Magna Neurologica Vol. 4, No. 2, July 2026

https://journal.uns.ac.id/magna-neurologica
DOI: 10.20961/magnaneurologica.v4i2.3062
e-ISSN 2985-3729 p-ISSN 2963-6027

MAGNA NEUROLOGICA

CASE REPORT **OPEN ACCESS**

ISCHEMIC STROKE IN A PATIENT WITH SUSPECTED CERVICAL CANCER: A THERAPEUTIC DILEMMA BETWEEN ANTICOAGULANT OR ANTIPLATELET STRATEGIES IN A RESOURCE-LIMITED SETTING: A RARE CASE REPORT

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Article History:
Received: December 23, 2025
Accepted: March 18, 2026
Published: July 15, 2026

Cite this as:
Arjuna YYE, Goenawan VB, Puspitasari V, Kurniawan YC. Ischemic Stroke in A Patient With Suspected Cervical Cancer: A Therapeutic Dilemma Between Anticoagulant or Antiplatelet Strategies in A Resource-Limited Setting: A Rare Case Report. Magna Neurologica 4(2) July 2026: 114-119. 10.20961/magnaneurologica.v4i2.3062

ABSTRACT

Background: Approximately 5% of strokes with no identifiable cause are linked to previously undiagnosed malignancies. The involvement of multiple vascular territories observed on brain imaging may indicate a hypercoagulable state related to cancer. Early identification is crucial for prompt cancer diagnosis.
Case: A 40-year-old woman with no established classic stroke risk factors presented with progressive left-sided weakness and difficulty with speech over three days. She had a history of persistent vaginal bleeding lasting four months. Non-contrast brain CT revealed multiple infarcts affecting the right frontoparietal region, left frontal lobe, and bilateral posterior as well as left anterior internal capsules. An abdominal ultrasound identified a 9.35 × 4.74 cm hypoechoic mass extending from the cervix to the uterus with bladder infiltration, findings highly suggestive of cervical carcinoma.
Discussion: Ischemic stroke associated with female reproductive malignancies is rare, with an incidence of around 0.3%. It is mainly caused by cancer-related hypercoagulability due to tumor-derived mucin, procoagulants, and inflammatory cytokines. Multifocal infarcts across various vascular territories may indicate a hypercoagulable state and suggest an underlying malignancy. There is no standardized anticoagulant protocol. Low-molecular-weight heparin (LMWH) is commonly used, though its effectiveness varies. Use of direct oral anticoagulants (DOACs) and antiplatelets is increasing, showing similar safety and efficacy.
Conclusion: To date, there is no definitive antithrombotic of choice for cancer-associated ischemic stroke. The selection of antithrombotic therapy should be carefully tailored based on individual risk factors and the patient's clinical condition.

Keywords: anticoagulant, antiplatelet, antithrombotic, cervical cancer, hypercoagulability, ischemic stroke

Introduction

Stroke is defined as an acute focal neurological deficit resulting from vascular injury to the central nervous system, including cerebral infarction, intracerebral hemorrhage, or subarachnoid hemorrhage.[1] Over the past three decades (1990-2021), the global incidence of stroke has risen markedly by nearly 70%.[2,3] Ischemic stroke remains the most prevalent subtype, accounting for approximately 87% of all stroke cases. It is associated with an estimated annual mortality of around 7 million and over 160 million individuals living with stroke-related disabilities worldwide.

The mainstay of ischemic stroke management involves reperfusion strategies such as thrombolytic therapy and mechanical thrombectomy, aimed at restoring cerebral perfusion rapidly and effectively. Once the acute phase is stabilized, determining the

underlying cause of the stroke is essential to guide secondary prevention and long-term management. While the majority of ischemic strokes are attributable to traditional metabolic risk factors, approximately 16% are categorized as embolic stroke of undetermined source (ESUS), characterized by the absence of a clearly identifiable etiology.[4,5] Notably, 5-10% of ESUS cases are later found to be associated with occult malignancies.[4,5] In younger adults (aged 14-59 years), malignancies most frequently linked to ischemic stroke include breast cancer (22.2%), gastrointestinal cancer (20%), and lung cancer (19.8%), while gynecologic cancers account for only about 0.3% of cases.[4,5]

