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CLINICAL AND LABORATORY OUTCOMES OF COMBINED L-ARGININE AND N-ACETYLCYSTEINE THERAPY FOR COMMUNITY-ACQUIRED PNEUMONIA IN PATIENTS WITH A HISTORY OF ISCHEMIC STROKE

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ABSTRACT

Background: Community-acquired pneumonia (CAP) may be clinically more severe in patients with previous ischemic stroke because neurological impairment, dysphagia, inflammation, and vascular dysfunction can interact to worsen respiratory recovery. Objective: This study evaluated clinical and laboratory outcomes associated with adjunctive L-arginine and N-acetylcysteine (NAC) in patients with CAP and a history of ischemic stroke. Methods: The study included 150 patients with CAP. Patients with previous ischemic stroke received standard therapy alone or standard therapy supplemented with L-arginine, NAC, or both agents. Outcomes included C-reactive protein (CRP), procalcitonin (PCT), vascular endothelial growth factor (VEGF), nitric oxide (NO), endothelin-1 (ET-1), National Institutes of Health Stroke Scale (NIHSS), CURB-65, and hospitalization duration. Results: At baseline, the post-stroke CAP group had higher CRP, PCT, VEGF, and ET-1 and lower NO than the non-stroke comparison group. The combined-treatment subgroup showed the largest reported changes: CRP decreased 79.5%, PCT 80.7%, VEGF 42.5%, and ET-1 46.0%, while NO increased 59.2%. Post-treatment NIHSS and CURB-65 scores were 5.8 and 1.0, and mean hospitalization was 10.4 days. Conclusion: Combined L-arginine and NAC was associated with the most favorable biomarker and clinical profile, warranting prospective controlled confirmation before causal efficacy is inferred.

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Keywords: *community-acquired pneumonia; ischemic stroke; L-arginine; N-acetylcysteine; endothelial dysfunction; inflammation.*

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Introduction

Community-acquired pneumonia (CAP) remains a major cause of acute respiratory morbidity and hospital utilization, especially among older adults and patients with substantial comorbidity. Contemporary CAP management requires prompt diagnosis, assessment of severity, appropriate antimicrobial therapy, and recognition of host factors that may alter prognosis (Metlay et al., 2019). In patients with a previous ischemic stroke, the clinical problem is more complex because persistent neurological deficits can impair airway protection, mobility, cough effectiveness, nutrition, and rehabilitation. These factors may increase vulnerability to respiratory infection even when pneumonia occurs outside the acute stroke period.

The association between stroke and pneumonia is well established. A systematic review and meta-analysis by Westendorp et al. (2011) estimated that pneumonia occurs in approximately 10% of patients

after acute stroke and is strongly associated with mortality. More recent evidence continues to identify stroke severity, dysphagia, mechanical ventilation, nasogastric tube use, age, and selected cardiometabolic comorbidities as important predictors of pneumonia after stroke (Ahmad et al., 2024). Dysphagia is particularly relevant because impaired swallowing facilitates aspiration of oropharyngeal material. Chang et al. (2022) found a markedly increased odds of pneumonia among stroke patients with dysphagia, reinforcing the importance of airway protection and swallowing assessment in neurologically vulnerable populations.

Although the present investigation concerns CAP in patients with a history of ischemic stroke rather than pneumonia arising within the first seven days of an acute cerebrovascular event, the stroke-associated pneumonia literature remains clinically informative. The Pneumonia in Stroke Consensus Group recommended standardized diagnostic terminology for pneumonia occurring early after stroke and emphasized that inflammatory biomarkers alone are insufficient for diagnosis (Smith et al., 2015). This distinction is relevant because CRP and PCT should be interpreted as components of a broader clinical picture rather than as isolated diagnostic endpoints. PCT has demonstrated prognostic associations in pneumonia, but its performance varies by disease severity and timing; longitudinal change may be more informative than a single measurement (Liu et al., 2016).

