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When ST Elevation Is Not STEMI: Autonomic-Mediated Repolarization Abnormalities After Subarachnoid Hemorrhage

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Female, in their 90s
Final Diagnosis: Subarachnoid hemorrhage
Symptoms: Headache
Clinical Procedure: —
Specialty: Cardiology • Neurology

Objective: Challenging differential diagnosis

Background: Subarachnoid hemorrhage (SAH) is a neurological emergency accounting for 5% of all strokes, with mortality exceeding 50% in patients over 80 years of age. Aneurysmal SAH is particularly lethal in the elderly due to atypical presentations, including ECG abnormalities mimicking acute coronary syndromes, leading to delayed diagnosis and high complication rates. This case report highlights the diagnostic pitfalls of SAH-induced STEMI mimicry.

Case Report: A in their 90s White woman with a remote history of diabetes presented to the Emergency Department (ED) with sudden-onset occipital headache, nausea, and hypertensive crisis (214/68 mmHg). Initial electrocardiography (ECG) showed ST-segment elevations (STEMI) in leads I/aVL with reciprocal depressions in III/aVF, prompting STEMI activation despite normal troponin levels. Emergency computed tomography angiography (CTA) revealed a ruptured 3×1 mm anterior communicating artery (ACOM) aneurysm with Fisher Grade 3 SAH. Echocardiography at admission showed normal ejection and no wall motion abnormalities. ECG performed 1 day after admission showed complete resolution of the ST-T segment changes and the patient did not require any coronary intervention. Despite successful endovascular coiling, her hospital course was complicated by Cronobacter sakazakii bacteremia, embolic infarcts, and refractory cachexia. Palliative care was initiated on hospital day 14 due to irreversible functional decline, culminating in hospice transition.

Conclusions: SAH-mediated autonomic dysregulation can produce STEMI-like ECG changes even in the absence of coronary ischemia. Geriatric SAH management requires balancing intervention risks against frailty and comorbidities. Early recognition of SAH-induced ECG changes from autonomic-mediated repolarization abnormalities is essential to avoid misdiagnosis and guide appropriate intervention, particularly in older patients in whom comorbidities and frailty complicate recovery trajectories.

Keywords: Aneurysm • Case Reports • Electrocardiography • Subarachnoid Hemorrhage

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/952180

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Introduction

Headache disorders account for approximately 3.5 million ED visits annually in the United States, with thunderclap presentations requiring urgent evaluation for life-threatening etiologies such as SAH, cerebral venous thrombosis, and reversible cerebral vasoconstriction syndrome [1]. While SAH classically presents with a sudden-onset "worst headache of my life," older patients frequently exhibit attenuated or atypical symptoms, such as nausea, altered mental status, or autonomic instability, which can overshadow neurological problems [2].

Aneurysmal SAH triggers a cascade of neurohormonal disturbances, including a catecholamine surge mediated by hypothalamic injury. This sympathetic storm is hypothesized to induce transient coronary vasospasm, myocardial stunning, and ECG abnormalities mimicking STEMI in 10% to 15% of cases, even in the absence of coronary artery disease [3]. These neurocardiogenic phenomena create diagnostic dilemmas, particularly when troponin levels remain normal, as seen in this case. Nonagenarians pose additional challenges due to baseline frailty, atypical SAH evolution, and high complication rates from invasive interventions [4].

We present the case of a in their 90s White woman whose SAH presented concurrently with lateral ST-elevations on ECG, followed by a cascade of systemic complications. This case highlights the critical role of recognizing stroke-associated ECG abnormalities in older patients with SAH, ensuring appropriate management and avoiding potential harm from acute coronary syndrome treatments.

Case Report

The patient was a in their 90s woman with body mass index (BMI) of 19.8 and a history of diabetes mellitus and skin cancer who presented to the ED via emergency medical services after experiencing sudden-onset nausea, vomiting, and an intractable occipital headache. She described the headache as the "worst pain of my life," radiating to her cervical spine, and denied chest pain, shortness of breath, or trauma. Initial triage revealed severe hypertension (214/68 mmHg), normal heart rate (82 beats per minute), and normal oxygen saturation (97% on room air). Physical examination demonstrated cervical rigidity but no focal neurological deficits. The patient was alert and oriented to person, place, and time (Glasgow Coma Scale score 15), with intact cranial nerves (II-XII), normal motor strength, and no sensory abnormalities.

