Septic Embolisation and Subarachnoid Haemorrhage Secondary to Fungal Endocarditis: A Case Report

Saba Zaidi[1], Muhammad Mubashir[2] and Mah Zareen[1]

[1]*Department of Neurology, Liaquat National Hospital and Medical College, Karachi, Pakistan*
[2]*Department of Medicine, Jinnah Postgraduate Medical Centre, Karachi, Pakistan*

ABSTRACT

Fungal endocarditis is a rare but severe condition that can lead to significant morbidity and mortality. The authors present a case of a 36-year female with no significant past medical history who developed fungal endocarditis, resulting in septic embolisation and subarachnoid haemorrhage. She presented with high-grade fever and altered mentation, accompanied by significant bicytopenia and elevated inflammatory markers. Blood cultures from a single site revealed the growth of *Candida albicans*, and high levels of galactomannan supported the diagnosis of fungal endocarditis. Although she received empirical antibacterial coverage, antifungal therapy was not initiated. The infectious disease team subsequently started treatment with intravenous fluconazole, and recommended urgent surgical intervention for mitral valve replacement. Unfortunately, the patient's clinical condition deteriorated, and a repeat CT scan revealed the development of a haematoma in the left superior parietal region and ischaemic infarction in the left frontal area. This case illustrates that fungal infections should be considered in the differential diagnosis even in previously healthy individuals presenting with severe illness. Prompt empirical antifungal therapy may have altered the clinical course and potentially saved the patient's life.

Key Words: *Infective endocarditis, Subarachnoid haemorrhage, Candida Albicans*.

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INTRODUCTION

Bacterial endocarditis (BE) comprises 80-90%, whereas fungal endocarditis (FE) accounts for 1-3% of all endocarditis instances.[1,2] Candida species are the primary culprits behind FE, responsible for less than 5% of all infective endocarditis (IE) instances but over half of all FE cases, with an in-hospital mortality rate of 36%. In recent years, the occurrence of IE has risen significantly. This is mainly due to the increased use of invasive medical procedures, artificial valves, and the growing number of patients with congestive heart failure or intravenous drug addiction.[3]

FE is primarily caused by Candida species, with *C. albicans* being the most common culprit. Other Candida species, such as *C. glabrata*, *C. tropicalis*, and *C. parapsilosis*, are also associated with the infection. Aspergillus species, including *A. niger*, *A. flavus*, and *A. fumigatus*, are frequently involved, particularly in cases of prosthetic valve endocarditis.

Correspondence to: Dr. Saba Zaidi, Department of Neurology, Liaquat National Hospital and Medical College, Karachi, Pakistan
E-mail: drsabazaidi@gmail.com

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Additionally, various other fungi linked to FE include *Cryptococcus neoformans*, *Histoplasma species*, *Microsporum species*, *Trichophyton species*, *Fusarium species*, *Paecilomyces species*, and *Pseudallescheria boydii*. FE has a higher mortality rate compared to BE, and there are distinct differences in the clinical courses of both infections.[4]

Diagnosing FE is often delayed, and the prognosis is usually very serious. Mortality rates for FE caused by pathogens range from 40 to 80%, but in some cases, a combination of antifungal medicines and valve replacement can lead to relatively satisfactory outcomes. The clinical symptoms of FE are similar to those of BE, with fever and abnormal heart murmurs being common among patients. For individuals with risk factors or a history of visceral infections, early echocardiography is essential for accurate diagnosis. Prolonged use of broad-spectrum antibiotics can make it difficult to diagnose FE, so it is necessary to obtain blood and/or valve biopsy cultures for confirmation. Screening for blood galactomannan can be helpful in certain cases. However, medical treatment options are limited and only result in temporary relief. Removing infected valve tissue is essential for completely eradicating the disease.[5-7]

This case is presented due to its unusual presentation of Candida endocarditis complicated by cerebral embolism and haemorrhage in a previously healthy young female.

Table I: Relevant laboratory workup.

