***Case report***

Massive Pulmonary Embolism Revealing Right-Sided Infective Endocarditis Complicated by Intracerebral Hemorrhage in a Non-IV Drug User: A Case Report

# Abstract

Background: Right-sided infective endocarditis (RSIE) is an uncommon form of endocardial infection predominantly seen in intravenous drug users. Its diagnosis is often delayed or missed in non-IV drug users due to the low index of clinical suspicion. The association of RSIE with pulmonary embolism and intracerebral hemorrhage is rare but often fatal.  
  
Case Presentation: We present the case of a 53-year-old woman with insulin-dependent diabetes and a recent gluteal abscess, who was admitted with progressive dyspnea and acute neurological symptoms. She had no history of intravenous drug use or prior valvular disease. Initial investigations revealed severe anemia, thrombocytopenia, hyperkalemia, and elevated inflammatory markers. Transthoracic echocardiography showed large tricuspid vegetations and right heart dilation. CT imaging confirmed a massive pulmonary embolism and an acute intracerebral hemorrhage. Blood cultures isolated Staphylococcus aureus. Despite aggressive supportive therapy and antibiotic treatment, the patient died within five days from septic shock.  
  
Conclusion: This case underscores the diagnostic and therapeutic challenges of RSIE in non-traditional patients. Clinicians must maintain a high index of suspicion for RSIE when encountering septic pulmonary emboli, even in the absence of classical risk factors, especially when complicated by neurological events. Early echocardiographic evaluation and multidisciplinary management are essential.

# Keywords

Right-sided infective endocarditis, pulmonary embolism, tricuspid valve, Staphylococcus aureus, intracerebral hemorrhage, non-IV drug user

# Introduction

Infective endocarditis (IE) remains a potentially fatal condition despite advances in antimicrobial therapy and cardiac surgery. While most cases of IE involve the left-sided cardiac valves, right-sided IE accounts for 5–10% of all episodes and is predominantly seen in intravenous drug users, patients with intracardiac devices, or those on long-term hemodialysis.  
  
Tricuspid valve involvement is the hallmark of RSIE, and its presentation is often insidious. Unlike its left-sided counterpart, RSIE frequently leads to septic pulmonary embolism rather than systemic embolization. Complications such as pulmonary embolism and neurological events, including intracranial hemorrhage (ICH), are rare but associated with high mortality.  
  
We report a case of RSIE in a non-intravenous drug user, revealed by a massive pulmonary embolism and complicated by an acute intracerebral hemorrhage, culminating in rapid clinical deterioration and death.

# Case Presentation

A 53-year-old woman with a history of insulin-dependent diabetes mellitus and a prior right foot amputation for diabetic gangrene was admitted to our department with progressive shortness of breath over three weeks (New York Heart Association Class III). Twenty-four hours before admission, she developed sudden-onset heaviness and weakness in the left upper limb.  
  
She denied any history of intravenous drug use, alcohol consumption, hemodialysis, recent surgery, or known cardiac valvulopathy. Her past medical history included a recent gluteal abscess drained three weeks prior at a local health facility, treated with oral antibiotics.  
  
On admission, her vital signs were:  
- Blood pressure: 102/66 mmHg  
- Heart rate: 98 bpm (regular)  
- Temperature: 37.2°C  
- Oxygen saturation: 92% on room air  
- Respiratory rate: 24 breaths/min  
  
The physical examination revealed mild respiratory distress but no murmurs, petechiae, splenomegaly, or peripheral stigmata of endocarditis. Neurological assessment noted a decreased strength (4/5) in the left upper limb without facial involvement or speech disturbance.  
  
Laboratory tests showed:  
- Hemoglobin: 5 g/dL (microcytic anemia)  
- White blood cells: 21,000/μL (marked leukocytosis)  
- Platelet count: 53,000/μL (thrombocytopenia)  
- C-reactive protein: 256 mg/L  
- Prothrombin time: 52%  
- Serum potassium: 8 mmol/L (severe hyperkalemia)  
- Liver and thyroid panels: Within normal limits  
  
Blood cultures were drawn immediately upon admission.  
  
ECG: Sinus tachycardia at 96 bpm, with no ischemic or conduction abnormalities.  
Chest X-ray: Right ventricular enlargement with bilateral infiltrates.  
  
Transthoracic echocardiography (TTE) showed:  
- Two large, mobile vegetations on the atrial side of the tricuspid valve  
- Right atrial and ventricular dilation  
- Moderate tricuspid regurgitation  
- Pulmonary artery systolic pressure (PASP): 52 mmHg  
- Preserved left ventricular ejection fraction  
  
Brain CT scan revealed an acute parietal intraparenchymal hemorrhage without herniation. A CT pulmonary angiogram confirmed massive pulmonary embolism, involving the right superior lobar branch and bilateral segmental branches.  
  
Two sets of blood cultures isolated methicillin-sensitive Staphylococcus aureus (MSSA) within 48 hours, confirming the diagnosis of infective endocarditis.  
  