An ECG performed in the ED showed ST-segment elevations in leads I (2 mm) and aVL (1.5 mm) with reciprocal depressions in leads III and aVF (1 mm) (**Figure 1**). Based on this initial ECG, Cardiology was consulted, who believed the ECG was concerning for STEMI. However, high-sensitivity troponin-I was within normal limits (28 ng/L; reference <45 ng/L) and transthoracic echocardiogram indicated normal function with ejection fraction of 60% to 65%, rendering further cardiac biomarker and imaging evaluation unwarranted. Non-contrast computed tomography (CT) of the head demonstrated diffuse SAH in the basal cisterns and Sylvian fissures, consistent with Fisher Grade 3 hemorrhage. Subsequent CTA of the head and neck

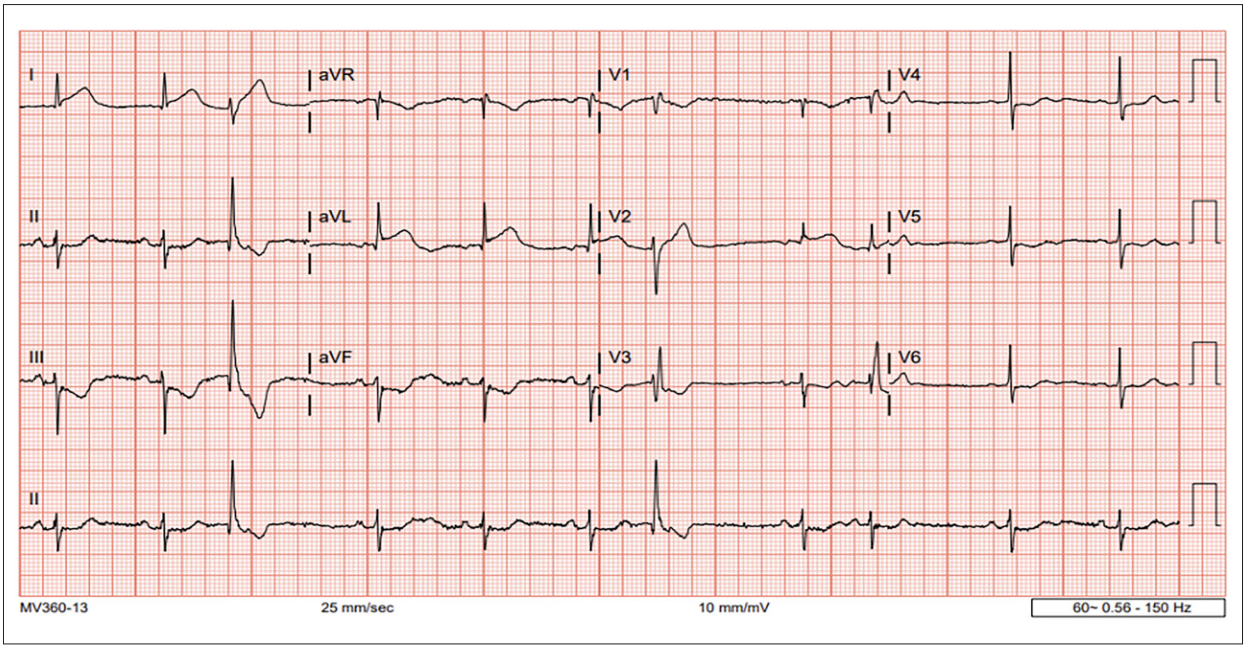

Figure 1. Admission electrocardiogram demonstrating ST-segment elevations in leads I and aVL with reciprocal ST depressions in leads III and aVF, a pattern concerning for lateral wall myocardial infarction.

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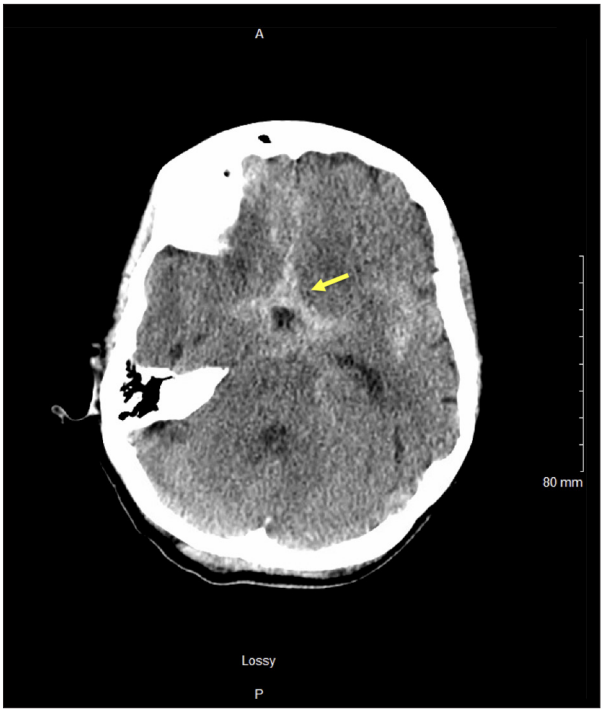