A second pathophysiological dimension is endothelial dysfunction. Systemic infection and inflammation can disturb vascular tone, microcirculatory regulation, and endothelial signaling. Endothelin-1 (ET-1) is a potent vasoconstrictor implicated in vascular dysfunction, whereas nitric oxide (NO) has vasodilatory, antiplatelet, and endothelial-protective actions (Böhm & Pernow, 2007). Vascular endothelial growth factor (VEGF), although essential for angiogenic signaling and endothelial homeostasis, may increase in inflammatory and hypoxic states and can therefore serve as an indirect indicator of altered vascular biology. In patients who already have cerebrovascular disease, an infection-related shift toward vasoconstriction, oxidative stress, and impaired NO bioavailability may be clinically important.

L-arginine and N-acetylcysteine (NAC) offer a biologically plausible combination for addressing this inflammatory-endothelial interface. L-arginine is the physiological substrate for nitric oxide synthase and therefore contributes to endogenous NO formation (Böger, 2007). Meta-analytic evidence suggests that short-term L-arginine supplementation can improve flow-mediated dilation in selected patients with impaired baseline endothelial function, although effects are heterogeneous and not consistently demonstrated across cardiovascular and metabolic populations (Bai et al., 2009; Rodrigues-Krause et al., 2019). NAC, meanwhile, acts as a mucolytic agent and as a cysteine donor for glutathione synthesis. Its antioxidant effects depend substantially on cellular glutathione biology rather than on direct radical scavenging alone (Rushworth & Megson, 2014).

The potential complementarity of these agents is therefore clinically interesting: L-arginine may support NO-dependent vascular regulation, while NAC may improve mucus clearance and support intracellular antioxidant capacity. However, biological plausibility does not by itself establish therapeutic efficacy, and evidence for either agent as an adjunct specifically in post-stroke CAP is limited. The objective of the present study was to evaluate the clinical and laboratory outcomes associated with standard therapy, L-arginine, NAC, and combined L-arginine plus NAC in patients with CAP and a history of ischemic stroke. Particular attention was given to inflammatory markers, endothelial-regulatory markers, neurological status, pneumonia severity, and length of hospitalization.

Method

Study design and participants. The available study record describes a clinical and laboratory study of 150 patients diagnosed with CAP and treated in a hospital setting. The principal study population included patients with CAP who had a previous ischemic stroke and were in the late recovery or remote period after the cerebrovascular event. A comparison group consisted of patients with CAP without a history of stroke. CAP was established from clinical, laboratory, and instrumental findings consistent with

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accepted diagnostic approaches. The original record did not provide recruitment dates, a detailed sampling frame, or a participant flow diagram; consequently, these elements cannot be reconstructed without additional primary documentation.

Treatment groups. Patients with CAP and a history of ischemic stroke were divided into four therapeutic subgroups: IA, standard therapy; IB, standard therapy plus L-arginine; IC, standard therapy plus NAC; and ID, standard therapy plus both L-arginine and NAC. The source results table reports n = 25 for each treatment subgroup. The original study record does not specify the method of allocation, medication doses, routes, treatment duration, or the complete components of standard therapy. These details are important for reproducibility and should be added from the clinical protocol before journal submission. In this revised manuscript, treatment effects are therefore described as associations with the reported therapeutic regimens rather than as proof of randomized causal efficacy.

Laboratory outcomes. Systemic inflammation and infection-related activity were assessed using CRP and PCT. Endothelial and vascular-regulatory status was characterized using VEGF, NO, and ET-1. These markers were measured before treatment and after treatment. The source manuscript reports group means and standard deviations for the baseline comparison and percentage changes for selected treatment outcomes. Assay manufacturers, sampling times, specimen handling procedures, and analytic coefficients of variation were not available in the provided record and are not inferred here.

Clinical outcomes. Neurological status was assessed using the National Institutes of Health Stroke Scale (NIHSS), a standardized neurological deficit scale originally developed for acute cerebral infarction assessment (Brott et al., 1989). Pneumonia severity was assessed using CURB-65, which incorporates confusion, urea concentration, respiratory rate, blood pressure, and age of 65 years or older and is widely used to stratify mortality risk in CAP (Lim et al., 2003). Duration of hospitalization was also recorded as a pragmatic indicator of clinical recovery and resource use.

