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Evaluation of Stroke-Associated Infections: A Single-Center Retrospective Cohort Study

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Abstract

Background

Stroke-associated infections (SAIs) are complications that develop after acute ischemic stroke (AIS) and dramatically increase in-hospital morbidity and mortality. Despite the known pathophysiology, reliable clinical factors to predict the development of infection in the early period are still limited. The aim of this study is to identify practical clinical risk factors that predict the development of infection in acute ischemic stroke patients.

Materials and Methods

This single-center, retrospective cohort study included patients hospitalized with a diagnosis of AIS between January 2025 and January 2026. Demographic data, stroke characteristics, baseline laboratory parameters, and National Institutes of Health Stroke Scale (NIHSS) scores were recorded. The primary endpoint was determined as the development of healthcare-associated infection during hospitalization. Independent risk factors were analyzed using a multivariate logistic regression model, and predictive power was evaluated with the Receiver Operating Characteristic (ROC) curve.

Results

A total of 139 AIS patients (mean age 72.2 ± 12.1 years; 49.6% male) were evaluated. During clinical follow-up, 19 patients (13.6%) developed an infection, with pneumonia (7.2%) and urinary tract infections (6.5%) being the most common. Patients who developed an infection were significantly older (79.8 ± 8.0 vs. 70.9 ± 12.2; $p < 0.001$) and had a higher baseline NIHSS score (median 10 vs. 6; $p < 0.001$) compared to those who did not. Baseline inflammatory biomarkers such as C-reactive protein (CRP) and neutrophil-to-lymphocyte ratio (NLR) did not reach statistical significance ($p > 0.05$). In multivariate analysis, being over 69 years of age (OR: 8.65, 95% CI: 1.79–41.83, $p = 0.007$) and having an NIHSS score above 8 (OR: 6.49, 95% CI: 2.17–19.42, $p < 0.001$) were identified as strong independent predictors for infection. The combined model of these two variables showed good discrimination (AUC: 0.770). In-hospital mortality was dramatically higher in the infection group (42.1% vs. 3.3%; $p < 0.001$).

Conclusion

Advanced age (> 69) and high stroke severity at admission (NIHSS > 8) are strong and independent clinical predictors for the development of secondary infections. Since routine inflammatory markers may be insufficient to reflect the early phase of stroke-induced immunosuppression, the importance of these practical bedside parameters increases. To prevent the development of infection and the associated

increased mortality in this high-risk patient group, new and effective preventive strategies are needed beyond the standard clinical approach.

Introduction

Acute ischemic stroke (AIS) is one of the leading causes of mortality and long-term severe morbidity worldwide [1]. Approximately two-thirds of all stroke cases globally occur in developing countries, including ours; similarly, 80–85% of all stroke-related morbidities and mortalities take place in this geography. One of the most important factors determining the clinical outcome in the course of AIS is the presence and severity of systemic complications developing in the acute phase [2]. The most critical sub-category of these clinical complications consists of stroke-associated infections (SAIs). Although the frequency of these infections is reported in a wide range depending on study populations in the literature, recent meta-analyses indicate that the cumulative post-stroke infection rate shows a decreasing trend and remains around 10–15% in modern stroke cohorts [3]. Among post-stroke infections, the most frequently observed conditions in clinical practice are stroke-associated pneumonia (SAP) and urinary tract infections (UTIs), respectively. Large-scale epidemiological data reveal that the frequency of both SAP and UTIs in stroke patients is individually around 10%. SAIs, and particularly SAP, are the most common and devastating clinical conditions that dramatically increase in-hospital morbidity and mortality [4–6]. These infections not only increase in-hospital mortality but also lead to prolonged hospital stays, increased treatment costs, and consequently, a significant rise in long-term functional morbidity [7].

Underlying stroke-associated infections are mechanical factors such as dysphagia, impaired consciousness, and aspiration, as well as the stroke-induced systemic immune dysfunction (SIDS) triggered by severe neurological damage [8]. Furthermore, although infarct volume, immunosenescence, and certain immunosuppressive mechanisms at the molecular level prepare a cellular ground for infection [9, 10], the main reflection of this susceptibility in daily clinical practice is monitored through measurable parameters such as the patient's age, the severity of the stroke experienced (NIHSS) [11], and prolonged hospital/intensive care unit stays [3, 10, 12]. However, data in the literature regarding reliable clinical factors to predict post-stroke infection development in the early phase and the direct effects of these infections on mortality remain limited.