Figure 2. Axial computed tomography angiography (CTA) of the head demonstrating a ruptured anterior communicating artery (ACOM) aneurysm (arrow) with surrounding subarachnoid hemorrhage, most prominent within the basal cisterns. These findings confirm aneurysmal subarachnoid hemorrhage as the etiology of the patient's presentation.

revealed a ruptured 3×1 mm anterior communicating artery (ACOM) aneurysm as the bleeding source (**Figure 2**).

The patient was urgently transferred to the neurosurgical intensive care unit (Neuro ICU), where she underwent endovascular coil embolization of the ACOM aneurysm via right femoral access. Post-procedurally, she developed hyponatremia (serum sodium nadir 131 mmol/L), which was managed with 3% hypertonic saline (100 mL/h for 24 h) and oral salt supplementation. Magnetic resonance imaging (MRI) of the brain on hospital day 3 demonstrated acute punctate infarcts in the left superior cerebellum, right external capsule, and bilateral parietal cortices, suggestive of embolic phenomena (**Figures 3, 4**).

Her hospital course was further complicated by bilateral pleural effusions attributed to hypoalbuminemia (serum albumin nadir 2.0 g/dL) and *Cronobacter sakazakii* bacteremia, treated

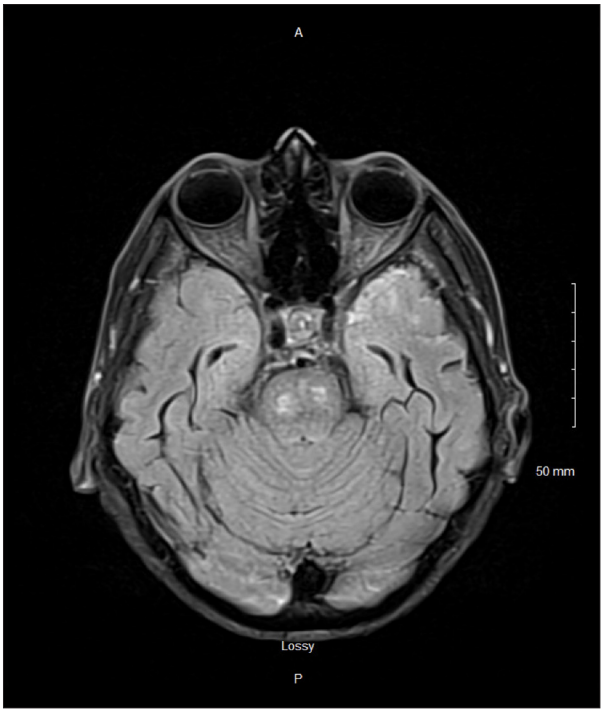

Figure 3. Axial magnetic resonance imaging (MRI) fluid-attenuated inversion recovery (FLAIR) sequence demonstrating multiple punctate hyperintensities in the cerebellum and parietal lobe, consistent with acute embolic infarcts in the setting of subarachnoid hemorrhage.