Laboratory investigations	Day 1	Day 2	Day 3	Day 7	Day 10
Haemoglobin (11-13 g/dl)	7.69	8.4	10.9	9.2	8.5
Total leucocyte count (4-11 × 10^3 /cumm)	17.2	29	27.5	32.64	27.37
Platelets (150-300 × 10^3 /cumm)	68	77	76	118	112
Urea (10-50 mg %)	78	98	66	63	80
Creatinine (0.5-1.5 mg%)	1.26	1.31	0.9	0.8	0.96
Sodium (137-150 mmol/L)	144	151	146	145	145
Potassium (3.5-5.3 mmol/L)	3.6	3.6	3.9	3.2	2.9
SGPT (40 IU/L)	36				
INR	1.4	1.32			
Procalcitonin (<0.05 ng/ml)	12.68				
FDPs (<10 mg/dl)	>20				
D-Dimer (<0.5)	3.35				
Fibronogen level (200-400 mg/dl)	181				
HIV serology	Negative				
Blood culture 1	No growth				
Blood culture 2	No growth				
Blood culture 3	*Candida albicans*				

CASE REPORT

A 36-year recently married female was brought to the emergency department (ED) with a high-grade fever, documented up to 102°F, accompanied by myalgias for the past three weeks. The fever partially subsided with antipyretics but recurred. Over the past two days, she became disoriented, confused, and experienced one episode of a generalised tonic-clonic seizure. She was initially rushed to the nearest hospital, where a CT scan of the head revealed a right frontal subarachnoid haemorrhage (Figure 1A).

Further evaluation in the ED revealed no known comorbidities, addictions, significant travel history, or medicine use. She had never undergone surgical interventions or received recent blood transfusions, and her family denied any exposure to pets. On arrival, her vital signs were as follows: pulse of 120 beats per minute, blood pressure of 112/68 mmHg, and oxygen saturation of 85% on room air.

On examination, her Glasgow coma scale (GCS) score was 11/15 (E3, M5, V3), and her pupils were reactive. She was not obeying commands, grimacing to painful stimuli, and experiencing localised pain in her left upper extremity. The left plantar reflex was extensor. Chest auscultation revealed bilateral basal crepitations, and no heart murmurs were noted. Due to her worsening clinical condition, she was intubated and mechanically ventilated. Her blood pressure dropped, suggesting septic shock, and she was started on IV fluid resuscitation along with inotropic support.

Her laboratory workup (Table I) revealed significant bicytopenia and elevated inflammatory markers, including a raised total leucocyte count, C-reactive protein (CRP), and procalcitonin (PCT). Given these concerning results, broad-spectrum antibiotic coverage with intravenous Meropenem and Vancomycin was initiated after obtaining three sets of blood cultures.

The neurosurgery team recommended an urgent CT angiogram of the head, which was unremarkable (Figure 1B). An urgent echocardiogram was ordered due to a high suspicion of IE, particularly in light of the aneurysmal subarachnoid haemorrhage. The echocardiogram revealed thickening of both mitral valve leaflets, with small vegeta-tions attached to the atrial surface, suggestive of infective vegetations. Additionally, a large echogenic mass (3.0 × 1.0 cm), likely a thrombus, was seen attached to the left atrium, moving across the mitral valve. The ejection fraction was 55%, and there was moderate to severe mitral regurgitation (Figure 2A, B).

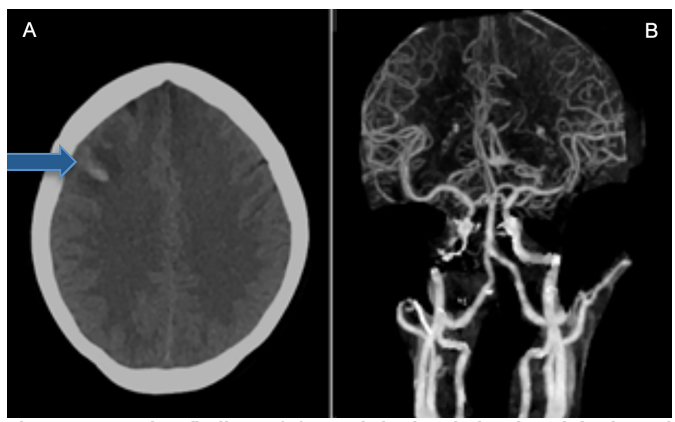

Figure 1: Imaging findings. (A) CT of the head showing right frontal subarachnoid haemorrhage (arrow). (B) CT angiogram of the head showing no evidence of an aneurysm.