Addressing the data gap in the current literature, the aim of this study is to identify practical and independent risk factors that will allow us to predict the development of healthcare-associated secondary infections in acute ischemic stroke patients in the early period.

Materials and Methods

Study Design and Patient Selection Approval for this study was obtained from the Ordu University Non-Interventional Scientific Research Ethics Committee on 30.07.2026 (Decision No: 2026/117). The research was planned as a single-center, retrospective cohort study in a secondary care state hospital in

accordance with the principles of the Declaration of Helsinki. Patients who applied to the emergency department between 01.01.2025 and 01.01.2026, were hospitalized with a preliminary diagnosis of acute ischemic stroke based on clinical evaluation, and completed their follow-up and treatment at our hospital were included in the study. Patients were identified by screening the International Classification of Diseases (ICD) codes through the Hospital Information Management System. Diagnoses were radiologically confirmed with cranial imaging results such as Computed Tomography and/or Magnetic Resonance Imaging, alongside the patients' clinical findings.

Patients who had a stroke diagnosis in the system codes but lacked sufficient clinical and laboratory data, those without acute ischemic stroke findings on neuroimaging, those diagnosed with primary intracranial hemorrhage on radiological evaluation, and those referred to another center for advanced investigation/treatment after emergency admission or during the early period of their hospitalization were excluded from the study. Furthermore, to evaluate only healthcare-associated infections in line with the study's aim, patients who had an active infection at the time of emergency admission or who developed an infection within the first 48 hours of hospitalization were also excluded.

Data Collection and Variables Patients' medical records were obtained retrospectively by scanning the hospital system. Within the scope of the study, the demographic characteristics and comorbidities of the patients were recorded. To determine stroke characteristics, radiological imaging reports were examined to identify stroke localizations, and stroke severity scores were calculated using the National Institutes of Health Stroke Scale (NIHSS) based on clinical data. Ischemic stroke etiology was classified according to the TOAST criteria. The patients' laboratory parameters at the time of admission to the emergency department (white blood cells, hemoglobin, platelets, neutrophils, lymphocytes, neutrophil-to-lymphocyte ratio [NLR], C-reactive protein [CRP], creatinine, urea, glucose, AST, ALT, sodium, and potassium levels) were also added to the dataset to be analyzed as potential biomarkers predicting infection development.

Recording Clinical Course and Infection Data Throughout their hospital stays, the patients' intensive care unit (ICU) requirements, mechanical ventilator needs, length of time on the mechanical ventilator, and total length of ICU stays were recorded on a daily basis. Healthcare-associated infections developing during ward and ICU follow-ups of all patients with and without ICU needs were examined. In this context, whether an infection developed, the time elapsed from admission to the diagnosis of infection, specific infection diagnoses made, and the pathogens isolated from clinical samples were recorded.

All healthcare-associated infection diagnoses (developing 48 hours after admission) evaluated in the study were retrospectively confirmed based on expert consultations and follow-up notes carried out during routine clinical operations by our hospital's Infectious Diseases and Clinical Microbiology clinic. The Centers for Disease Control and Prevention (CDC) National Healthcare Safety Network (NHSN) standard surveillance criteria were used as the basis for diagnosing infections. In this context:

- The diagnosis of healthcare-associated pneumonia was made by radiologically demonstrating new or progressive infiltration/consolidation areas on chest X-ray or thorax computed tomography, in

addition to clinical and laboratory findings such as new-onset purulent sputum, fever (> 38°C), or leukocytosis/leukopenia.

- The diagnosis of healthcare-associated urinary tract infection was confirmed by the detection of pyuria in complete urinalysis and significant pathogen growth (≥ 10^5 CFU/mL) in urine cultures taken under appropriate conditions, in the presence of clinical symptoms such as fever, dysuria, or suprapubic tenderness.

Clinical Outcome The primary endpoint of the study was defined as the development of healthcare-associated infection during the patients' hospital stay. Secondary endpoints were recorded as the patients' total length of hospital stay, discharge status, in-hospital mortality rates, and the time elapsed from the day of admission to the day of exitus in patients who developed mortality.

