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Case Report: Administration of empirical antimicrobial therapy on an acute stroke patient with Suspected Hospital-Acquired Infection

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Abstract

Background: Principles of stroke management has established several aspects that can improve or worsen patient's clinical neurological status and overall outcome. One of the factors that can significantly affect patient outcome is hospital-acquired infection (HAI). **Objective:** This case report aims to illustrate how a stroke patient seems to develop HAI, which hinders the progress of clinical improvement. **Methods:** A 53-year-old male patient presents with sudden onset of right-sided weakness and inability to speak five days ago. Patient is also somnolent, with dysphagia and global aphasia. Patient undergoes the workup for acute onset stroke, and the clinical, radiology and laboratory data is observed. Patient is diagnosed with left middle cerebral artery territory ischemia, with the risk faktor of hypercholesterolemia and stage 1 hypertension. Patient gradually gained full consciousness, can understand verbal commands, and can swallow soft foods. On the fourth day of inpatient stay, patient develops a fever. Additional tests are then ordered to investigate the suspected HAI, including inflammation and infection markers. **Results:** Patient showed elevated leukocyte levels specifically neutrophilia, elevated CRP, clear chest radiography. Hospital stay is lengthened for three more days on account of intravenous administration of empirical antibiotics, continued with oral antibiotics as blood culture results is awaited. No microbial growth was found on blood culture. **Conclusion:** This case highlights the challenges of administering empirical antimicrobial therapy on patients that show signs of inflammation caused by bacterial infection, which interferes with the main condition's clinical progression. Several points may be taken into consideration to design a protocol that can manage such cases more efficiently and appropriately.

Keywords: Acute stroke care; hospital-acquired infection; c-reactive protein; antimicrobial resistance.

Cite This Article

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INTRODUCTION

Stroke is a leading cause of morbidity and mortality worldwide, often resulting in prolonged hospitalization, functional impairment, and high healthcare costs. Patients who suffer acute ischemic or hemorrhagic stroke are particularly vulnerable to complications during their hospital stay, including the risk of acquiring infections that were not present or incubating on admission. Hospital-acquired infections (HAIs) in this population, such as pneumonia, urinary tract infections, and bloodstream infections, have been shown to significantly worsen outcomes. For example, in a retrospective cohort of ischemic stroke patients, about 20% developed a post-stroke infection, with higher stroke severity and comorbid conditions (notably diabetes mellitus) identified as independent predictors. These patients faced substantially worse functional outcomes at 90 days and markedly higher healthcare costs compared to those without infection.[1]

The concern over HAIs among stroke patients is not only clinical but also economic and public health in nature. Infected patients tend to have prolonged length of stay (pLOS) in hospital, which inflates cost and increases the risk of further complications. Studies demonstrate that the occurrence of infections among patients with ischemic stroke is strongly associated with poor short-term functional outcome, and much of the adverse effect of prolonged hospitalization appears mediated by infection. Moreover, infection risk scores have been developed using admission factors (age, diabetes status, stroke severity) to help predict which patients are at higher risk and might benefit from preventive interventions. Given the largely preventable nature of many HAIs, the burden of disability, cost, and mortality in stroke populations underscores the urgency of targeted infection control strategies.[2,3]

Stroke remains one of the major causes of disability and death globally, and in Indonesia and across Asia, its burden is growing due to increasing prevalence of risk factors such as hypertension, diabetes, and aging populations. Stroke patients are especially vulnerable to hospital-acquired infections (HAIs), given their impaired neurological function, possible swallowing difficulties, reduced consciousness, and frequent need for invasive procedures. These infections—pneumonia, urinary tract infections, bloodstream infections—can significantly worsen morbidity and mortality, extend hospital length of stay, increase healthcare costs, and reduce the chance of functional recovery.

Local data show for example that pneumonia occurs in nearly 28.3% of acute stroke patients in Bandung, associated with risk factors such as dysphagia, use of nasogastric tube, large infarct size, and lowered consciousness. In Indonesia especially, recent studies have begun to quantify the prevalence, risk factors, and specific pathogen profiles of HAIs after stroke. A retrospective study in Jakarta found that about 17.9% of hospitalized acute ischemic stroke patients developed bacterial infections during their hospital stay. This case report aims to present a case of an acute stroke patient appears to develop an infection during his hospita stay, which added to the length of hospital stay, medications administered, tests ordered for investigation and evaluation of the infection, and overall burden of cost.[1,3,4]

METHODS

Study design and setting

The design is direct observation of a case from patient's hospital admission to discharge, and presented as a case report.