Statistical presentation. The revised analysis preserves the numerical values and significance statements reported in the source manuscript. Baseline values are presented as mean ± standard deviation. The source reports highly significant between-group differences for the baseline biomarkers ($p < .001$) and significant post-treatment differences for clinical outcomes ($p < .001$), but it does not identify the statistical tests, assumptions, multiplicity adjustments, or exact comparison model. No new inferential tests were generated because patient-level data were not available. Relative baseline differences discussed below were calculated descriptively from the reported means, and the figures reproduce reported percentages or means only. This approach strengthens transparency while avoiding unsupported statistical reconstruction.

Results

Baseline inflammatory profile. Patients with CAP and a history of ischemic stroke demonstrated a more pronounced inflammatory profile than patients with CAP without previous stroke (Table 1). Mean CRP was 82.6 ± 24.8 mg/L in the post-stroke group compared with 61.3 ± 19.5 mg/L in the comparison group, corresponding to an approximately 34.7% higher reported mean. PCT showed a similar pattern: 3.59 ± 1.38 ng/mL versus 2.67 ± 1.17 ng/mL, or approximately 34.5% higher in the post-stroke group. These findings indicate greater systemic inflammatory and infection-related activity in the neurologically vulnerable cohort at baseline.

Baseline endothelial-regulatory profile. The vascular markers showed an additional imbalance. VEGF was 486.5 ± 82.4 pg/mL in patients with previous ischemic stroke compared with 312.8 ± 64.7 pg/mL in the non-stroke CAP group, representing an approximately 55.5% higher mean. ET-1 was also higher (4.86 ± 0.82 vs. 3.74 ± 0.69 pg/mL; approximately 29.9% higher). In contrast, NO was lower in the post-stroke group (24.8 ± 4.9 vs. 29.7 ± 5.1 μmol/L), an approximately 16.5% reduction relative to the comparison mean. Considered together, increased ET-1 and reduced NO are consistent with an unfavorable vasoregulatory balance, while elevated VEGF may reflect inflammatory, hypoxic, or reparative endothelial signaling.

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Table 1

Baseline Indicators of Systemic Inflammation and Endothelial Regulation

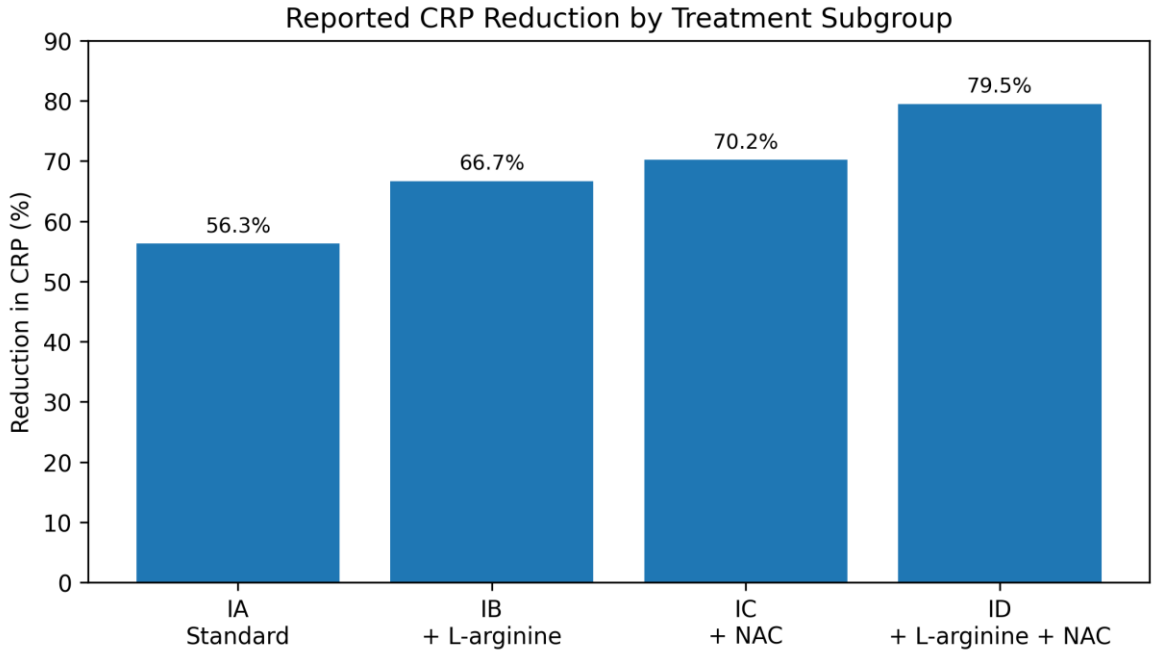
Marker	CAP + previous ischemic stroke (n = 38)	CAP without stroke (n = 20)
CRP, mg/L	82.6 ± 24.8*	61.3 ± 19.5
Procalcitonin, ng/mL	3.59 ± 1.38*	2.67 ± 1.17
VEGF, pg/mL	486.5 ± 82.4*	312.8 ± 64.7
NO, µmol/L	24.8 ± 4.9	29.7 ± 5.1*
ET-1, pg/mL	4.86 ± 0.82*	3.74 ± 0.69

