



Multi-Territorial Ischemic Stroke with Hemorrhagic Transformation and Secondary *Staphylococcus hominis* Septicemia: A Comprehensive Neuro-Critical Case Analysis

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Article History :

Received date : 2026/01/17

Revised date : 2026/02/02

Accepted date : 2026/03/11

Published date : 2026/04/24



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E-ISSN :

ISSN 3048-1368



P-ISSN

ISSN 3048-1376



ABSTRACT

BACKGROUND

Acute ischemic stroke involving multiple vascular territories (multi-territorial) presents a significant clinical challenge with high morbidity and mortality rates.¹ This condition is often exacerbated by severe acute hypertensive responses, which increase the risk of hemorrhagic transformation (HT).³ Beyond the primary neurological injury, intensive care unit (ICU) patients are highly susceptible to nosocomial infections that can trigger systemic sepsis.⁵ *Staphylococcus hominis*, a member of the Coagulase-Negative Staphylococci (CoNS) group, is increasingly recognized as a relevant opportunistic pathogen in critically ill and immunocompromised patients.⁵

CASE REPORT

A 54-year-old female (Mrs. PS) presented with sudden loss of

consciousness (GCS 3-4) and a hypertensive crisis (240/150 mmHg). Head CT scan revealed extensive multi-territorial cerebral infarction involving the left parietotemporal cortical-subcortical area, right cerebellum, and bilateral basal ganglia, accompanied by signs of hemorrhagic transformation and ventriculomegaly. The patient was admitted to the ICU with mechanical ventilator support and aggressive blood pressure control. During treatment, the patient's clinical condition deteriorated, and blood cultures identified the growth of methicillin-resistant *Staphylococcus hominis ssp hominis* (MRCoNS). Despite intensive resuscitation and stabilization efforts, the patient passed away on the fifth day of care due to cardiac and respiratory arrest.

DISCUSSION

Analysis of this case highlights the interaction between autonomic dysregulation following a severe stroke (NIHSS score 29) and systemic failure.⁹ The high NIHSS score and low GCS at admission are powerful predictors of in-hospital mortality.¹⁰ The emergency hypertension likely contributed to the hemorrhagic transformation through blood-brain barrier disruption.¹³ The discovery of *S. hominis* in blood cultures complicated clinical management; although often considered a contaminant, the consistent growth pattern (4 out of 4 bottles) and resistance to oxacillin and linezolid indicated a true pathogenic role in worsening the prognosis through nosocomial sepsis.¹⁴ Blood pressure management was tailored to balanced guidelines, targeting a systolic blood pressure (SBP) of 160-180 mmHg to stabilize the HT while maintaining cerebral perfusion.¹⁶

CONCLUSION

This case reinforces that multi-territorial stroke with an extremely high NIHSS score carries a very poor prognosis.¹⁸ Management requires an aggressive multidisciplinary approach to control hemodynamic parameters, cerebral edema, and to prevent or treat nosocomial infections.²⁰ Early recognition of multi-drug resistant pathogens like *Staphylococcus hominis* is crucial in determining therapeutic success in the ICU environment.²²

KEYWORDS

Multi-Territorial Ischemic Stroke; Hemorrhagic Transformation; Hypertensive Emergency; *Staphylococcus hominis*; ICU Sepsis; NIHSS 29.

INTRODUCTION

The global burden of cerebrovascular disease remains one of the greatest public health challenges in the 21st century, serving as the second leading cause of mortality and the third leading cause of disability worldwide.²³ Ischemic stroke accounts for approximately 75% to 85% of all stroke cases.²⁴ Among these, cases involving damage across multiple vascular territories are particularly destructive, often reflecting central embolic sources or profound systemic hemodynamic failure.¹

Acute hypertensive responses are found in over 80% of patients during the initial phase of ischemic stroke.¹³ While elevated blood pressure can be a physiological response to maintain cerebral perfusion pressure in the penumbra, extreme elevations (e.g., SBP >220 mmHg) are associated with malignant brain edema and hemorrhagic transformation (HT).³ HT is a dreaded complication that occurs when damaged vessels in the infarcted area leak or rupture, dramatically worsening the patient's functional prognosis.²⁷