Inclusion criteria and data collection

This study included patients in the acute phase of stroke, either hemorrhagic or non-hemorrhagic, with or without comorbidities. Eligible participants were adults aged 18 years or older who showed no clear signs of community-acquired infection at the time of hospital admission or initiation of inpatient care.

Patients were considered to have developed a hospital-acquired infection (HAI) if clinical and/or laboratory evidence of infection appeared at least three days of hospital stay (3×24 hours after hospital admission). Indicators of HAI included:

- Fever (body temperature >37.4°C);
- Clinical evidence of infection, such as phlebitis at the site of peripheral venous access, rhonchi on pulmonary examination, or other findings as determined by the attending physician;
- Objective evidence from laboratory or imaging studies, such as leukocytosis on repeat complete blood count, chest radiograph demonstrating new bronchopneumonia compared to the initial admission film, or leukocytosis on complete urinalysis

Tests that may be performed toward the patient include, but not limited to, the following:

- Quantitative serum C-reactive protein (CRP)
- Microbial culture and antibiotic susceptibility testing of urine/sputum/blood, as appropriate

*Ethical concideration*s

The study was conducted in accordance with the Declaration of Helsinki, and the protocol was approved by the Ethics Committee as stated in the Statement Letter No. 3060/UN21.8/PT.01.04/2025 released by the Faculty of Medicine and Health Sciences Universitas Jambi.

RESULTS

Case

A 49-year-old male came to the emergency department sudden right-sided weakness in the last two days. The patient also had difficulty of speaking, difficulty to eat/swallow, and the right side of his mouth appeared to be drooping. Previously, the patient had complained of a headache the night before. Nausea, vomiting, fever, or seizure were denied with no history of trauma or long-term medication use.

On physical examination the beginning when the patient first came obtained results in the form of vital signs, blood pressure of 145/79 mmHg, pulse 61 beats per minute and regular strong palpable, breathing frequency obtained 22 times per minute, symmetrical, blood oxygen 94% free air, and 36.5 degrees Celsius temperature. On generalist physical examinations showed mouth drooping and flat nasolabial fold on the right side. On neurological examinations showed GCS E3M5Vaphasia, whereas the motoric examination obtained flaccid hemiparesis on the right side. While the sensory and autonomic examination cannot be assessed. Also, the patient was unable to produce or understand language, repeat phrases, identify objects, read, or write on the following day.

From the results of the initial investigation, the laboratory tests showed an increase in the value of leukocytes to 13,900. Chest X-Ray (AP view) (Figure 1) showed signs of cardiomegaly with pulmonary edema. Non-contrast head CT-scan (Figure 2) was performed and presented as ischemia and infarction of the left

cerebral hemisphere, particularly the regions supplied by the left middle cerebral artery (MCA).