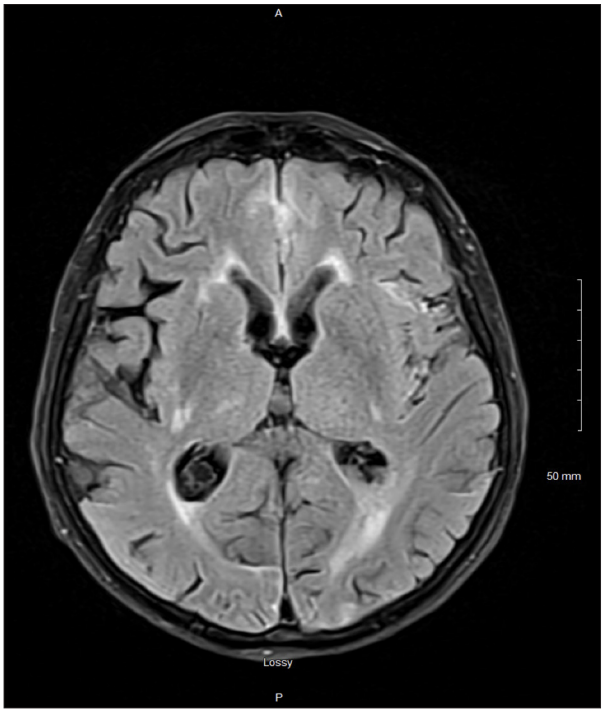

Figure 4. Axial magnetic resonance imaging (MRI) fluid-attenuated inversion recovery (FLAIR) sequence demonstrating multiple punctate hyperintensities in the parietal lobe, consistent with acute embolic infarcts in the setting of subarachnoid hemorrhage.

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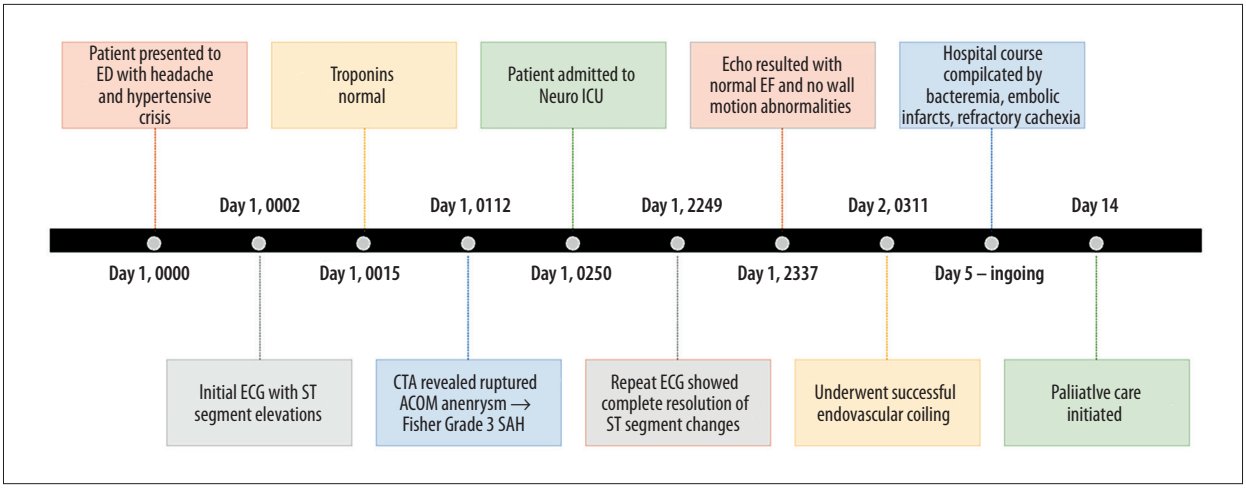

Figure 5. Timeline illustrating the patient's clinical course, including key diagnostic findings, interventions, and clinical progression, with time standardized relative to initial presentation.