Stroke severity is clinically quantified using the National Institutes of Health Stroke Scale (NIHSS). A score above 20 defines a severe stroke, and scores above 25 are typically associated with a very high probability of death or permanent disability.¹⁸ Patients with extreme scores, such as the NIHSS of 29 seen in this case, represent the highest risk group requiring intensive neuro-critical care.¹⁹

During ICU care, severe stroke patients face not only primary neurological injury but also systemic complications such as bloodstream infections (BSI).⁶ *Staphylococcus hominis*, a member of the Coagulase-Negative Staphylococci (CoNS), was traditionally viewed as a harmless skin commensal.⁷ However, recent literature identifies it as a lethal opportunistic pathogen in critically ill patients, particularly when showing multi-drug resistance.⁵

This report provides a detailed analysis of a patient with severe multi-territorial infarction,

exploring the interplay between hemodynamic management in hypertensive emergencies, the pathophysiology of HT, and the microbiological challenges of nosocomial sepsis.

CASE REPORT

Patient Identity and History

Mrs. PS, a 54-year-old female (DOB 07/02/1971), was admitted to the Emergency Department (ED) on December 16, 2025, with a chief complaint of sudden loss of consciousness. According to her family, she was resting at home around 16:00 when she suddenly lost consciousness. Prior to this, she reportedly complained of a severe headache, had slurred speech (*pele*), and vomited twice. She had a long-standing history of hypertension but was not taking her medications at the time of admission.

Initial Physical and Neurological Examination

At the initial assessment in the ED (21:38), the patient appeared critically ill and comatose.

Vital Parameter	Result	Status/Notes
Blood Pressure	240/150 mmHg	Hypertensive Emergency
Pulse	120 bpm	Tachycardia
Respiratory Rate	22 bpm	Tachypnea
Temperature	36.6 °C	Afebrile
SpO2 (Room Air)	92%	Mild Hypoxemia
SpO2 (4 lpm NK)	99%	Corrected

Neurological evaluation showed anisocoric pupils and right-sided lateralization. Pathological Babinski reflexes were positive bilaterally (+/+), while meningeal signs were negative. The initial NIHSS score was 29, and the GCS was 4 (E2V1M1).

Laboratory Findings

Comprehensive laboratory tests were performed to evaluate metabolic status, organ function, and coagulation.

Table 1: Routine Hematology (16/12/2025)

Parameter	Result	Unit	Reference Value
Hemoglobin	14.3	g/dL	11.7 - 15.5
Leukocytes	10.3	$10^3/\mu l$	3.6 - 11.0
Platelets	296	$10^3/\mu l$	150 - 440
Hematocrit	42	%	35 - 47

Table 2: Blood Chemistry and Electrolytes

Examination	16/12	17/12	18/12	19/12	20/12	Unit	Ref Value
Random Glucose	144	176	150	139	146	mg/dL	70 - 140
Blood Urea	39	-	-	-	68	mg/dL	10 - 50
Creatinine	1.31	-	-	-	1.28	mg/dL	0.45 - 0.75

Sodium (Na)	142	-	-	-	-	mmol/L	135 - 147
Potassium (K)	3.5	-	-	-	-	mmol/L	3.5 - 5.0
Chloride (Cl)	108	-	-	-	-	mmol/L	95 - 105
SGOT (AST)	14	-	-	-	-	U/L	< 35
SGPT (ALT)	12	-	-	-	-	U/L	< 35

The findings indicated early-stage acute kidney injury (AKI) with a creatinine of 1.31 mg/dL at admission and persistent hyperglycemia throughout the stay.