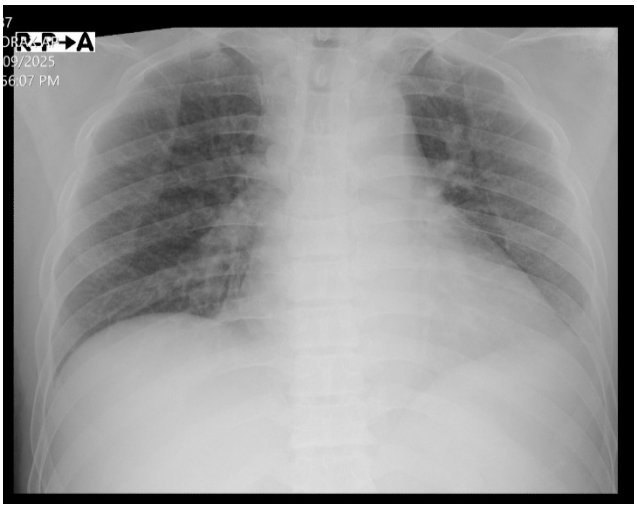

Figure 1. Chest X-Ray of MR. S

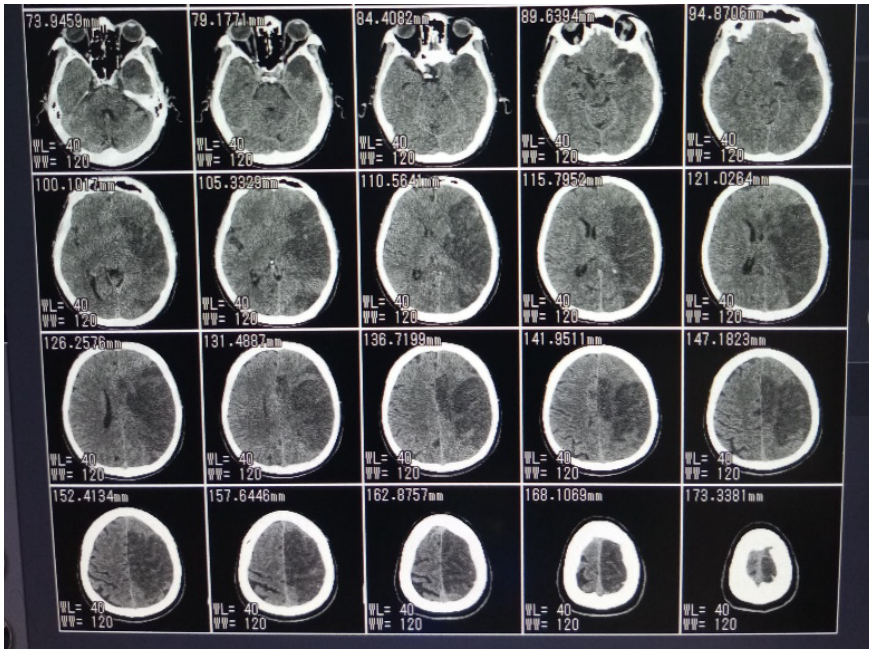

Figure 2. non-contrast head CT-scan of MR. S

The patient was managed with a series of interventions aimed at stabilizing his condition. He was positioned with his head elevated at 30 degrees and was placed on bed rest. Oxygen was administered via nasal cannula at 3 liters per minute, which resulted in an increase in blood oxygen from 94% to 97%. For the initial management, a loading dose of 200 cc of manitol was given, followed by 100 cc intravenously four times a day. Citicoline was administered intravenously at a dose of 1000 mg three times a day. In addition, patients also received 2 gr of ceftriaxone and 40 mg of omeprazole once a day intravenously. The patients then admitted to the intensive care unit (ICU) for further monitoring.

Approximately four hours after admission, the patient's pulse drops to 50 beats per minute with regular strong palpable. Other vital signs are in normal range with no symptoms. The ECG shows sinus bradycardia and the cardiologist suggested injecting 4 ampoules of atropine sulfate if the pulse was below 50 beats per minute. When starting intensive treatment on day three, the patient consciousness began to improve. The patient was fully alert and would follow orders but still could not speak, so concluded the GCS of patient obtained E4M6Vmotor aphasia. On physical examination, the patient's vital signs obtained blood pressure 138/94, pulse 57 beats per minute, respiration rate 16 beats per minute and temperature 36.1 degrees Celsius. On neurological examinations, motor strenght of right upper and lower limb from distal to proximal was still 1/1/1/1. The laboratory examination showed increased in total cholesterol to 268 mg/dl and LDL to 184.2 mg/dl (Table 3). The patient was prescribed atorvastatin 20 mg once daily and the mannitol dose is reduced to 50 cc intravenously six times a day, further tapered off for subsequent days. The patient was also transferred ot the regular ward.

On the 5th day of treatment, the patient can undestand speech but still unable to respond verbally. The vital sign is normal and still has weakness on the right side of the body. The patient developed a fever of 38,7°C. Another routine hematology test was ordered, and the leukocytosis rendered this patient eligible to be included as a sample for this study. After a blood cultured sample was obtained, empirical antibiotics namely ceftriaxone was given intravenously with the dose of 2x1g. After three days the fever subsided, the patient was consulted to the medical rehabilitation department for physical rehabilitation program, mannitol was tapered off and the patient was discharged with secondary stroke prevention therapy and oral cefixime to be taken at home. The prognosis of these patients is Quo ad vitam ad bonam, Quo ad functionam dubia ad malam, and Quo ad sanactionam dubia ad malam.

