



Intracranial Hemorrhage in the Shadows of Infective Endocarditis as a Silent Menace: A Case Report

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Abstract

It is surprising when a patient's care-taker informed the doctor that their family member could suddenly speak in a different accent. Here we reported a case of a young female with infective endocarditis (IE), who later developed a foreign accent syndrome during her stay in the hospital ward. A plain computed tomography (CT) scan of the brain was performed and it showed a coarse and irregular calcific lesion in the left frontal region. Her condition deteriorated rapidly the next day and she died of intracranial haemorrhage. The haemorrhage was possibly caused by a ruptured mycotic aneurysm, a known complication of IE. [*Bangladesh Journal of Infectious Diseases, December 2022;9(2):76-79*]

Keywords: Infective endocarditis; foreign accent syndrome; mycotic aneurysm; intracranial haemorrhage

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Introduction

This case report presented a patient with sudden behavioural changes, in which she suddenly could speak in a different accent. With the underlying newly diagnosed infective endocarditis (IE), we highlighted the need to screen for central nervous system pathology in the event of sudden behavioural changes, although no significant neurological deficit was observed in this patient.

Case Presentation

We presented a case of a 36-year old Malay female with one year history of hypertension and end-stage renal failure (ESRF). Previously, she was on

Continuous Ambulatory Peritoneal Dialysis (CAPD). However, it was changed to haemodialysis a month before admission due to recurrent CAPD peritonitis. A right indwelling jugular catheter was inserted for vascular access. The patient had a one-day history of lethargy with fever, chills, and rigour during her haemodialysis treatment at a private centre. She was then referred to a tertiary centre for further assessment. During the examination, she was alert and conscious. Her lungs were clear and her abdomen was non tender. A strong systolic ejection murmur was heard over the left sternal edge during auscultation. Certainly! Here's the improved sentence with the inclusion of the increased CRP:

Upon admission, her laboratory results showed a total increase in white blood cells ($13.8 \times 10^9/L$), a decrease in haemoglobin (6.0 g/L), a decreased platelet count ($130 \times 10^9/L$), an increased serum creatinine (426 $\mu\text{mol/L}$), an elevated C-reactive protein (CRP) level of 8.3 mg/dL, a significantly reduced estimated glomerular filtration rate (11 mL/min/1.72 m²), elevated blood urea nitrogen (12.7 mmol/L), and an echocardiogram (ECHO) was performed, revealing a vegetation over the aortic valve, measuring approximately 2.0 cm (height) x 0.8 cm (width).

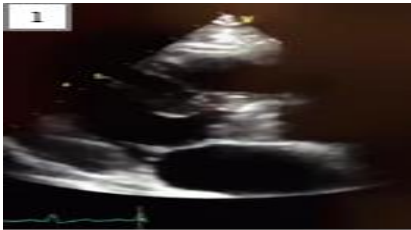


Figure I: Vegetation seen over the aortic valve, measuring approximately 2.0 cm (height) x 0.8 cm (width)

Initially, the patient was suspected of having Methicillin-resistant *Staphylococcus aureus* (MRSA) infection and started on intravenous (IV) Vancomycin. However, her blood culture later grew *Corynebacterium Striatum*. After seven days of antibiotic treatment, she started to develop behavioural changes. According to her husband, she started talking in a different accent and occasionally did not recognise him. Apart from speaking in a recognisably 'Sabahan' accent, there was no other cognitive dysfunction. She also denied having vomiting, headaches, or blurring of vision. Neurological examination did not show any significant neurological sign. A plain computed tomography (CT) scan of the brain was performed and the scan showed a coarse and irregular calcific lesion in the left frontal region of the brain, particularly anterior to the left Sylvian fissure, with evidence of minimal perilesional oedema. A magnetic resonance imaging (MRI) scan of the brain was scheduled the next day for further investigation. Unfortunately, the patient suddenly deteriorated the next morning after completing her regular haemodialysis treatment.