Table 3: Coagulation Profile (16/12/2025)

Examination	Result	Unit	Reference Value
Prothrombin Time (PT)	13.6	sec	11.0 - 18.0
APTT	25.1	sec	27.0 - 42.0 (Low)
INR	1.1	-	0.8 - 1.1

Table 4: Blood Gas Analysis (BGA)

Parameter	16/12 (23:38)	17/12 (05:31)	Reference Value
pH	7.193	7.396	7.350 - 7.450
PCO2 (mmHg)	88.0	37.3	35.0 - 45.0

PO2 (mmHg)	139.2	206.4	80.0 - 100.0
CHCO3st (mmol/L)	34.2	23.1	21.0 - 28.0
tHb (g/dL)	14.9	15.4	11.5 - 17.4
Hct (%)	45.0	46.0	35.0 - 50.0
BE (mmol/L)	3.3	-0.8	-

Initial BGA showed severe respiratory acidosis likely due to central hypoventilation. Following intubation, the patient's acid-base status improved by December 17.

Imaging Findings

Non-Contrast Head CT (16/12/2025): Showed multiple ill-defined hypodense lesions in the left parietotemporal cortical-subcortical area, right cerebellum, and bilateral basal ganglia. Findings included ventriculomegaly and atherosclerosis of the posterior and middle cerebral arteries.

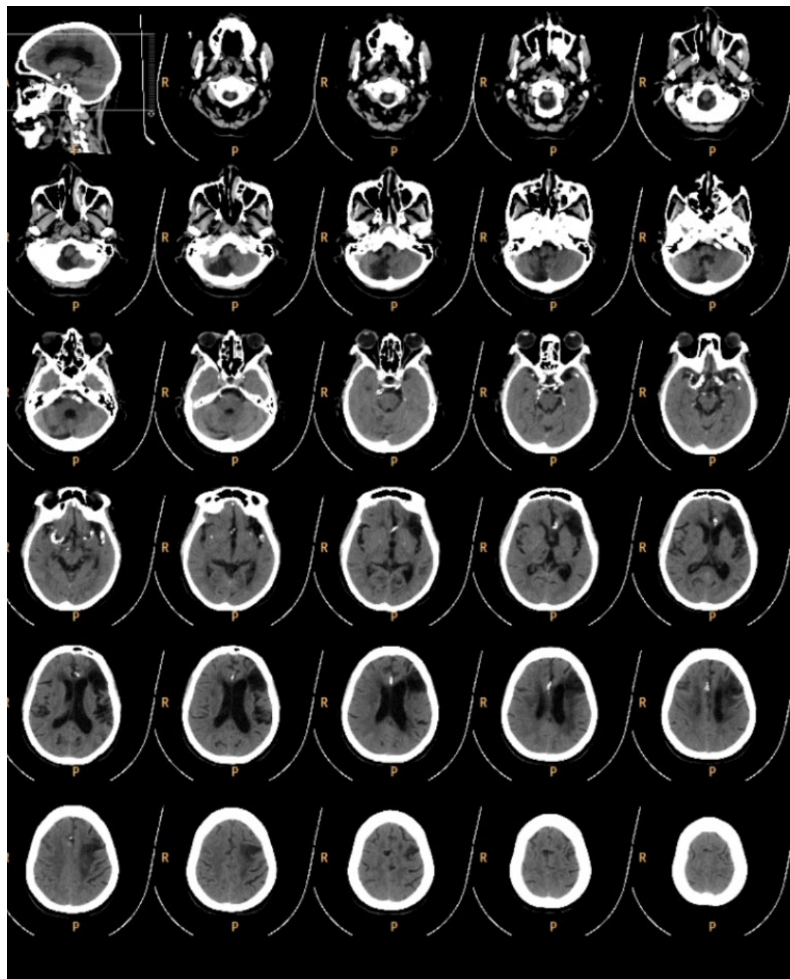


Figure 1. Non-Contrast Head CT (16/12/2025)

Chest X-Ray (16/12/2025): Revealed cardiomegaly, aortic atherosclerosis (calcification), and increased bronchovascular markings suggestive of bronchitis.