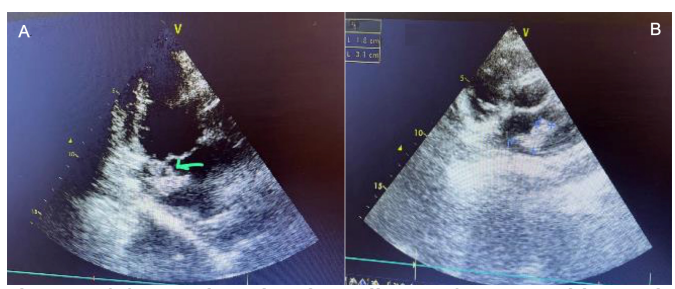

Figure 2: (A) Transthoracic echocardiogram (parasternal long axis view), green arrow shows the vegetation attached to the left atrium. (B) Blue marks, large echogenic mass moving across the mitral valve.

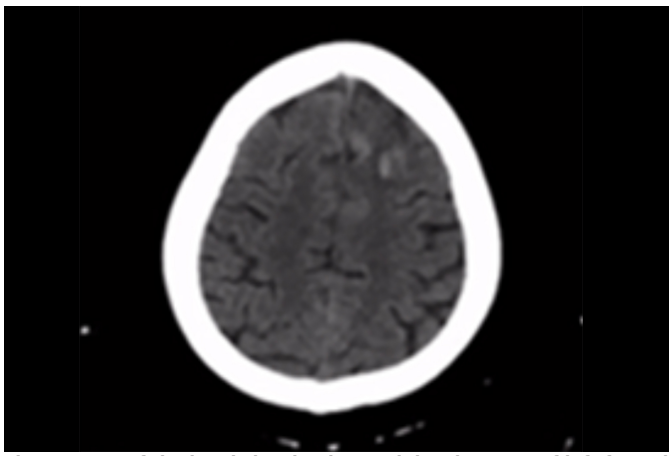

Figure 3: CT of the head showing interval development of left frontal haematoma.

Multiple expert teams were consulted, and the patient was transferred to the medical ICU. The infectious disease (ID) team advised awaiting blood culture results and conducted a thorough examination for possible injection sites or tattoo marks, which were absent. The cardiothoracic team suggested a transesophageal echocardiogram (TEE) for a more detailed assessment of the heart valves. The TEE revealed an enlarged echogenic mass attached to the posterior wall of the left atrium, obstructing the left ventricle and causing pseudo-mitral stenosis. Additionally, vegetations were noted on both leaflets of the mitral valve measuring 2.7 × 1.7 cm.

Blood cultures from three different sites are detailed in Table I, with one culture showing the growth of *Candida albicans*. Concurrently, elevated levels of galactomannan confirmed the diagnosis of FE, leading to septic embo-lisation and sub-arachnoid haemorrhage. The ID team escalated the antibiotic regimen to intravenous fluconazole and recom-mended life-saving surgical intervention, including mitral valve replace-ment.

However, due to her worsening clinical condition, a repeat CT scan of the head performed after 10 days of hospita-lisation revealed the interval development of a haematoma in the left superior parietal region and adjacent cortical sulci, along with a small area of ischaemic infarction in the left frontal region (Figure 3).

The cardiothoracic team recommended surgery, despite the high risk of intraoperative death due to extensive systemic involvement. However, the family deferred surgical intervention and changed the patient's code status to Do Not Resuscitate (DNR) after 15 days in the ICU without clinical improvement. Following the withdrawal of ventilatory support, the patient passed away immediately.