Note. Values are mean ± SD. *p < .001 as reported in the source manuscript. The relationship of these baseline denominators to the total sample of 150 requires clarification from the original study records.

Inflammatory response to treatment. All four therapeutic subgroups demonstrated reductions in CRP, but the magnitude differed across regimens. The reported decrease was 56.3% with standard therapy (IA), 66.7% with L-arginine added (IB), 70.2% with NAC added (IC), and 79.5% with the combined regimen (ID). Thus, the combined subgroup had the largest reported reduction in CRP (Figure 1). For PCT, the source manuscript provides the most detailed percentage for the combined group, in which PCT decreased by 80.7%. Because corresponding PCT percentages for all other subgroups were not provided, they are not estimated in this revision.

Figure 1

Reported Percentage Reduction in C-Reactive Protein by Treatment Subgroup



Note. Values reproduce the reported percentage decrease in CRP for subgroups IA–ID; no unreported values were estimated.

Endothelial response to treatment. The combined L-arginine plus NAC subgroup also showed the largest reported favorable change in endothelial markers. VEGF decreased by 42.5% and ET-1 decreased by 46.0%, while NO increased by 59.2%. The direction of these changes is biologically coherent with improved NO availability and reduced vasoconstrictor signaling. Nevertheless, the absence of complete post-treatment means and dispersion measures for every subgroup limits calculation of standardized effect sizes or confidence intervals. Table 2 therefore focuses on the clinical outcomes for which complete subgroup means are reported, while Table 3 summarizes the documented percentage changes without extrapolating missing values.

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Clinical severity and hospitalization. Clinical improvement was observed in each subgroup, with the most favorable post-treatment values reported for subgroup ID. Mean NIHSS changed from 9.3 ± 2.0 before treatment to 5.8 ± 1.5 after treatment in ID. Corresponding post-treatment NIHSS values were 7.8 ± 1.9 in IA, 6.9 ± 1.7 in IB, and 6.6 ± 1.6 in IC. CURB-65 showed a parallel pattern: subgroup ID decreased from 2.7 ± 0.6 to 1.0 ± 0.3, compared with post-treatment values of 1.8 ± 0.5 in IA, 1.5 ± 0.4 in IB, and 1.4 ± 0.4 in IC (Table 2). The reported pre-treatment subgroup differences were not significant ($p > .05$), whereas post-treatment differences were reported as $p < .001$.