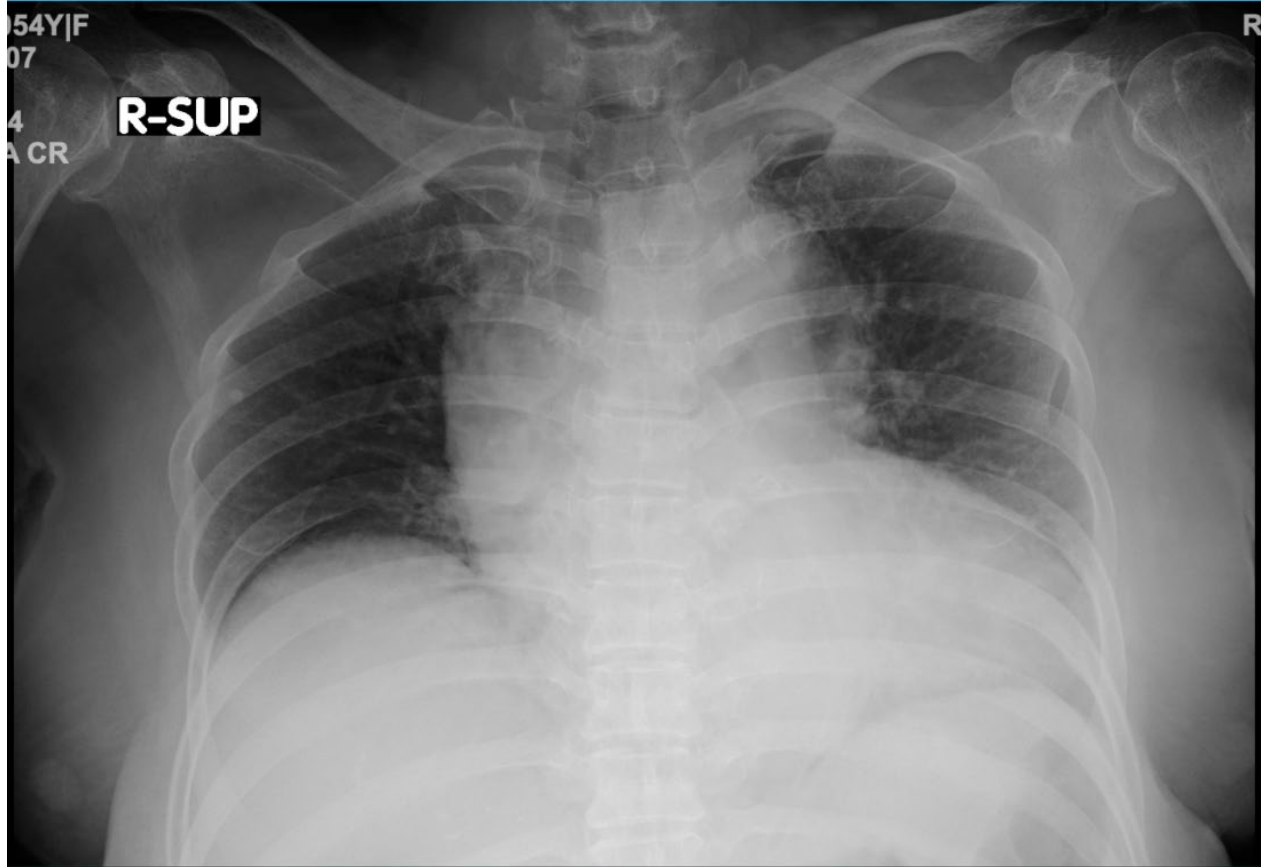


Figure 2. Chest X-Ray (16/12/2025):

Microbiology

Blood culture taken on December 17, 2025, identified:

- **Organism:** *Staphylococcus hominis ssp hominis*.
- **Resistance:** Benzylpenicillin, Oxacillin (Cefoxitin screen positive), Linezolid, Nitrofurantoin.
- **Sensitivity:** Gentamicin, Ciprofloxacin, Levofloxacin, Erythromycin, Clindamycin, Tetracycline, Tigecycline.