Therefore, a comprehensive clinical evaluation during the acute phase of ischemic stroke is essential, particularly to explore potential cancer-associated etiologies. However, in resource-limited settings, the diagnosis of stroke remains challenging.[6] Limited access to diagnostic facilities often leads to delayed diagnosis, suboptimal management, and increased in-hospital mortality.[6] This remains a major issue in many developing countries, where healthcare access is still unevenly distributed. This report highlights the diagnostic challenges and therapeutic considerations in a patient with ischemic stroke suspected to be associated with an underlying malignancy, particularly the therapeutic dilemma between choosing anticoagulant and antiplatelet therapy in the context of limited diagnostic resources.

Case Report

A 40-year-old woman presented to the emergency department of a general hospital with a primary complaint of progressive left-sided body weakness that had worsened over the past three days. The weakness was accompanied by mild impairment of consciousness, as the patient appeared drowsy and had difficulty communicating, though she was still able to comprehend verbal commands. The weakness on the left side had initially appeared approximately two weeks earlier, for which she was hospitalized at another facility for eight days. However, after discharge, her symptoms deteriorated again within the following three days.

The patient also reported continuous and irregular vaginal bleeding over the past four months, requiring six to seven pad changes per day. Her history of chronic diseases, such as diabetes mellitus and hypertension, was uncertain. It was noted that she had intermittently taken metformin and amlodipine, belonging to her mother, without medical supervision. The patient's family provided limited information regarding her health condition in the preceding months.

Upon admission, physical examination revealed a cachectic patient with a blood pressure of 150/90 mmHg, heart rate of 94 beats per minute, respiratory rate of 18 breaths per minute, body temperature of 36.2 °C, and oxygen saturation of 99% on room air. During three days of observation, her blood pressure ranged from 90/50 to 110/70 mmHg without antihypertensive medication. Neurological examination revealed a Glasgow Coma Scale (GCS) score of 13 (E4V3M6), left central facial nerve paresis, and left hemiplegia. Motor strength was 3/5 in the right upper and lower extremities and 0/5 in the left upper and lower extremities.

Laboratory results demonstrated anemia (Hb 10.4 g/dL, HCT 31.6%) with normal red blood cell indices (MCV 80 fL, MCH 26.6 pg, MCHC 32.9 g/dL) and leukocytosis (18,300/ L), renal function tests showed elevated urea and creatinine levels (61 mg/dL and 1.74 mg/dL, respectively) with an estimated glomerular filtration rate (eGFR) of 38 mL/min/1.73 m. Random blood glucose levels during observation ranged from 100 to 190 mg/dL in the absence of antidiabetic medication. Electrocardiography revealed a regular sinus rhythm at 95 beats per minute with a normal cardiac axis, and chest X-ray findings were unremarkable (Figure 1).