She developed a few bouts of seizures that responded to IV Valium. Her Glasgow Coma Scale (GCS) dropped to 8 over 15. Upon examination, both of her pupils were unequally dilated. The right side of her upper and lower limbs was remarkably weaker than the left side of her upper and lower limbs. Her blood pressure was 139/62 mm Hg with

a heart rate of 122 beats per minute. Her body temperature was 37.7 °C and her oxygen saturation level was 100% under ambient air. Her blood glucose was 6.6 mmol/L and blood Vancomycin level was 22 mcg/mL (slightly higher than the upper limit). A second CT Brain scan was performed immediately. There was a large acute intraparenchymal haemorrhage with peri-haemorrhagic oedema in the left frontal and parietal lobes. The lesion measurement was 8.0 cm (AP) x 5.2 cm (W) x 6.2 cm (CC), with a midline shift of 1.2 cm to the right and uncal herniation.

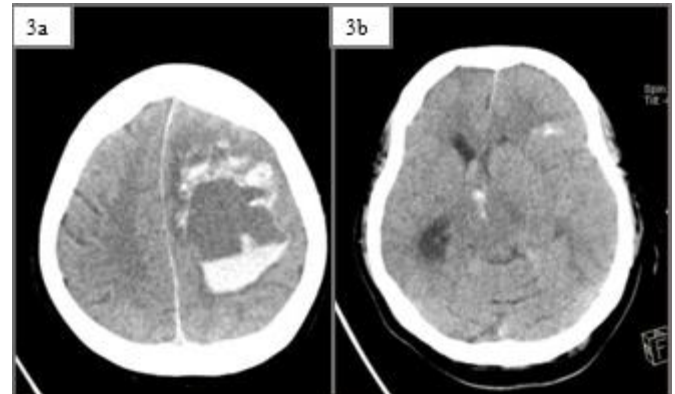


Figure IIIa-b: The Contrasted-Enhanced CT Brain showed a large acute intraparenchymal haemorrhage with peri-haemorrhagic oedema in the left frontal and parietal lobes. The lesion measurement was 8.0 cm (AP) x 5.2 cm (W) x 6.2 cm (CC), with a midline shift of 1.2 cm to the right and uncal herniation.

The patient was immediately referred to the neurosurgery team. Following a consultation session with the patient's family, we decided that the patient would be treated under palliative care. This decision was made based on the patient's poor prognosis and deteriorating condition. More importantly, we could not perform surgery on her as the risks associated with this procedure were high. Despite our efforts in finding the best treatment for her illness, she passed away the next morning. The cause of death was reported as intracranial haemorrhage. The haemorrhage was possibly caused by a ruptured mycotic aneurysm, which was a complication of IE.

Discussion

Chronic Haemodialysis (CHD) patients have a high risk of developing IE. The incidence of IE among CHD patients is approximately 50 to 60 times higher than the healthy population.¹ A retrospective study reported that the incidence of IE in CHD patients was 2.5% cases.²