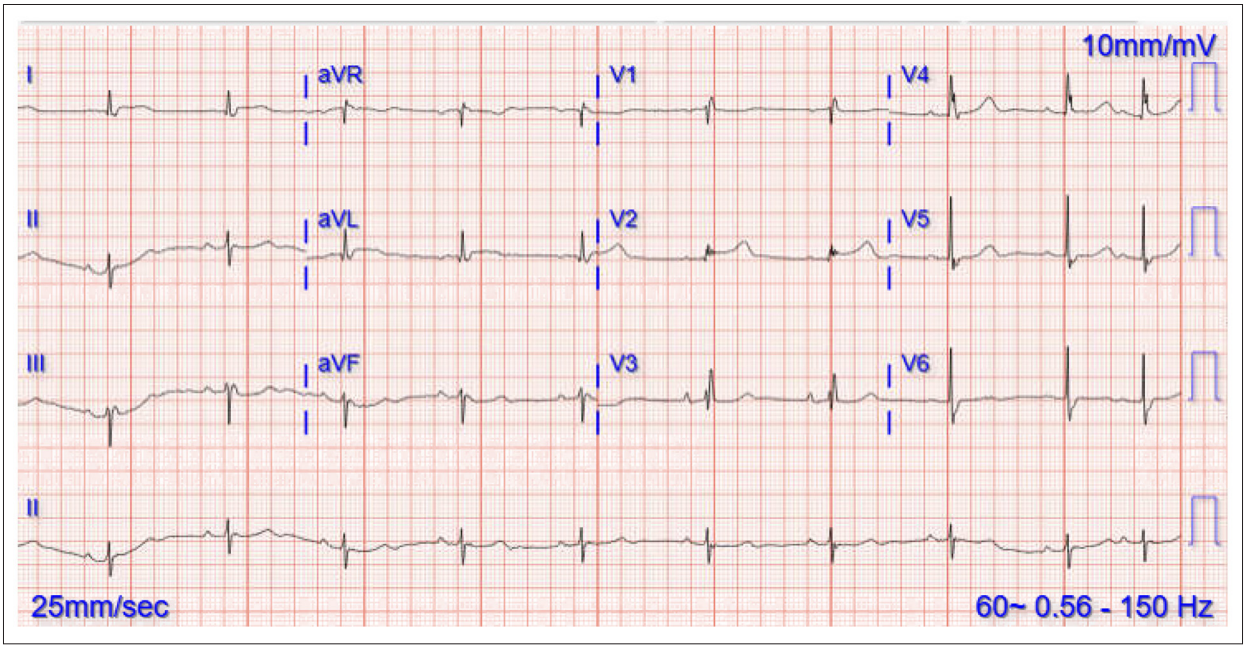

Figure 6. Repeat electrocardiogram obtained 1 day after initial presentation, demonstrating resolution of prior ST-segment elevations, supporting a non-ischemic etiology of the initial ECG abnormalities in the setting of subarachnoid hemorrhage.

empirically with cefepime (2 g intravenously every 8 h). During her second week of hospitalization, she developed a tender left knee effusion. Synovial fluid analysis revealed 11 000 white blood cells/μL (95% segmented neutrophils), but bacterial and fungal cultures remained negative. Despite aggressive and timely medical management (**Figure 5**), her functional status deteriorated due to cachexia and generalized pain, rendering her unable to participate in physical or occupational therapy. Given the poor functional prognosis and patient preference for non-invasive measures, a transition to comfort-focused care was initiated.

Discussion

This case exemplifies the diagnostic and therapeutic challenges of SAH in older patients, particularly when presenting with ECG abnormalities mimicking STEMI. Our patient's initial presentation with ST-segment elevations in leads I and aVL, alongside reciprocal depressions in III and aVF, created diagnostic ambiguity. However, the absence of troponin elevation and preserved left ventricular function on echocardiography (ejection fraction 60%-65%) precluded other etiologies such as stress cardiomyopathy or stroke-heart syndrome; and the resolution of ECG changes (**Figure 6**) following aneurysm coiling made

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thrombotic coronary occlusion very unlikely, although transient vasospasm-induced ischemia could not be definitively excluded. Notably, neurogenic stunned myocardium (NSM) is a catecholamine-mediated process reported in up to 28% of SAH cases, with SAH demonstrating the strongest association among neurological conditions [5]. NSM results from hypothalamic injury during SAH, triggering a sympathetic surge that induces coronary vasospasm and transient myocardial dysfunction [3]. Although our patient's echocardiography did not indicate significant cardiac dysfunction, elevated serum norepinephrine (>2000 pg/mL) in acute SAH has been shown to directly correlate with ECG abnormalities, particularly ST deviations in lateral leads, and typically resolves after aneurysm repair [6].

The pathophysiology of SAH-induced ECG changes mimicking myocardial infarction involves 2 interrelated mechanisms: (1) catecholamine toxicity, where excessive norepinephrine alters calcium handling in cardiomyocytes, prolonging action potentials and producing ST-segment deviations [7], and (2) microvascular dysfunction, where sympathetic hyperactivity induces coronary vasospasm, mimicking ischemia on ECG despite normal coronary anatomy [8]. These mechanisms underscore the necessity of rapid neuroimaging in older patients presenting with headache and autonomic instability, and diagnostic caution when ECG abnormalities suggest cardiac ischemia [9]. In this case, immediate CTA allowed prompt identification of the ACOM aneurysm, avoiding detrimental antiplatelet and anticoagulation administration while guiding timely endovascular intervention.