Table 2

Neurological and Pneumonia Severity Before and After Treatment

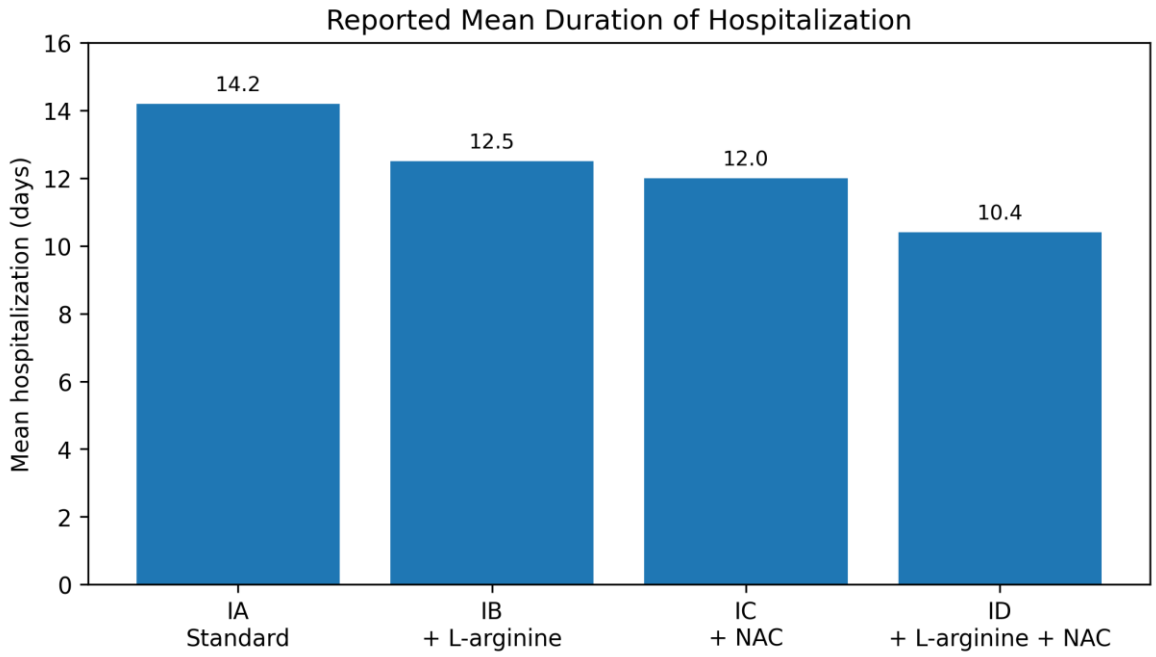
Outcome	IA (n = 25)	IB (n = 25)	IC (n = 25)	ID (n = 25)
NIHSS, before	9.4 ± 2.1	9.2 ± 2.3	9.5 ± 2.2	9.3 ± 2.0
NIHSS, after	7.8 ± 1.9	6.9 ± 1.7	6.6 ± 1.6	5.8 ± 1.5
CURB-65, before	2.8 ± 0.6	2.7 ± 0.5	2.8 ± 0.5	2.7 ± 0.6
CURB-65, after	1.8 ± 0.5	1.5 ± 0.4	1.4 ± 0.4	1.0 ± 0.3

Note. Values are mean ± SD. The source manuscript reports $p > .05$ for baseline subgroup comparisons and $p < .001$ for post-treatment subgroup comparisons; the specific statistical test was not provided.

Length of hospital stay was shortest in the combined-treatment subgroup. Mean hospitalization was 14.2 ± 2.1 days in IA, 12.5 ± 1.8 days in IB, 12.0 ± 1.7 days in IC, and 10.4 ± 1.5 days in ID (Figure 2). Relative to standard therapy, the combined regimen was associated with a mean stay that was 3.8 days shorter, or approximately 26.8% lower. This outcome is clinically relevant because length of stay integrates multiple domains of recovery, although it can also be influenced by discharge practices, rehabilitation access, social circumstances, and institutional policy.

Figure 2

Reported Mean Duration of Hospitalization by Treatment Subgroup



Note. Mean ± SD hospitalization values were IA 14.2 ± 2.1, IB 12.5 ± 1.8, IC 12.0 ± 1.7, and ID 10.4 ± 1.5 days; $p < .001$ was reported for the subgroup comparison.

Table 3

Summary of Reported Treatment-Associated Changes

Outcome	IA	IB	IC	ID
CRP reduction	56.3%	66.7%	70.2%	79.5%
PCT reduction	NR	NR	NR	80.7%

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Outcome	IA	IB	IC	ID
VEGF reduction	NR	NR	NR	42.5%
ET-1 reduction	NR	NR	NR	46.0%
NO increase	NR	NR	NR	59.2%
Hospitalization	14.2 days	12.5 days	12.0 days	10.4 days

Note. NR = not reported in the supplied manuscript. This table intentionally leaves unavailable subgroup percentages blank rather than estimating them.