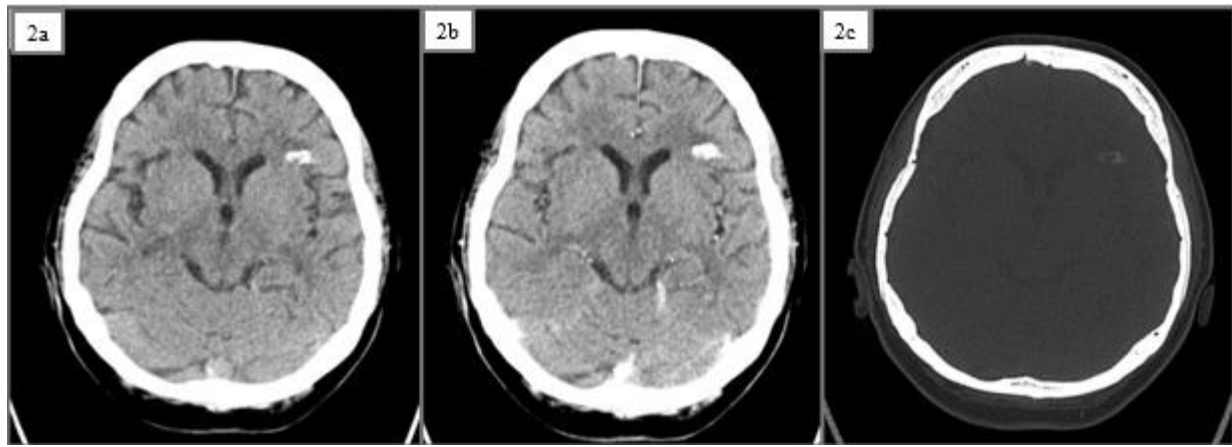


Figure IIa-c: A plain CT Brain showed a coarse and irregular calcific lesion in the left frontal region of the brain, particularly anterior to the left Sylvian fissure, with evidence of minimal perilesional oedema.

In CHD patients, the involvement of the right side of the heart in IE is relatively rare¹. This is true even if the patient is always inserted with a central venous indwelling catheter or arterio-venous fistula for vascular access. The incidence rate of IE depends on the type of heart valve. Mitral valve involvement (40.0% to 56.0%) accounted for a majority of the cases, followed by the aortic valve (21.0% to 43.0%) and tricuspid valve (0.0% to 26.0%)³. A study showed that 5.0% patients with IE manifested intracranial haemorrhage (ICH)¹. ICH is mostly attributed to the rupture of a mycotic aneurysm, though it is difficult to prove this as the lesion often disintegrates during the haemorrhagic process⁴. ICH may arise from abrasion of the arterial wall that occurs during uncontrolled infection where the emboli weaken the blood vessel wall. This condition is also exacerbated by the fact that the endothelium integrity of CHD patients is affected by persistent exposure to the uraemic toxin⁵. Therefore, CHD patients are usually prescribed with an anticoagulant during their dialysis treatment to prevent blood from clotting and to avoid any ischemic events, such as stroke. Stroke is caused by the obstructing emboli that could precipitate haemorrhagic transformation. A prompt antibiotic initiation is the main treatment to prevent the development of ICH. Embolism occurs in 22.0% of patients, and embolic events (including ones that contribute to ICH) are considered indications for valvular surgical intervention, as outlined by the European Society of Cardiology⁶. However, the guidelines provided by the American Heart Association (AHA) and the Society of Thoracic Surgeons recommended that surgery in IE patients should be delayed by at least four weeks from the onset of cerebral complications such as ICH⁷.

A study by Yoshioko et al⁸ suggested that early surgical intervention within two weeks of onset could minimise neurological deficit that may result from the exacerbation of cerebral haemorrhage during the surgery. However, it is also worth noting that most observational studies found that patients who are undergoing CHD have high perioperative mortality. For example, Dohmen and colleagues reported that CHD patients who had the disease for approximately five years showed a 0% survival rate after they underwent surgery for IE⁹. However, this could be that the patients selected for surgeries were already at a late stage of the disease where serious complications. On a positive note, another study found that CHD patients who underwent valvular surgery had marginally better survival rates than those who underwent nonsurgical treatment (56% versus 30% survival in one year)¹⁰.

Conclusion

In this case report, we presented a patient with CHD and IE, who later developed sudden behavioural changes. As mentioned above, there is no definitive consensus regarding the best time for surgical intervention. However, a few studies suggested that early intervention minimise the risk of postoperative neurological deficits from the exacerbation of haemorrhagic lesions. The occurrence of haemorrhagic lesions, however, is relatively low.

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Conflict of Interest

The authors declared no conflict of interest.

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Contribution to authors

Author's contribution: Conception: AZMS, UA; Collection and assembly of data: AZMS, UA, MRA; Writing manuscript: AZMS, UA; Editing and approval of final draft: AZMS, UA

Data Availability

Any inquiries regarding supporting data availability of this study should be directed to the corresponding author and are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate

Not applicable

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