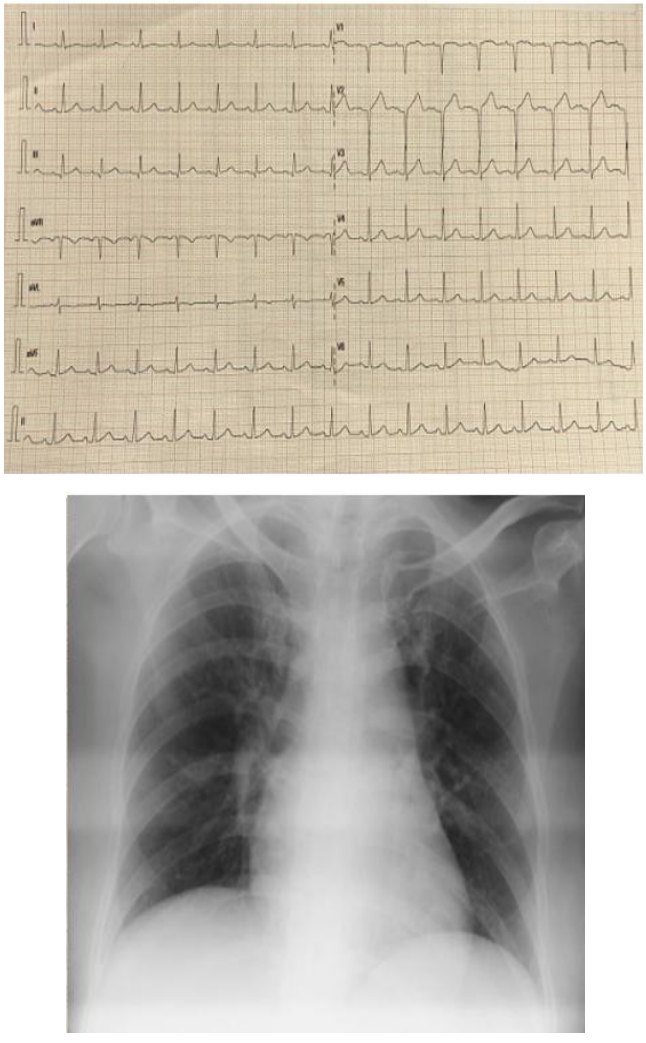

Figure 1. Electrocardiogram (ECG) and chest X-ray (PA view) showing no abnormalities

A non-contrast head scan demonstrated subacute to chronic infarctions involving the right frontoparietal lobe, left frontal lobe, and the bilateral posterior and left anterior portions of the internal capsule. Abdominal ultrasonography revealed a heterogeneous hypoechoic mass originating from the cervical region and extending into the uterus, measuring 9.35x4.74 cm, with poorly defined margins, suggestive of cervical malignancy with possible invasion into the urinary bladder. Bilateral grade II hydronephrosis and proximal ureteral dilatation were also noted (Figure 2,3).

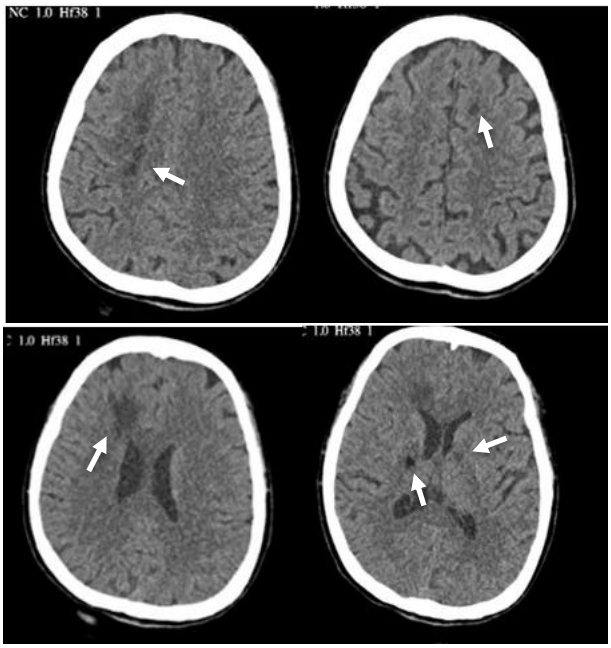

Figure 2. Non-contrast head CT scan showing multiple infarctions (white arrow)

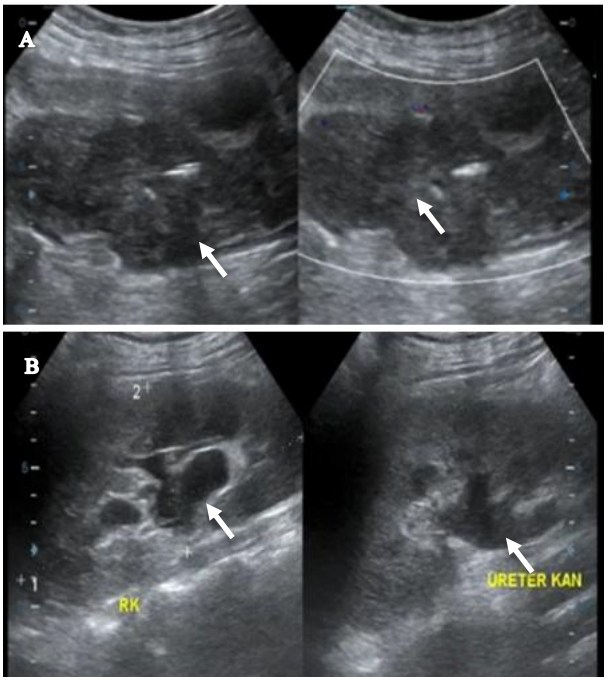

Figure 3. Abdominal ultrasound a) hypoechoic mass in the cervix extending into the uterus (white arrow); b) accompanied by hydronephrosis (white arrow)