Discussion

The principal finding of this study is that CAP in patients with a history of ischemic stroke was accompanied by a more adverse inflammatory and endothelial-regulatory profile than CAP in patients without previous stroke, and that the subgroup receiving both L-arginine and NAC had the most favorable reported laboratory and clinical changes. The results extend the clinical observation that stroke survivors remain vulnerable to respiratory complications by suggesting that vascular-regulatory disturbances may coexist with infection-related inflammation. This interpretation is consistent with the broader literature showing that pneumonia is a consequential complication of stroke and that neurological severity, dysphagia, and impaired airway protection contribute to risk (Ahmad et al., 2024; Chang et al., 2022; Westendorp et al., 2011).

The baseline CRP and PCT findings are clinically plausible. CRP is a nonspecific acute-phase reactant, while PCT can provide additional information regarding systemic bacterial inflammatory activity and prognosis. In a meta-analysis of pneumonia, elevated PCT was associated with increased mortality risk, although diagnostic and prognostic performance varied by clinical setting and threshold (Liu et al., 2016). The present study should therefore not be interpreted as establishing PCT as an independent diagnostic marker in post-stroke CAP. Rather, the higher PCT and CRP concentrations support the presence of a more intense systemic inflammatory response in the post-stroke cohort. This distinction is consistent with the PISCES consensus, which cautioned against overreliance on biomarkers for defining pneumonia after stroke (Smith et al., 2015).

The endothelial findings provide a mechanistic hypothesis for the observed treatment pattern. NO is central to vascular homeostasis, and reduced NO bioavailability is a common feature of endothelial dysfunction. ET-1 has opposing vasoconstrictor and proliferative effects and is implicated in vascular disease (Böhm & Pernow, 2007). The combination of lower NO and higher ET-1 at baseline can therefore be viewed as a marker pattern compatible with impaired endothelial regulation. Elevated VEGF is less specific: it may reflect endothelial activation, hypoxia, inflammation, or compensatory angiogenic signaling. Consequently, the reduction in VEGF after therapy should be interpreted as normalization of an altered signaling environment rather than as evidence that lower VEGF is universally beneficial.

L-arginine offers a plausible pathway through which NO signaling might be supported because it is a substrate for nitric oxide synthase (Böger, 2007). Earlier meta-analysis suggested that short-term supplementation improves flow-mediated dilation when baseline endothelial function is impaired (Bai et al., 2009). However, a later systematic review and meta-analysis found no consistent overall improvement in selected endothelial markers across heterogeneous cardiovascular and metabolic populations (Rodrigues-Krause et al., 2019). This mixed evidence is important for interpreting the present results. The 59.2% increase in NO in subgroup ID is noteworthy, but without placebo control, allocation details, treatment dose, complete post-treatment distributions, and individual-level data, it cannot establish a specific pharmacological effect of L-arginine.

NAC may have contributed through different but potentially complementary mechanisms. It is a well-established mucolytic agent and provides cysteine for intracellular glutathione synthesis. Rushworth and Megson (2014) emphasized that NAC's antioxidant benefit depends on the cellular context and the capacity to restore glutathione, rather than representing a simple direct antioxidant effect. In CAP, improved mucus rheology could theoretically facilitate airway clearance, while glutathione support may

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help buffer oxidative stress associated with infection and inflammation. The stronger CRP reduction in the NAC subgroup than in standard therapy, and the still larger reduction in the combined subgroup, are consistent with complementarity, but the available data cannot separate pharmacological synergy from baseline differences, co-interventions, or nonrandom allocation.

The clinical outcomes strengthen the potential relevance of the laboratory findings. Subgroup ID had the lowest post-treatment NIHSS and CURB-65 scores and the shortest hospitalization. CURB-65 is a validated severity tool for CAP, with increasing scores associated with increasing mortality risk (Lim et al., 2003). The reduction from 2.7 to 1.0 in the combined subgroup therefore suggests movement toward a lower-risk clinical category, although CURB-65 was designed primarily for initial severity assessment and should not be treated as a stand-alone treatment-response instrument. Similarly, NIHSS is a validated measure of neurological deficit (Brott et al., 1989), but changes in patients remote from stroke may reflect multiple influences, including systemic illness, hydration, oxygenation, delirium, and rehabilitation, rather than direct neurorestorative effects of the adjunctive drugs.