Table 1. Laboratory results of Mr. S

Parameter	Value			Normal range
	13/09/25	18/09/25	21/09/25	
Hemoglobin	13.6	13.8	14.0	14 – 18 g/dL
WBCs	**13.90**	**15.70**	**12.96**	5.0 – 10.0 x !0*3/uL
Neutrophil	73.2	73.9	63.6	37 – 75 %
Lymphosite	18.5	13.2	16.1	20 – 40 %
Monosite	7.3	9.6	**14.2**	2 – 10 %
Eosinophil	1.0	3.3	**5.2**	0 – 5 %
Basophil	0.0	0.0	0.9	0 – 2 %
Platelets	329	319	320	150-350 x 10*3/uL
Ureum	32			17 – 50 mg/dL
Creatinine	0.9			0.6 – 1.1 mg/dL
Sodium	139.72			135 – 145 mg/dL
Potassium	4.13			3.50 – 5.50 mg/dL
Choride	96.86			96 – 106 mg/dL
Calcium	1.15			1.1 – 1.35 mg/dL
Total cholesterol	268			<200 mg/dL
Triglyceride	124			60 – 150 mg/dL
HDL	59			>45 mg/dL
LDL	184.2			<150 mg/dL
CRP		108.23		<10 mg/L
Blood Culture		No Microbial growth detected		

DISCUSSION

Patients with large MCA strokes are at high risk of hospital-acquired infections especially stroke-associated pneumonia (SAP) and catheter-associated urinary tract infection (CA-UTI). The strongest, repeatedly-reported predictors are: advanced age, greater stroke severity (high NIHSS), dysphagia/aspiration, impaired consciousness, and exposure to invasive devices (NGT, urinary catheter, mechanical ventilation).[1,3,4,5] These factors interact with stroke-induced immune changes to raise HAI incidence and worsen outcomes.

More severe deficits such as immobility and impaired airway protection can increase these risks. Furthermore, the presence of impaired swallowing (neurogenic dysphagia) presents aspiration risk of food and drink, and provides additional risk of pneumonia due to aspiration of oropharyngeal flora, aside from the patient already being bedridden.[3,6,7,8] In the case of Mr. S, the patient was briefly dysphagic so a nasogastric tube was inserted but through the duration of hospital stay his ability to swallow returned, and semisolid foods were re-introduced gradually. Patient was fully conscious from the start but highly immobile as his entire right side was plegic.

During stay, the patient showed signs consistent with an active inflammatory process which is shown by significant fever; however, the nature of the process is not specifically bacterial, as shown by the white blood cell count that is elevated but not particularly showing neutrophilia (Table 1). After obtaining blood sample for culture and CRP, empirical broad-spectrum antibiotics was started. Ceftriaxone was used because cephalosporin is known to cover many gram-positive and gram-negative pathogens[9,10,11]. It has a favorable safety profile, the pharmacokinetics are fairly predictable and tissue penetration is good into most body tissues.[12]

C-reactive protein (CRP) is an acute-phase reactant synthesized by hepatocytes under the stimulation of interleukin-6 (IL-6), with additional contributions from interleukin-1β and tumor necrosis factor-alpha. It serves as a nonspecific but sensitive biomarker of systemic inflammation. Under normal conditions, serum CRP levels are typically below 5 mg/L; however, concentrations can increase rapidly within 6 to 8 hours following an inflammatory stimulus, reaching a peak at approximately 36 to 48 hours. The plasma half-life of CRP remains constant at around 19 hours, which means that circulating levels primarily reflect the rate of hepatic synthesis rather than changes in clearance. Consequently, elevated CRP values are indicative of ongoing inflammatory activity or tissue injury.[13,14,15]

Interpretation of an elevated CRP level must always consider the clinical context. Mild elevations (10–40 mg/L) are often associated with viral infections, postoperative inflammation, or minor trauma, whereas moderate elevations (40–100 mg/L) are suggestive of more significant inflammatory responses, including localized bacterial infection or autoimmune activity. Markedly elevated concentrations (>100 mg/L) are strongly correlated with severe bacterial infections, sepsis, or major tissue necrosis, while extremely high values exceeding 500 mg/L almost always indicate severe bacterial sepsis or necrotizing infections. In contrast, viral infections and chronic inflammatory conditions typically produce lower CRP responses.[16]