The patient received empirical intravenous antibiotics (vancomycin + ceftriaxone) later tailored to oxacillin upon culture results. Supportive measures included blood transfusion, potassium correction with insulin-glucose, and oxygen therapy.  
  
Given the presence of cerebral hemorrhage, anticoagulation was contraindicated, and surgical intervention was deferred due to her critical status.  
  
Despite aggressive resuscitation and targeted therapy, the patient's condition worsened, culminating in septic shock and death on day five of hospitalization.

# Discussion

This case illustrates a fulminant course of RSIE in a non-IV drug user, characterized by diagnostic delays, multiple organ complications, and limited therapeutic options.  
  
1. Epidemiological Considerations  
RSIE is traditionally associated with intravenous drug use, accounting for 90% of cases. Other risk factors include pacemakers, central venous catheters, congenital heart defects, and immunosuppression. In our patient, none of these were present. The only potential source of bacteremia was the recent gluteal abscess.  
  
Diabetes mellitus, particularly poorly controlled, is a recognized predisposing factor due to impaired immune defenses and frequent skin infections. The absence of classical signs (e.g., murmur, Janeway lesions, splinter hemorrhages) contributed to diagnostic delay.  
  
2. Diagnostic Challenges  
Tricuspid valve endocarditis often presents with nonspecific symptoms: fever, malaise, and respiratory complaints. Pulmonary symptoms may mimic pneumonia or pulmonary embolism. In our case, massive PE was the first clue prompting echocardiographic investigation.  
  
3. Septic Pulmonary Embolism (SPE)  
Septic pulmonary emboli are a hallmark of RSIE, caused by embolization of infected vegetations into the pulmonary circulation. CT imaging typically shows nodular infiltrates, cavitation, or filling defects, as in our patient. The association of massive PE and RSIE, though rare, may serve as an important diagnostic cue.  
  
4. Neurologic Complications  
Neurologic involvement occurs in up to 35% of IE cases, and hemorrhagic events represent 12–30%. Mechanisms include:  
- Rupture of mycotic aneurysms  
- Hemorrhagic transformation of embolic infarcts  
- Coagulopathy (e.g., thrombocytopenia, anticoagulant therapy)  
- Septic vasculitis  
  
In this case, the acute ICH may have been multifactorial: severe thrombocytopenia, possible septic vasculitis, and sepsis-induced coagulopathy.  
  
5. Management Dilemmas  
Standard RSIE treatment involves prolonged intravenous antibiotics. Surgery is considered for:  
- Persistent sepsis despite antibiotics  
- Large vegetations (>20 mm) with recurrent PE  
- Valve dysfunction with heart failure  
  
Our patient met criteria for urgent surgery. However, recent ICH precluded surgical intervention and anticoagulation for PE, limiting our treatment options.

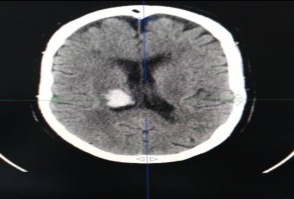
# Conclusion

This case highlights the lethal triad of RSIE, septic pulmonary embolism, and intracranial hemorrhage in a diabetic, non-IV drug user. It emphasizes the need for:  
- Early echocardiographic evaluation in patients with unexplained PE or sepsis  
- Vigilance for RSIE in patients with skin/soft tissue infections, even without classic risk factors  
- Prompt, multidisciplinary management to navigate therapeutic dilemmas  
  
Greater awareness and high clinical suspicion are essential to improve prognosis in such fulminant presentations.

# Figures

Figure 1: Transthoracic echocardiography showing large mobile vegetations on the tricuspid valve.

  
  
Figure 2: Brain CT scan revealing acute parietal intracerebral hemorrhage.



# References

[1] Habib G, et al. 2015 ESC Guidelines for the management of infective endocarditis. Eur Heart J. 2015;36(44):3075–3128.  
[2] Baddour LM, et al. Infective Endocarditis in Adults: Diagnosis and Management. Circulation. 2015;132(15):1435–86.  
[3] Murdoch DR, et al. Clinical presentation, etiology, and outcome of infective endocarditis in the 21st century. Arch Intern Med. 2009;169(5):463–473.  
[4] Eichenberger EM, et al. Right-sided infective endocarditis: review of management strategies. Infect Dis Clin North Am. 2018;32(3):539–555.  
[5] Thuny F, et al. Neurologic complications of infective endocarditis. Lancet Neurol. 2020;19(9):779–792.  
[6] Cooper HA, Thompson EC, Laureno R. Subarachnoid and intracerebral hemorrhage in infective endocarditis. Stroke. 2015;46(3):885–887.  
[7] Kang DH, et al. Early surgery vs conventional treatment for infective endocarditis. N Engl J Med. 2012;366(26):2466–2473.  
[8] Youssef G, et al. CT imaging of septic pulmonary embolism. Eur Radiol. 2021;31(1):15–25.  
[9] Liu C, et al. Clinical characteristics of right-sided infective endocarditis: a retrospective cohort study. BMC Infect Dis. 2020;20:705.  
[10] García-Cabrera E, et al. Neurological complications of infective endocarditis. Neurologia. 2013;28(3):143–149.