Statistical Analysis Statistical analyses of the obtained data were performed using SPSS software. The conformity of continuous variables to normal distribution was evaluated using visual methods and analytical methods such as the Kolmogorov-Smirnov and Shapiro-Wilk tests. Descriptive statistics were expressed as mean ± standard deviation for normally distributed continuous variables, and as median and interquartile range (IQR) for non-normally distributed continuous variables. Categorical variables were presented as numbers and percentages. In comparing two independent groups, Student's t-test or the Mann-Whitney U test was used for continuous variables, depending on the distribution of the data. Inter-group comparisons of categorical variables were made using Pearson's Chi-Square test or Fisher's exact test based on expected cell frequencies. To evaluate potential relationships between continuous and ordinal clinical variables, Pearson or Spearman correlation analyses were applied appropriately for the data distribution. Logistic regression analysis was used to identify independent risk factors predicting the development of infection. Receiver Operating Characteristic (ROC) curve analysis was performed to determine the discrimination power and cut-off values of the fundamental clinical parameters found to be significant. Area Under the Curve (AUC) values, sensitivity, and specificity rates were calculated for each independent variable and the multiple combined prediction model derived from these variables via logistic regression analysis. A two-tailed p-value of < 0.05 was considered statistically significant in all analyses.

Results

Demographic and Baseline Clinical Characteristics Of the 139 acute ischemic stroke patients included in the study, 69 (49.6%) were male and 70 (50.4%) were female. The mean age of the entire cohort was 72.2 ± 12.1 years, the median stroke severity (NIHSS) was 6 (IQR: 4–8), and the median length of hospital stay was 4 (IQR: 3–6) days. During clinical follow-up, 19 patients (13.6%) developed a stroke-associated secondary infection. The mean age (79.8 ± 8.0) and median NIHSS score (10, IQR: 6–13) of the patients in the infection group were statistically significantly higher than those in the non-infection group ($p < 0.001$ and $p < 0.001$, respectively). Similarly, the length of hospital stay for patients who developed an infection was found to be significantly longer ($p < 0.001$). No significant difference was observed between the groups in terms of gender distribution ($p = 0.645$) (Table 1).

Table 1
Demographic and Baseline Clinical Characteristics of the Patients

Age (years), mean ± SD	**All Patients (n = 139)**	**No Infection (n = 120)**	**Infection (n = 19)**	**p-value**
	72.2 ± 12.1	**70.9 ± 12.2**	**79.8 ± 8.0**	**< 0.001**
Gender (Male), n (%)	69 (49.6%)	61 (50.8%)	8 (42.1%)	0.645
Stroke Severity (NIHSS), median (IQR)	6 (4–8)	6 (4–8)	10 (6–13)	< 0.001
Length of Stay (days), median (IQR)	4 (3–6)	4 (3–5)	12 (8–34)	< 0.001

SD: Standard Deviation; IQR: Interquartile Range; NIHSS: National Institutes of Health Stroke Scale; n: Number of patients. A p-value < 0.05 was considered statistically significant.

Comorbidities, Stroke Etiology, and Localization At least one comorbidity was present in 82.7% (n = 115) of the patients, with the most common being hypertension (50.4%) and diabetes mellitus (35.3%). Although a borderline difference (p = 0.078) was detected between the groups in terms of ischemic stroke etiology (TOAST), no statistical difference was found between the infection and non-infection groups concerning the presence of comorbidities or specific chronic diseases (p > 0.05). In the radiological evaluation, 39.6% of the ischemic stroke lesions in the entire cohort had right hemisphere involvement, 38.1% left hemisphere, and 21.6% bilateral involvement. Corticosubcortical (30.2%) and subcortical (23.7%) infarcts were the most frequently observed; no statistically significant relationship was found between the hemisphere affected or the anatomical lesion localization and the development of infection (p > 0.05). The detailed comorbidity profile, TOAST etiology, and radiological characteristics of the entire cohort are presented in Supplementary Table 1 and Supplementary Table 2.

Laboratory Parameters When examining the initial laboratory values at emergency admission, although the CRP level was proportionally higher in the infection group, it did not reach statistical significance (p = 0.067). Hemogram parameters, kidney, and liver function tests were similar between the groups (p > 0.05). All biochemical and hematological laboratory data are presented in detail in Supplementary Table 3.

