CASE REPORT

# Stroke Chameleon: Acute Bilateral Middle Cerebral Artery Infarct Complicated with Acute Pulmonary Oedema

***Baharuddin KA[a], Wanshah N[a], Abdul Halim S[b], Ghazali FR[c]***

[a]Department of Emergency Medicine, School of Medical Sciences, Universiti Sains Malaysia, Kelantan, Malaysia
[b]Department of Internal Medicine, School of Medical Sciences, Universiti Sains Malaysia, Kelantan, Malaysia
[c]Department of Radiology, School of Medical Sciences, Universiti Sains Malaysia, Kelantan, Malaysia

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**Corresponding Author**
Prof. Dr. Kamarul Aryffin Baharuddin
Department of Emergency Medicine, School of Medical Sciences, Universiti Sains Malaysia, Kelantan
E-mail: amararyff@usm.my

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## ABSTRACT

Acute bilateral middle cerebral artery (MCA) infarctions are an extremely rare event. However, it can cause disruption of bilateral corticothalamic networks leading to coma. Detection of this condition is challenging as the early ischemic signs found on the non-contrast CT (NCCT) is depending on comparison between infarcted region and its normal counterpart. We report a case of 60-year-old man presented with acute comatose state and pulmonary oedema secondary to hypertensive emergency. His NCCT brain showed acute infarction of bilateral MCA territories. His acute pulmonary oedema subsequently resolved with the treatment. However, he succumbed to his illness and its complications on day 7 of admission. In this case report, we explore the strategy to improve detection of bilateral infarction on NCCT and navigating the differential diagnosis of comatose state and acute pulmonary oedema brought about by the hypertensive emergency.

## INTRODUCTION

A stroke chameleon is a situation in which a stroke is revealed by clinical symptoms that are not usual in stroke patients. This leads to false negative diagnostic errors in which the incidence in Emergency Department (ED) varies between 2 to 26%. Most stroke chameleons involve posterior circulation strokes in which the common presentations are dizziness and altered mental status.[1] The commonest presentation of acute stroke cases is focal neurological deficits due to the sophisticated anatomy of cerebral arteries supplying one side of the brain. However, bilateral middle cerebral artery (MCA) occlusions are rare and the clinical presentations ranging from asymptomatic to fatal stroke. We report a case of acute simultaneous bilateral middle cerebral artery infarction presenting with coma and complicated by probable acute neurogenic pulmonary oedema.

## CASE PRESENTATION

A 60-year-old male was found unconscious with copious oral secretions at approximately 7:30 AM. He had been seen in good health just a few minutes earlier by his wife while they were together in the kitchen before he decided to go outside. According to the wife, the patient had been complaining of dizziness the day before the event. His underlying medical conditions were diabetes mellitus and hypertension and he has poor adherence to his medications. He was brought to an Emergency Department (ED) at 8:50 AM via an ambulance call.

Upon arrival to the ED, the patient remained unconscious. His blood pressure was 206/136 mmHg, heart rate of 100 beats per minute, respiratory rate of 30 breaths per minute, oxygen saturation ($SpO_2$) of 86% under room air, and afebrile with a body temperature of 37°C. There were frothy secretions coming out from his mouth and presence of crepitations on bilateral lower zones up to the midzones. However, there was no lower limbs oedema noted. His Glasgow Coma Scale (GCS) score was 3/15 and his pupils were equal and reactive bilaterally. His blood glucose level was 17.9 mmol/L. A bedside point of care ultrasound revealed poor cardiac

contractility with plethoric inferior vena cava. His lungs ultrasound shown bilateral B-profiles with no pleural effusion. His chest radiograph (Figure 1) showed features of pulmonary oedema.

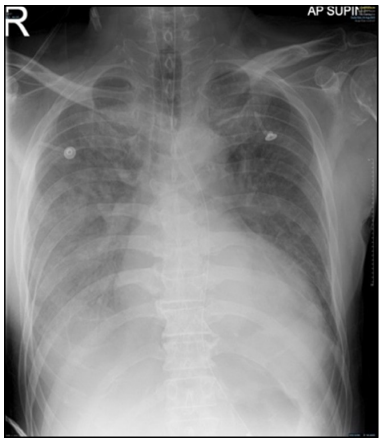


**Figure 1:** Chest radiograph showing features of pulmonary oedema - alveolar opacification, air bronchograms, upper lobe diversions and right pleural effusion

The patient's electrocardiogram revealed sinus rhythm with left ventricular hypertrophy, ST depression and T wave inversion at lateral leads. The patient was intubated for airway protection and respiratory distress and treated as acute pulmonary edema secondary to hypertensive emergency. He was started on nitrate infusion and sent for non-contrast computed tomography (NCCT) of the brain due to sudden onset of loss of consciousness. A consultation with the general medical and intensive care teams were made. Due to an access block, the patient was managed in the ED. An initial review of the CT scan by ED team was normal.