The patient was treated with tranexamic acid 500 mg three times daily, sodium bicarbonate 500 mg three times daily, atorvastatin 20 mg once daily, paracetamol 1000 mg three times daily, ceftriaxone 2 g once daily, and omeprazole 40 mg twice daily. Antiplatelet therapy that had been prescribed at the previous hospital was discontinued due to the occurrence of bleeding. After 8 days of hospitalization, the patient was discharged with an overall improvement in general condition. The GCS at discharge was E4M5V (global aphasia). However, the patient did not return for scheduled follow-up visits.

The choice between antiplatelet and anticoagulant therapy remained challenging in this case of probable cancer-associated ischemic stroke. Clinical worsening had been observed when the patient previously received antiplatelet therapy at the referring hospital. At the same time, a hypercoagulable state related to an underlying malignancy was suspected. In this resource-limited setting, further evaluation of hypercoagulability markers and alternative etiologies including computed tomography angiography (CTA)/ magnetic resonance angiography (MRA), Holter monitoring, echocardiography, D-dimer, and fibrinogen testing could not be performed because of financial constraints, complicating therapeutic decision-making and posing an ongoing clinical challenge.

Discussion

Metabolic risk factors such as hypertension, hyperglycemia, and dyslipidemia remain the predominant contributors to stroke, accounting for approximately 68.8% of all cases.[2,7,8] However, about 5% of strokes previously classified as cryptogenic have been identified as being associated with occult malignancies.[5,9,10] Epidemiological studies indicate that cancer-associated strokes are most commonly linked to lung and gastrointestinal cancers, while malignancies of the female reproductive system contribute to only around 0.3% of total cases.[5,9,10] Notably, cervical cancer-associated ischemic stroke tends to occur in women younger than 51 years.[5,9,10]

The principal mechanism underlying cancer-associated ischemic stroke involves tumor-induced hypercoagulability.[9,11] Malignant cells enhance the expression of procoagulant molecules such as tissue factor, which activates coagulation factors VII and X, and secrete inflammatory cytokines and vascular endothelial growth factor (VEGF), both of which promote coagulation and angiogenesis. Tumor-related cytokines further recruit leukocytes, amplifying inflammatory and prothrombotic cascades.[9,11]

Additionally, certain tumors release mucin that activates platelets and endothelial cells via P- and L-selectin pathways and stimulates neutrophils to form neutrophil extracellular traps (NETs), thereby intensifying thrombosis and inflammation.[9,11] Although not all malignancies produce mucin, some, such as ovarian carcinoma, are known to induce the formation of platelet-rich microthrombi.[9,11] Tumor cells may also precipitate microvascular vasculopathy, resembling cerebral intravascular coagulopathy or thrombotic microangiopathy.[9,11]

Other mechansims of stroke in cancer patients include antineoplastic theraphy, which promotes procoagulant activity, endothelial dysfunction, and accelerated atherosclerosis.[9,12-17] Long-term immunosuppression and radiotherapy may further contribute to large-artery atherosclerosis, particulary in the cervical internal carotid arteries.[9,12-17] Malignancy related immunosuppression also increases infection risk, leading to systemic inflammation and vascular injury that may predispose to intracerebral hemorrhage.[9,12-17] In addition, cancer-related ESUS may result from occult nonbacterial thrombotic endocarditis (NBTE), characterized by platelet-fibrin vegetation on cardiac valves that are often difficult to detect.[9,12-17] Disseminated intravascular coagulation with in situ cerebral arterial thrombosis represents another potential mechanisms.[9,12-17]