In the setting of acute ischemic stroke, CRP levels may show a modest increase within the first 24 hours as part of the sterile inflammatory response to cerebral tissue injury, often not exceeding 30 to 40 mg/L. However, a secondary or progressive rise in CRP beyond 48 to 72 hours after stroke onset may suggest the development of a hospital-acquired infection, such as stroke-associated pneumonia or catheter-associated urinary tract infection. Serial measurement of CRP is therefore clinically useful in differentiating post-stroke inflammatory reactions from infectious

complications. A declining trend following antimicrobial therapy generally indicates treatment response and infection control, whereas persistently elevated or rising levels may imply treatment failure, resistance, or other ongoing sources of inflammation.[17]

Although CRP is a valuable indicator of inflammatory activity, it lacks specificity for the underlying cause. Elevated levels may occur in infection, autoimmune diseases, malignancy, or following surgery or trauma. Furthermore, CRP cannot reliably distinguish bacterial from nonbacterial inflammation, and its rise or fall may lag behind clinical improvement or deterioration. Patients with significant hepatic dysfunction may also exhibit a blunted CRP response due to impaired synthesis. Nonetheless, when interpreted in conjunction with clinical findings, white blood cell count, procalcitonin levels, and microbiological results, CRP remains a practical and widely used biomarker for monitoring disease progression and therapeutic response in hospitalized patients, including those with acute stroke.[18]

In this patient, the elevated leukocyte and CRP already raised a clinical suspicion of inflammation that may be infectious in nature, but the blood culture yielded negative for any microbial growth. Blood culture remains the diagnostic gold standard for identifying bloodstream infections; however, it is associated with several important limitations that can reduce its clinical utility.[19,20]

The most significant limitation is its relatively low sensitivity, particularly in patients who have received prior antibiotic therapy, which may suppress bacterial growth and yield false-negative results. Sensitivity is further influenced by the volume of blood collected, the timing of sampling, and the number of culture sets obtained, as inadequate sample volume or single-set cultures may fail to detect intermittent or low-grade bacteremia.[21,22,23] Another major issue is the occurrence of contamination, typically due to skin flora such as Staphylococcus epidermidis or Corynebacterium species, which can lead to false-positive results and unnecessary antibiotic use. Reported contamination rates in clinical practice range between 2% and 6%, depending on aseptic technique and collection site. Additionally, the time required for pathogen growth and identification—usually 24 to 72 hours—can delay targeted antimicrobial therapy, forcing clinicians to rely on empirical broad-spectrum antibiotics during the interim.[24]

In the case of the above patient, there are several factors that may be put into consideration for the initiation of empirical antimicrobial therapy in the patient. As mentioned previously, stroke patients are already susceptible to infection and HAI due to a combination of factors. Pneumonia typically develops within the first week after stroke and is strongly associated with large hemispheric infarctions, especially those involving the middle cerebral artery territory, where dysphagia and reduced consciousness are more prevalent.[25,26,27] For a patient recovering within the acute phase and transitioning into the rehabilitative phase of stroke, it is essential that comorbid factors such as metabolic factors, immobility, and infection be managed and treated as early as possible to prevent superinfection or sepsis.[27,28,29] Ceftriaxone was still chosen as the drug of choice, and a blood culture profile that shows susceptibility to cephalosporins would provide essential information on the compatibility of this protocol as a means to inhibit HAI in a stroke patient. However, limitations of blood culture should be taken into consideration for a more appropriate management of inflammatory processes during stroke to be viewed as reactive or infectious in nature, as is the technique and criteria for an ideal blood sample collection for an ideal microbial growth.

CONCLUSIONS

This case highlights the challenges of administering empirical antimicrobial therapy on patients that show signs of inflammation caused by bacterial infection, which interferes with the main condition's clinical progression. Several points may be taken into consideration to design a protocol that can manage such cases more efficiently and appropriately.

CONFLICT OF INTEREST

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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DECLARATION OF ARTIFICIAL INTELLIGENCE USE

This study used artificial intelligence (AI) tools and methodologies in the following capacities: Manuscript writing support: AI-based language models for the purpose of, Language refinement, Content summarization, and Technical writing assistance to provide suggestions for structuring complex technical descriptions more effectively. We confirm that all AI-assisted processes were critically reviewed by the authors to ensure the integrity and reliability of the results. The final decisions and interpretations presented in this article were solely made by the authors.

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