Infection Characteristics, Clinical Course, and Mortality Analyzing the developed infection foci revealed that the most common complication was stroke-associated pneumonia, detected in 7.2% of the entire cohort (n = 10, 52.6% of the infected cases), followed by urinary tract infections in 6.5% of the entire cohort (n = 9, 47.4% of the infected cases). The most frequently isolated microbiological agent in cultures was *Staphylococcus aureus* (31.6%) (Supplementary Table 4). Time-to-event analysis showed that patients developed an infection at a median of 4 days (IQR: 3–9) after hospital admission, and remained under medical treatment/intensive care for a median of an additional 8 days (IQR: 2–24) post-diagnosis. The ICU admission rate in the infection group (78.9%) was significantly higher than in the non-infection group (8.3%) (p < 0.001). All 6 patients who required mechanical ventilation (MV) were in the

infection group (p < 0.001). The total mortality rate of the patients followed during the study was 8.6% (n = 12). While 4 patients (3.3%) in the non-infection group died due to stroke severity and other causes, this number was 8 (42.1%) in the infection group. Following statistical analysis, a highly significant relationship was found between mortality, which is the secondary clinical endpoint, and the development of secondary infection (p < 0.001) (Table 2).

Table 2
Comparison of Ischemic Stroke Patients in Terms of Clinical Course and Mortality

Clinical Course	**All Patients (n = 139)**	**No Infection (n = 120)**	**Infection (n = 19)**	**p-value**
Intensive Care Unit (ICU) Admission, n (%)	25 (18.0%)	10 (8.3%)	15 (78.9%)	**< 0.001**
ICU Length of Stay (days), median (IQR)*	-	4.5 (3.0-9.2)	12.5 (5.7–23.7)	**0.043**
Need for Mechanical Ventilation, n (%)	6 (4.3%)	0 (0.0%)	6 (31.6%)	**< 0.001**
Clinical Outcome				
In-Hospital Mortality, n (%)	12 (8.6%)	4 (3.3%)	8 (42.1%)	**< 0.001**

ICU: Intensive Care Unit; IQR: Interquartile Range; n: Number of patients. Categorical variables are presented as frequencies and percentages (%); Fisher's Exact Test was used for comparisons between groups due to expected frequencies being < 5. ICU length of stay was calculated using the Mann-Whitney U test only for patients admitted to the ICU (n = 25). A p-value < 0.05 was considered statistically significant.

Logistic Regression and ROC Curve Analysis in Predicting Infection ROC curve analysis was performed to determine the discrimination power of the baseline variables found to be statistically significant in predicting post-stroke infection risk. When individual parameters were evaluated, being over 69 years of age (AUC: 0.722, Sensitivity: 94.7%, Specificity: 42.5%) and having an NIHSS score above 8 (AUC: 0.747, Sensitivity: 63.2%, Specificity: 74.2%) were identified as the main cut-off values predicting the development of infection. According to the multivariate logistic regression model, being over 69 years of age independently increases the infection risk by 8.65 times (OR: 8.65, 95% CI: 1.78–41.82, p = 0.007) and having a stroke severity (NIHSS) score above 8 increases it by 6.49 times (OR: 6.49, 95% CI: 2.17–19.41, p = 0.001) (Table 3). The discrimination power of the combined prediction model obtained by integrating these two basic clinical parameters at the time of emergency admission in predicting infection was found to be good (AUC: 0.770) (Fig. 1).

Table 3
Logistic Regression and ROC Analysis Results of Independent Risk Factors Predicting Post-Stroke Secondary Infection

Age	**Cut-off**	**AUC**	**Sensitivity (%)**	**Specificity (%)**	**OR (95% CI)***	**p-value**
	> 69 years	**0.722**	**94.7%**	**42.5%**	**8.65 (1.78–41.82)**	**0.007**
Stroke Severity (NIHSS)	> 8	0.747	63.2%	74.2%	6.49 (2.17–19.41)	**0.001**
Combined Model**	Age > 69 and NIHSS > 8	0.770	-	-	-	**< 0.001**

OR: Odds Ratio; CI: Confidence Interval; AUC: Area Under the Curve; NIHSS: National Institutes of Health Stroke Scale. *Independent risk factors were calculated using a multivariate logistic regression model. OR values are presented based on the cut-off references of > 69 for age and > 8 for NIHSS. *The combined model reflects the overall predictive power (AUC) when both parameters are used together.*

Discussion

The primary finding of this study is that healthcare-associated infections developing after acute ischemic stroke significantly increase in patients with an admission NIHSS score above 8 and those older than 69, and in-hospital mortality is heavily concentrated in this high-risk group. While advanced age and a high stroke severity index were observed as strong and independent predictors for upcoming infections, no statistical difference was found between infection risk and the presence of comorbidities that increase with advanced age, stroke subtypes that significantly affect clinical outcomes and prognosis, or the localizations of ischemic lesions.