The patient exhibited multiple cerebral infarctions across several vascular territories without classic vascular risk factors, indicating an embolic rather than atherosclerotic mechanism.[18-20] The presence of multifocal infarctions, particularly in both anterior and posterior circulations, supports the likelihood of cancer-associated stroke.[18-20] These findings are consistent with reports by Lee et al. (2014) and Chi et al. (2020), who observed that ischemic strokes in cancer patients often occur independently of conventional vascular risk factors and typically present as multifocal embolic infarctions caused by tumor-induced hypercoagulability.[18-20]

Abdominal ultrasonography revealed a cervical mass measuring 9.35x4.74 cm with evidence of bladder invasion, raising a strong suspicion of cervical carcinoma. Cervical cancer is generally asymptomatic during early stages and is frequently diagnosed incidentally.[20] In advanced stages, clinical features may include abnormal vaginal bleeding or discharge, pelvic discomfort, hematuria, dysuria, limb edema, and systemic symptoms such as weight loss and anorexia.[20]

The management of ischemic stroke secondary to malignancy remains challenging. Although secondary prevention is a key component of post-stroke care, there is no clear consensus regarding optimal therapy for cancer-associated ischemic stroke. The 2021 American Heart Association/American Stroke Association (AHA/ASA) guidelines suggest that direct oral anticoagulants (DOACs) are preferred over warfarin in cancer patients with atrial fibrillation (Class IIa), while anticoagulant therapy for ESUS provides no proven benefit (Class III).[21] Currently, low molecular weight heparin (LMWH) remains the most frequently recommended treatment for cancer-associated ischemic stroke, despite limited evidence of its efficacy in preventing recurrence.[21]

Evidence from the Hokusai-VTE trial (2013) demonstrated that DOACs were more effective than LMWH in preventing thromboembolic events among cancer patients, though with a higher bleeding risk.[20] The OASIS-Cancer study suggested that reducing hypercoagulability with anticoagulants, reflected by lower D-dimer levels, may improve one-year survival outcomes.[23] However, Nam et al. (2017) reported high short-term mortality rates and frequent recurrence (>50%) of new ischemic lesions, even among patients receiving anticoagulants.[24] Currently, there is no established consensus regarding the use of anticoagulant versus antiplatelet theraphy for the management of cancer-associated ESUS.[22]

Platelets also play a central role in the prothrombotic state associated with cancer, which has led to the consideration of antiplatelet therapy as an alternative or adjunct. The ARCADIA trial (2024) compared apixaban with aspirin in 137 patients with a history of malignancy and found no significant differences in ischemic or bleeding outcomes.[25] Similarly, Navi et al. (2019) found that the use of LMWH and aspirin showed no significant variation in the incidence of thromboembolic events or death.[9] Currently, there is no established consensus regarding the use of anticoagulant versus antiplatelet theraphy for the management of cancer-associated ESUS. Therefore, in the absence of definitive indications, secondary stroke prevention strategies should be tailored to the individual patient through multidisciplinary decision making involving both neurologists and oncologists.[26-28]

Several limitations should be acknowledged. First, ischemic stroke linked to malignancy remains relatively rare, and current evidence is primarily derived from observational data. Second, advanced diagnostic modalities such as MRI, hemostatic assays, and additional diagnostic or ancillary investigations could not be performed due to financial and administrative constraints. Third, incomplete clinical information from the patient and family limited the assessment of potential risk factors. Lastly, therapeutic decisions between antiplatelet and anticoagulant therapy were complicated by restricted access to diagnostic tools necessary to determine stroke etiology with certainty.

Conclusion

Anticoagulant therapy for cancer-associated ischemic stroke remains controversial because of concerns regarding bleeding risk. The efficacy of anticoagulant therapy in preventing recurrent stroke in this population is still not well defined. Moreover, there is a lack of consensus and limited data regarding the most appropriate treatment regimen for stroke patients with cancer-associated hypercoagulable conditions who are on anticoagulant therapy.

Acknowledgement

None

Conflict of Interest

None

Funding

No funding was received for the preparation or publication of this case report.

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