly comparing a rapid POC lateral flow assay with a standardized automated immunoassay, we demonstrated that suPAR can be reliably quantified across two analytically distinct platforms, showing strong correlation in the stroke cohort. Although the POC method produced slightly higher absolute values at the upper end of the concentration range, this difference did not diminish its clinical utility; instead, its superior sensitivity in distinguishing stroke patients from controls suggests practical advantages for real-time triage in emergency settings.

The key strength of the POC platform lies in its rapid turnaround time and operational simplicity. In busy EDs, particularly those with limited laboratory capacity or during high-volume situations, such as pandemics or mass casualty events, fast-acting biomarkers may meaningfully support early risk stratification. Our results indicate that suPAR, when measured at the bedside, could serve as an adjunct to clinical examination and imaging, helping clinicians identify patients who may require closer monitoring, have heightened inflammatory burden, or be at increased risk for complications. Given that inflammation is increasingly recognized as a central component of stroke pathophysiology and recovery, such a tool could integrate seamlessly into acute stroke workflows.

Our study has certain limitations, such as a small and heterogeneous sample and a lack of connection to clinical data, such as stroke subtype, severity, imaging findings, timing of sample collection relative to stroke onset, concomitant infections, comorbidities, or medications. Therefore, broader validation is necessary to fully define suPAR's role in cerebrovascular disease. Larger, multicenter studies are required to establish robust reference ranges, risk thresholds, and clinical cut-offs adapted to different stroke subtypes and patient profiles. Future research evaluating outcomes, severity, or time course should also investigate whether sequential POC measurements, taken during the 1st h and days following stroke

onset, enhance prognostic accuracy or enable early detection of secondary complications, such as infection, edema, or hemorrhagic transformation.

In summary, suPAR measurement, whether performed through rapid POC testing or routine laboratory analysis, has the potential to enhance clinical decision-making for patients with stroke. Our results demonstrate that POC testing is a viable, sensitive, and operationally valuable method that could support improved triage, individualized risk assessment, and more efficient allocation of healthcare resources in acute stroke care. With further validation, suPAR may become an integral component of future multimodal stroke assessment pathways.

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Conflict of interest

The authors declare they have no competing interests.

Author contributions

Conceptualization: Kali Makedou, Areti Kourti
Data curation: Alexandra Skoura, Eirini Keskilidou
Formal analysis: Athanasia Samara, Paraskevi Thanou
Methodology: Kali Makedou, Paraskevi Karalazou, Katerina Thisiadou
Visualization: Paraskevi Karalazou, Katerina Thisiadou
Writing–original draft: Areti Kourti
Writing–review & editing: Areti Kourti, Kali Makedou

Ethics approval and consent to participate

The study was approved by the Ethics Committee of Aristotle University of Thessaloniki (approval number: 673/22-12-2025). All measurements were performed during the diagnosis process. The study is retrospective and, therefore, informed consent was not needed.

Consent for publication

No named patient's or control's data are published; therefore, there was no need for their consent for publication.

Data availability statement

Data cannot be accessed because it can be found within the LIS system of the hospital and protected by GDPR law.

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