The length-of-stay finding may be the most readily interpretable clinical signal. A mean stay of 10.4 days in subgroup ID compared with 14.2 days under standard therapy represents a 3.8-day difference. If replicated in a controlled trial, such a reduction could have substantial implications for bed utilization, rehabilitation transitions, and patient costs. Yet hospital stay is also susceptible to confounding by discharge criteria, social support, availability of post-acute care, clinician practice, and complications not captured in the dataset. For that reason, future studies should prespecify objective discharge criteria and report time-to-clinical-stability outcomes in addition to total days hospitalized.

From a broader clinical perspective, the data support a multimodal view of CAP in stroke survivors. Standard antimicrobial management remains fundamental and should follow established CAP guidance (Metlay et al., 2019). Adjunctive strategies should not distract from aspiration prevention, dysphagia screening, mobilization, respiratory support, and management of comorbidity. The stroke literature demonstrates that dysphagia substantially increases pneumonia risk (Chang et al., 2022), and the diagnosis of pneumonia after stroke is methodologically challenging when clinical signs overlap with neurological impairment (Kishore et al., 2015; Smith et al., 2015). Any future trial of L-arginine and NAC should therefore standardize swallowing assessment, aspiration risk, pathogen testing, antimicrobial therapy, and respiratory care to isolate the contribution of the adjunctive regimen.

The present study is best viewed as hypothesis-generating evidence for a combined endothelial-supportive and antioxidant/mucolytic approach. The consistency across several outcome domains—CRP, PCT, VEGF, NO, ET-1, NIHSS, CURB-65, and hospital stay—is encouraging. At the same time, mechanistic coherence can increase confidence only when methodological rigor is adequate. The strongest next step would be a prospective, randomized, adequately powered trial with concealed allocation; explicit L-arginine and NAC dosing; standardized antibiotic protocols; serial biomarker sampling; prespecified primary and secondary outcomes; adverse-event monitoring; and multivariable adjustment for age, dysphagia, stroke severity, comorbidities, renal function, and baseline pneumonia severity. Such a design would permit estimation of effect sizes and confidence intervals and would clarify whether the apparent advantage of combined therapy is reproducible and clinically meaningful.

Limitations and Reporting Considerations

Several limitations materially affect interpretation. First, the source manuscript reports 150 total participants, while the treatment table accounts for 100 patients across four subgroups of 25, and the baseline biomarker table reports group sizes of 38 and 20. The relationship between these denominators is not explained. This discrepancy should be reconciled against the original case report forms or study database before submission. Second, the allocation method is not described, so selection bias and baseline imbalance cannot be excluded. Third, treatment dose, route, duration, adherence, and adverse events are

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absent. Fourth, laboratory assay methods and post-treatment means with standard deviations are incomplete, preventing independent effect-size calculation.

Fifth, the statistical methods underlying reported p values are unspecified, and patient-level data were unavailable for reanalysis. Sixth, key confounders—including age distribution, sex, stroke location, dysphagia, aspiration status, functional dependence, renal function, smoking, diabetes, antimicrobial regimen, pathogen profile, and oxygen requirement—are not reported by subgroup. Seventh, the study appears to be single-center or limited-center, which may restrict generalizability. Finally, ethics approval and informed-consent information were not included in the supplied manuscript. These reporting gaps do not invalidate the observed numerical patterns, but they substantially limit causal interpretation and should be addressed in any final journal version.

Conclusion

Patients with CAP and a history of ischemic stroke demonstrated higher reported CRP, PCT, VEGF, and ET-1 concentrations and lower NO concentrations than patients with CAP without previous stroke, suggesting a combined inflammatory and endothelial-regulatory disturbance. Across the reported therapeutic subgroups, standard therapy supplemented with both L-arginine and NAC was associated with the largest reductions in CRP, PCT, VEGF, and ET-1, the largest increase in NO, the lowest post-treatment NIHSS and CURB-65 scores, and the shortest mean hospitalization.

These findings provide a biologically plausible rationale for investigating combined L-arginine and NAC as adjunctive therapy in neurologically vulnerable patients with CAP. However, incomplete reporting of participant flow, treatment allocation, dosing, assays, and statistical methods precludes firm causal conclusions. The regimen remains investigational and requires confirmation in rigorously reported prospective randomized trials before benefit beyond guideline-concordant CAP and stroke care can be established.

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