While many patients with SAH-induced ECG changes present with normal cardiac biomarkers and preserved systolic function, as in our patient, this is not universally the case. Banki et al observed that a significant proportion of SAH patients with evidence of functional denervation exhibited elevated troponin levels and transient regional wall motion abnormalities on echocardiography, most commonly involving the apical and midventricular segments—closely resembling takotsubo cardiomyopathy [3]. This suggests that autonomic-mediated repolarization abnormalities and stress cardiomyopathy share overlapping mechanisms, including catecholamine-mediated microvascular dysfunction and direct myocardial toxicity, warranting diagnostic diligence when a patient presents with simultaneous SAH and ST elevation on ECG [3,6].

In addition to lateral ST elevations seen in this case, SAH-related ECG findings vary and can mimic several acute coronary syndromes. Common patterns include deep, symmetric T-wave inversions, QT interval prolongation, and diffuse ST depressions (such as often observed in NSTEMI) consistent with subendocardial ischemia [7]. True STEMI patterns, such as anterior or lateral ST-segment elevation with reciprocal changes, have also been reported, complicating differentiation from primary ischemic events [5]. Wall motion abnormalities, when present, often follow noncoronary distributions similar to echocardiographic findings in takotsubo cardiomyopathy and typically resolve within 1 to 3 weeks after hemorrhage, supporting their functional and reversible nature [6,8].

Outcomes in patients with SAH and cardiac involvement tend to be worse, with higher rates of delayed cerebral ischemia, vasospasm, and longer ICU stay [6]. However, most neurocardiogenic systolic dysfunctions improve with supportive care, and mortality is usually driven by the neurologic injury rather than the cardiac component [3,5]; therefore, early recognition of neurocardiogenic injury is essential not only to avoid inappropriate therapies but also to anticipate complications and guide hemodynamic support in neurocritical care settings.

Our patient's MRI findings of acute punctate infarcts in the cerebellum and parietal cortex highlight SAH-associated hypercoagulability, a well-documented complication of cerebral hemorrhage, as platelet activation, endothelial dysfunction, and systemic inflammation after SAH promote thromboembolic events, even in the absence of traditional risk factors such as atrial fibrillation [10]. This hypercoagulable state, combined with immobility and critical illness, likely contributed to the observed embolic phenomena. Clinicians must remain vigilant for thromboembolic complications in SAH patients, particularly those with prolonged hospitalization or frailty.

Hyponatremia (Na 131 mmol/L) in this patient likely resulted from cerebral salt wasting (CSW), supported by elevated urine sodium (82 mmol/L) and clinical response to isotonic fluids. CSW occurs in 30% to 50% of SAH cases due to disrupted natriuretic peptide regulation and is a key contributor to electrolyte instability in this population [11]. Differentiating CSW (a hypovolemic state) from syndrome of inappropriate antidiuretic hormone (a euvolemic state) with urine studies is critical, as fluid management strategies diverge significantly between these entities.

The *Cronobacter sakazakii* bacteremia, although rare in immunocompetent adults, reflects the vulnerability of older SAH patients to nosocomial infections. In our patient, frailty (body mass index 15.6, hypoalbuminemia) and use of invasive devices (eg, peripherally inserted central catheters) compound this risk, underscoring the importance of rigorous infection control measures in this population [12]. Additionally, *Cronobacter sakazakii* is a gram-negative pathogen typically associated with contaminated powdered infant formula and has rarely been reported in older hospitalized patients, emphasizing the need for broad-spectrum antibiotic coverage in sepsis of unknown origin [13].

This case report is limited by its single-patient design, which prevents determination of causality between SAH, neurocardiogenic ECG changes, and subsequent complications. Additionally,

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the patient's advanced age and multiple concurrent conditions introduce confounding factors that complicate interpretation of the clinical course. Furthermore, the absence of coronary angiography and obtainment of serum catecholamine levels prevent definitive exclusion of occult coronary disease and sympathetic storm, respectively.

Conclusions

Subarachnoid hemorrhage in older patients can mimic ST-elevation myocardial infarction via catecholamine-mediated ECG changes, making early neuroimaging essential to avoid misdiagnosis and inappropriate treatment. Frailty increases the risk of complications, underscoring the need for prompt, multidisciplinary care. This case highlights the importance of diagnostic vigilance and the systemic impact of aneurysmal hemorrhage in this vulnerable population.

Institution Where Work Was Done

Northeast Georgia Medical Center, Gainesville, GA, USA.

Patient Consent

Informed consent was obtained from the patient.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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