Clinical Course in the ICU

The patient was managed with a diagnosis of Loss of Consciousness (DOC) due to CVD Infarct with Hemorrhagic Transformation and Hypertensive Emergency.⁸

- **Dec 16:** Initial management included O₂, IVFD NaCl 0.9%, and a Nicardipine drip (5-15 mg/hr) targeting SBP 160-180 mmHg. Medications included Citicoline, Mecobalamine, and Mannitol.
- **Dec 17:** Patient remained under sedation on full ventilator support. BP was 170/96 mmHg. Aspilet 80mg was added via NGT.
- **Dec 18:** Continued sedation/ventilator. BP 156/94 mmHg. Ceftriaxone 1x2g IV was initiated (Day 2).
- **Dec 19:** Sudden BP spike to 186/113 mmHg. Nicardipine dose increased; oral Amlodipine 10mg and Candesartan 16mg added.
- **Dec 20 (Death):** At 06:28, respiratory and cardiac arrest were noted. Pupils were fixed and dilated. Following family agreement on *Do Not Resuscitate* (DNR) status, the patient was pronounced dead at 06:50.

DISCUSSION

Pathophysiology of Multi-Territorial Stroke and HT

The case of Mrs. PS demonstrates a catastrophic cerebrovascular event involving the left parietotemporal (MCA territory), right cerebellum (PCA/cerebellar territory), and bilateral basal ganglia.⁸ Multi-territorial infarcts typically indicate a central embolic source (e.g., atrial fibrillation) or severe hemodynamic failure.¹ The extreme hypertensive response (240/150 mmHg) likely triggered hemorrhagic transformation (HT) by increasing hydrostatic pressure against a damaged blood-brain barrier.³

Prognostic Significance of NIHSS and GCS

An NIHSS score of 29 categorizes the stroke as "very severe." Studies show that scores above 25 correlate with an extremely high probability of mortality or severe disability.¹⁸ Furthermore, the initial low GCS (4) and severe hypercapnia (PCO₂ 88.0 mmHg) indicated early brainstem compression or failure of central respiratory drive, both of which are markers for poor outcomes.¹¹

Blood Pressure Management Guidelines

AHA/ASA 2022/2024 guidelines emphasize balancing BP to prevent hematoma expansion while maintaining perfusion.³⁴ While targets for spontaneous ICH are often lower (SBP 140 mmHg), in ischemic stroke with HT, targets of 160-180 mmHg are frequently used to protect the ischemic penumbra.¹⁶ The use of Nicardipine in this case was appropriate for its rapid titration and ability to limit BP variability.³⁵

Staphylococcus hominis as an ICU Pathogen

The identification of *Staphylococcus hominis* (MRCoNS) from 4 out of 4 blood culture bottles confirms it as a true pathogen rather than a contaminant.¹⁴ While CoNS are less virulent than *S. aureus*, they form robust biofilms on ICU devices.⁵ The resistance to Oxacillin and Linezolid suggests a multi-drug resistant (MDR) hospital-acquired strain.⁵ The initial use of Ceftriaxone was likely ineffective against this MRCoNS, potentially allowing sepsis to worsen the patient's terminal state.²²

Ethical Dilemmas and Outcomes

The decision for a DNR status was made as the patient faced near-certain mortality given the extent of the multi-territorial damage and secondary systemic failure.⁸ This aligns with neuro-critical care principles where limiting life-sustaining treatment is considered when there is no hope for functional recovery.³⁴

CONCLUSION

This case documents a catastrophic multi-territorial ischemic stroke exacerbated by a hypertensive emergency and secondary MRCoNS sepsis. The NIHSS score of 29 was an accurate predictor of the poor outcome. Management of such cases requires delicate hemodynamic balancing and vigilant microbiological monitoring. The presence of multi-drug resistant *S. hominis* highlights the ongoing threat of nosocomial infections in neuro-critical care units.

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