On the second day of hospitalization, the patient was treated as septic shock secondary to aspiration pneumonia, as there was newly documented fever. His total white cell count was 14.1 $x10^9$ /L and his C-reactive protein more than 293.3 mg/L. He was started on antibiotic therapy and vasopressor support. A re-evaluation of the initial CT scan by the managing team raised suspicion of acute ischemic changes in the bilateral MCA territories, indicated by the dense bilateral MCA sign and blurring of both basal ganglia. The formal radiology report from the previous evening noted an ASPECT score of 0 on the right side and an ASPECT score of 2 on the left side (Figure 2 ). The patient then was started on antiplatelet and referred to the neurology team.

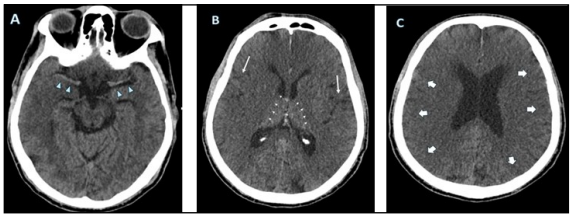


**Figure 2**: Initial NCCT shows (A) bilateral hyperdense MCA; (B) vanishing sylvian fissure (long arrow) and blurring of basal ganglia white matter and grey matter (arrowhead); (C) bilateral cortical sulcal effacement

A follow-up CT brain scan revealed generalized cerebral oedema with pseudo-subarachnoid hemorrhage (Figure 3).

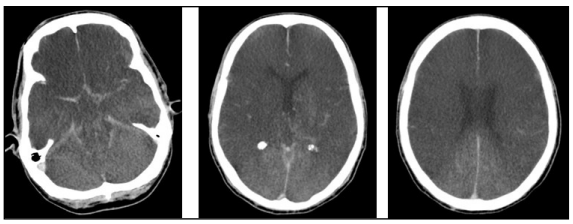

**Figure 3:** Repeated NCCT brain showing diffuse cerebral edema and appearance of pseudo subarachnoid haemorrhage.

The treatment strategy was then modified to focus on cerebral protection, incorporating osmotic therapy and desmopressin infusion. A consultation on potential surgical intervention was made with neurosurgery team. However, a conservative medical management was decided upon. Despite the efforts, the patient succumbed to his illness seven days after the initial presentation. The final diagnosis was acute ischemic stroke with bilateral MCA infarcts, complicated by septic shock secondary to aspiration pneumonia.

## DISCUSSION

A comatose condition can result from disturbances in the ascending reticular activating system or widespread insults to the bilateral brain hemispheres, such as occlusion of bilateral middle cerebral arteries (MCAs). Additionally, hypertensive emergencies may also induce coma due to posterior reversible encephalopathy syndrome (PRES) or brainstem infarctions. However, the decline in consciousness associated with these conditions is typically less abrupt.

Based on published case reports summarized in Table I, the initial presentation of bilateral MCA strokes varies from unilateral neurological deficits to comatose states, with the former being the commonest.[2] This variation arises as some patients may initially present with focal

**Table I:** Studies included in the literature review

| Authors/Year | Design | Sex | Risk Factor | Pulmonary Oedema | Stroke Occlusion Site | Admission NIHSS | GCS | Initial ASPECTS | i.v. rt-PA | MT | mRS 90 days |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Hu et al. 2007[3] | Medical | Female | Atrial fibrillation | NR | M1-M1 | NR | 4 | NR | NR | NR | NR |
| Nawashiro et al. | Case report | Male | Atrial fibrillation | NR | MCA-MCA | NR | 4 | NR | NR | NR | NR |
| Pop et al. 2013[2] | Case report | Female | Atrial fibrillation | NR | MCA - ICA | 26 | NR | 9 | Yes | bilateral MT | 0 |
| | Case report | Female | Atrial fibrillation | NR | distal ICA - | 18 | NR | 10 | Yes | bilateral MT | 3 |
| Dietrich et al. 2014[2] | Case report | Male | Atrial fibrillation, hypertension | NR | M1-M1 | NR | 15 | 9 | No | bilateral MT | NR |
| Ramos et al. 2018[2] | Case report | Female | Atrial fibrillation, Diabetes, hypertension | - | M1-M2 | 12 | NR | NR | NR | NR | 4 |
| | Case report | Female | Atrial fibrillation, Diabetes, hypertension | - | M1-ICA | 6 | NR | 10 | Yes | NR | 6 |
| London et al. 2019[2] | Case report | Female | Atrial fibrillation, hypertension | NR | M1-M1 | 11 | NR | NR | yes | bilateral MT | 1 |
| Braksick et al. 2018[5] | Case report | Female | NR | NR | M1-M1 | NR | NR | 10 | Yes | bilateral MT | 5 |
| Storey et al. 2019[2] | Case report | Female | Atrial fibrillation | NR | M1-M2 | NR | 5 | NR | NR | bilateral MT | NR |
| Escalard et al. 2020[2] | Case report | Female | Atrial fibrillation | NR | M1-M1 | coma | 3 | NR | NR | bilateral MT | 2 |
| Heyworth et al. | Case report | Female | Atrial fibrillation | NR | M1-M1 | 12 | NR | NR | NR | bilateral MT | NR |
| Khanna et al. 2021[2] | Case report | Female | Hypertension, COVID-19 infection | NR | M1-M2 | NR | NR | NR | NR | bilateral MT | 0 |
| Wu et al. 2021[2] | Case report | NR | Atrial fibrillation, ischaemic cardiomyopathy | NR | M1-M1 | 28 | | 7–10 | NR | bilateral MT | 5 |
| Kaesmacher et al. 2020[2] | Case series of 10 patients | Female 60% (6/10) | Atrial fibrillation 50% Diabetes mellitus 10% Arterial hypertension 70% | - | M1-M1 = 2 M2-M2 = 2 M1-M2 = 1 ICA-M1 = 4 ICA-M2 = 1 | 21 (IQR 5.5–30) | - | 7–10 | 7–10 | - | 20% mRS 0–2 |
| Joyce et al. 2022[2] | Case report | Female | Atrial fibrillation, hypertension, hyperlipidaemia | NR | M1-M1 | 13 | NR | 8 | NR | bilateral MT | 6 |
| Saad et al. 2022[2] | Case series | - | - | - | - | - | | - | - | - | - |
| Govindappa et al. | Case report | Female | Aortic stenosis | NR | M1-M1 | 21 | NR | 8 | Yes | bilateral MT | 0 |
| White et al. 2023[7] | Case report | Female | Diabetes, hypertension, hyperlipidaemia, previous cerebrovascular accident without neurological deficit | NR | M1-M1 | 12 | NR | - | NR | bilateral MT | NR |
| Li et al. 2023[8] | Case report | Male | Atrial fibrillation | NR | M1-M1 | 15 | NR | NR | Yes | bilateral MT | 2 |
| Srichawla et al. | Case report | Male | Atrial fibrillation | NR | M1-M1 | 7 | NR | NR | No | bilateral MT | NR |
| Bernava et al. 2023[2] | Case report | Female | Atrial fibrillation | NR | M1-M2 | 6 | 15 | NR | NR | bilateral MT | 0 |
| | Case report | Female | - | NR | M1-M1 | 29 | 5 | NR | NR | bilateral MT | 4 |
| Current case | Case report | Male | Diabetes, Hypertension | Yes | M1-M2 | NR | 3 | 0-2 | No | No | 6 |