DISCUSSION

Among the Candida species, *Candida albicans* is the most frequently identified, representing about 35-60% of all FE cases.[8,9] Risk factors for FE encompass prosthetic heart valves or structural heart disease, cardiac implantable electronic devices, injection medicine use, indwelling central catheters, immunosuppression, a history of IE, low birth weight, and male gender. Remarkably, this patient did not exhibit any of these risk factors and had a negative human immunodeficiency virus (HIV) serology as well. Candida endocarditis can manifest with a range of symptoms. Initially, it might appear as a subacute condition with vague symptoms such as weight loss, sweating, chills, general discomfort, and exhaustion over weeks to months, making it hard to differentiate from symptoms caused by bacterial endocarditis. Fever stands out as the most common symptom at the onset of endocarditis. Embolic phenomena such as Osler's nodes, Janeway lesions, and Roth spots are rarely found in FE.[10] This patient had a high-grade fever (102 F) and myalgias along with one episode of a generalised tonic-clonic seizure, which made it challenging to diagnose. Diagnosis of FE is based on the modified Duke criteria, which comprise clinical observations, microbiological confirmation in blood cultures, and imaging characteristics indicating the presence of IE. Our patient met both of the two major criteria required for a conclusive diagnosis of IE, namely, three positive blood cultures and the presence of vegetation on an echocardiogram. The cell counts revealed notably elevated inflammatory indicators and abnormal D-dimer levels, suggesting severe sepsis. Initially suspecting bacterial endocarditis, broad-spectrum antibiotics were initiated after obtaining blood cultures, but the patient did not show improvement. A week later, the blood culture results indicated the presence of *Candida albicans*, changing our diagnosis to FE.

Common complications of FE can be categorised into intracardiac and extracardiac manifestations. Intracardiac issues may involve myocardial infarction, heart block, heart failure, and aneurysms. Extracardiac complications can include splenic embolism, brain embolism, limb embolism, and mycotic aneurysms. Septic embolism occurs in 30-80% of cases of Candida endocarditis. Subarachnoid haemorrhage resulting from FE is a rare occurrence, with only a limited number of cases documented in the literature, and its pathogenesis remains unclear. Proposed mechanisms include focal arteritis, vessel rupture, and spontaneous occlusion of a leaking aneurysm following haemorrhage. CT angiography (CTA) and magnetic resonance angiography (MRA) are typically the preferred initial imaging techniques for patients exhibiting neurological symptoms, such as a seizure in this instance. In this case, however, the CTA was unremarkable, maybe because an aneurysm might have been present, which ruptured, while the CT scan of the head indicated a right frontal subarachnoid haemorrhage.

Treatment for the patient commenced as soon as she was identified as septic, given the elevated inflammatory markers,

abnormal D-dimer levels, and declining blood pressure. In the ICU, she received intravenous fluid resuscitation and inotropic support while awaiting blood culture results. After a week, the cultures indicated growth of *Candida albicans*, prompting the initiation of fluconazole treatment and a recommendation for mitral valve replacement, which the family chose to defer. A study by Meena *et al*. concluded that in patients with FE, the combination of surgical intervention and antifungal therapy was linked to lower mortality rates compared to antifungal therapy alone.[2] This patient experienced clinical deterioration over the next 15 days and passed away.

It is important to highlight that the patient had no identifiable risk factors, making it challenging to determine what may have triggered the event. While rare, subarachnoid haemorrhage can occur as a complication of FE. It is crucial to consider this possibility in patients presenting neurological symptoms in the context of FE to ensure timely intervention for a better outcome. Additionally, further studies are needed to comprehend the pathogenesis of this complication.

FE, though rare, should be considered in patients presenting with sepsis, regardless of immune status or prior comorbidities. This case underscores the critical importance of prompt recognition, timely initiation of antifungal therapy, and a multidisciplinary approach, including early surgical consultation. Delays in appropriate management can lead to devastating complications such as embolic strokes and intracerebral haemorrhage, as tragically demonstrated in this young, previously healthy patient.

PATIENT'S CONSENT:
Informed consent was obtained from the patient's caretaker to publish the data concerning this case.

COMPETING INTEREST:
The authors declared no conflict of interest.

AUTHORS' CONTRIBUTION:
SZ: Conceptualisation, writing of the original draft, review, and editing.
MM: Writing of the original draft, reviewing, and editing.
M: Data curation.
All authors approved the final version of the manuscript to be published.

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