The cumulative secondary infection (13.6%), specific pneumonia (7.2%), and urinary tract infection (6.5%) rates detected in our study strongly align with the current trend of decreasing prevalence in the literature [3]. As highlighted in current AHA/ASA clinical guidelines [13], routine dysphagia screenings, organized stroke unit care, and multidisciplinary rehabilitation approaches have significantly reduced this hospital-acquired infection burden in recent years. Due to the retrospective nature of our study, specific swallowing function scores and dysphagia screening results of our patients could not be included in the dataset. Additionally, our relatively small patient cohort constitutes our major limitations, restricting the generalizability of our findings. However, despite these advancements in care standards, independent of purely mechanical factors, our analyses demonstrate that advanced age (> 69) remains an independent and non-modifiable risk factor for infection development. The fact that comorbidities were found to be statistically insignificant in our cohort ($p > 0.05$) supports the idea that increased infection susceptibility in elderly patients is driven by pure biological mechanisms, such as age-related cellular immunity loss

caused by SIDS and immunosenescence—which are well-described in the literature—rather than by chronic disease burden [8, 9, 14].

In our study, the identification of admission stroke severity (NIHSS > 8) as another independent predictor for infection development is an expected finding [15]; high neurological deficit scores directly bring about mechanical processes that predispose to infection, such as immobilization and increased aspiration risk [7]. In contrast, it is noteworthy that ischemic lesion localizations and stroke subtypes did not have a statistically significant effect on infection risk in our data ($p > 0.05$). This suggests that susceptibility to infections may be more related to the general ischemic burden of the brain rather than damage to a specific anatomical region. Although radiological infarct volume was not quantitatively evaluated in our study, it can be said that the NIHSS cut-off value of > 8 functions as a clinical threshold reflecting the SIDS caused by increased ischemic stress. Indeed, the median secondary infection development day of 4 in our cohort also supports this neuro-immunological theory; it is consistent with the literature that this acute cellular immunosuppression triggered by stroke rapidly deepens in the first 24–72 hours, preparing the ground for clinical infections in the immediately following days [7, 9]. The time-dependent nature of immunosuppression also explains why routine inflammatory biomarkers at the time of admission were found to be insignificant in predicting infection development in our study ($p > 0.05$). Similarly, the literature has shown that laboratory values measured on the first day of stroke are insufficient to predict infection, and immunosuppressive responses only reflect on laboratory parameters in the following days [16]. Due to the retrospective design of our study, the lack of standardization in blood sampling timing during patients' follow-ups necessitated that analyses be conducted exclusively on initial admission data. Consequently, follow-up laboratory parameters that might predict infection risk could not be evaluated. The inadequate early-stage predictive power of these routine inflammatory biomarkers creates a need for practical clinical predictive tools that can determine infection risk. The performance of the multivariate logistic regression model created to predict the development of secondary infection in our study is quite remarkable. The high AUC value obtained by integrating the parameters of advanced age (> 69) and admission NIHSS score (> 8) indicates that this could be a practical tool for determining the patient's infection risk in the early period.

Our study has shown that secondary infections developing in AIS patients not only prolong hospital stays but also significantly increase in-hospital mortality in these patients. Since major infection predictors like advanced age and a high NIHSS score are non-modifiable patient factors, modifiable preventive strategies must be identified to prevent this scenario. Current guidelines [13] and major phase clinical trials [17] show that routine systemic antibiotic prophylaxis applied to AIS patients does not significantly prevent the development of secondary infections or improve survival; therefore, this approach is currently not recommended. For these reasons, in addition to standard infection prevention methods applied to all patients, we believe that adapting nasal/oropharyngeal carriage screenings and specific local decolonization strategies [18]—which are known to significantly reduce pneumonia in mechanically ventilated patients in intensive care units—to this risky group in stroke units could be a beneficial future research area. Stroke patients are highly susceptible to microaspiration due to SIDS and swallowing dysfunction. Therefore, even if they are not intubated, performing oropharyngeal and

nasopharyngeal colonization screening at admission and eradicating pathogens through preemptive local decontamination before they reach the lungs has the potential to create an effective barrier against secondary infections. Moreover, this strategy is completely in line with the principles of antimicrobial resistance management. However, the clinical benefit and cost-effectiveness of this theoretical approach must be confirmed by larger-scale prospective studies in the future.