Abbreviation: NIHSS=National Institutes of Health Stroke Scale; GCS=Glasgow coma Scale; ASPECTS= Alberta stroke program early CT score; i.v. rt-PA=intravenous recombinant tissue plasminogen activator; MT=mechanical thrombectomy; mRS=modified Rankin score; NR=not reported; MCA=middle cerebral artery; ICA=internal carotid artery

neurological deficits and often have single-vessel occlusions, which subsequently progress to multivessel occlusions affecting both hemispheres, leading to a decline in consciousness. Therefore, clinicians must be vigilant for bilateral large vessel occlusions in cases of acute onset of coma or progression to coma from initial focal neurological deficits.

Acute ischemic stroke with simultaneous bilateral occlusion of the anterior circulation is a very rare event with a reported incidence around 0.3% with the commonest site of artery occlusion is internal carotid artery-middle cerebral artery, followed by MCA-MCA.[2] Cardioembolic stroke is more likely to cause acute simultaneous occlusion of both MCA in this case as compared to large artery atherosclerosis stroke subtype. The emboli has higher chance of affecting both MCA simultaneously as they branch off the internal carotid arteries.

A diagnosis of bilateral MCA infarctions based on NCCT scan is challenging. Hyperdense artery sign and hypoattenuation of ischemic parenchyma are among the early ischemic signs seen on NCCT and the detection relies on comparing them from their normal counterpart. Both hyperdense MCA sign and focal attenuation are of value in distinguishing unilateral MCA occlusion and thus very challenging in the case of bilateral MCA occlusion as in our case. The interpreter should be vigilant of other early ischemic signs on NCCT such as blurring of basal ganglia's grey and white matter, cortical sulcal effacement and vanishing Sylvian fissure so as to improve the recognition of multiple large vessels occlusion stroke.

Apart from the acute bilateral MCA infarction, the elevated blood pressure coincidently causes acute pulmonary oedema in this case. Thus, the hypoxia-induced agitation commonly displayed in acute pulmonary oedema is absent in this case since the patient is already

comatose from the disruption of the neural networks responsible for the consciousness. Thus, it is possible that our patient had acute neurogenic pulmonary oedema (NPE) precipitated by bilateral MCA infarction. It is challenging to differentiate acute NPE and cardiogenic pulmonary oedema (CPE) as both conditions have similar presenting symptoms and signs with an abrupt onset and possibility of rapid resolution. However, the treatment strategy is slightly different in which control of circulating volume is an important aspect in CPE, whereas optimisation of the circulating volume is favourable in NPE to maintain perfusion in acutely ischemic brain tissue. However, both conditions are associated with high mortality rate with CPE has higher mortality rate compared to NPE.[10]

## CONCLUSION

Acute simultaneous bilateral MCA infarctions with concurrent acute NPE makes the diagnosis of stroke chameleon more challenging. High clinical suspicion and early multi-disciplinary team approaches may improve the diagnosis and expedite the treatment.

### CONFLICTS OF INTEREST

None.

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