Conclusion

In conclusion, this study supports existing literature indicating that advanced age and high admission stroke severity in AIS patients are independent and strong clinical predictors for the development of healthcare-associated secondary infections. The inadequacy of routine inflammatory laboratory markers at emergency admission to reflect the early phase of stroke-induced immunosuppression further increases the importance of practical parameters like age and NIHSS score that can be evaluated directly at the bedside. Because secondary infections developing in this high-risk group dramatically increase in-hospital mortality, the follow-up of elderly stroke patients with severe neurological deficits should go beyond standard approaches, and larger-scale studies are needed to develop and integrate targeted, proactive infection prevention strategies into clinical practice.

Abbreviations

AIS
Acute Ischemic Stroke
AUC
Area Under the Curve
CRP
C-Reactive Protein
ICU
Intensive Care Unit
NIHSS
National Institutes of Health Stroke Scale
NLR
Neutrophil-to-Lymphocyte Ratio ROC:Receiver Operating Characteristic SAI:Stroke-Associated Infection
SAP
Stroke-Associated Pneumonia
SIDS
Stroke-Induced Systemic Immune Dysfunction
TOAST
Trial of Org 10172 in Acute Stroke Treatment
UTI
Urinary Tract Infection

Declarations

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki. Ethics committee approval for this study was obtained from the Ordu University Non-Interventional Scientific Research Ethics Committee on 30.07.2026 (Decision No: 2026/117). Due to the retrospective nature of the study, the requirement for informed consent was waived by the ethics committee.

Consent for publication

Not applicable.

Competing Interests

The authors declare that they have no competing interests.

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Authors' contributions

L.Ş. conceptualized the study, performed data collection and statistical analysis, and drafted the initial manuscript. R.E.Y. contributed to data interpretation and critically revised the manuscript for important intellectual content. All authors read and approved the final manuscript.

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During the preparation of this work, the author(s) used Gemini to translate the initial draft from Turkish to English. After using this tool/service, the author(s) reviewed and edited the content as needed and take full responsibility for the content of the publication.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Figures

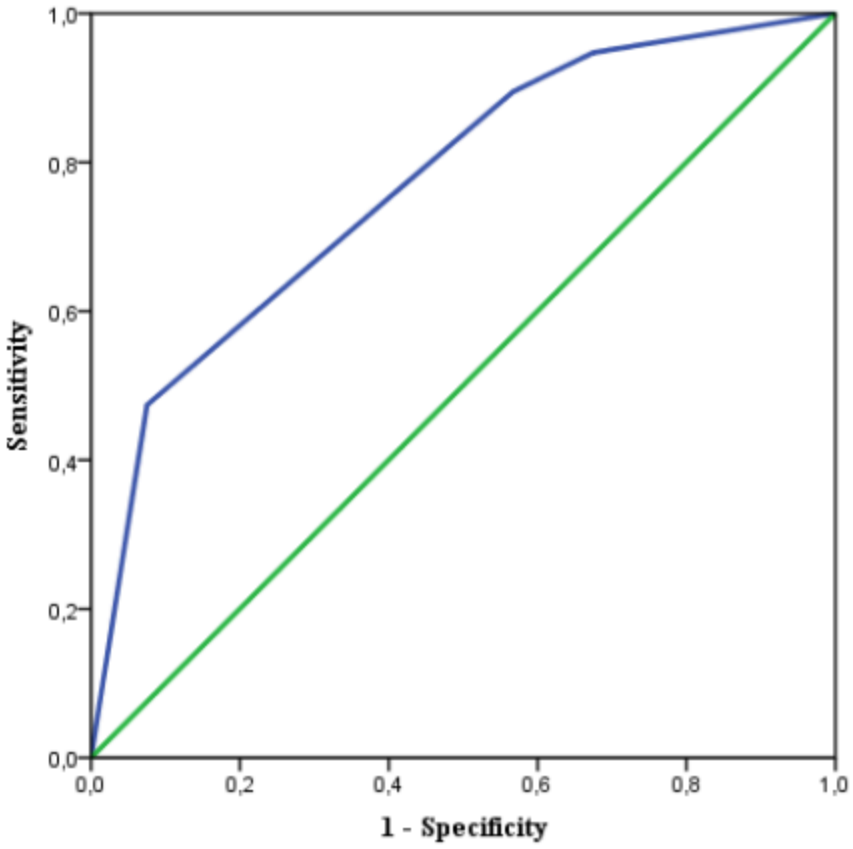


Figure 1

ROC curve for predicting post-stroke infection based on the combined model (Age >69 and NIHSS >8).

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Supp.Table1.docx
- Supp.Table2.docx
- Supp.Table3.docx
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