# Cardiocerebral Infarction Presenting as Opalski Syndrome: A Case Report

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## Abstract

**Introduction.** Opalski Syndrome is a rare type of stroke variant presenting with signs of lateral medullary syndrome plus ipsilateral hemiparesis. A confirmed myocardial infarction simultaneously occurring with this acute ischemic stroke syndrome makes this an even more challenging case of Cardiocerebral infarction.

**Case.** The patient is a 48-year-old female, a known diabetic and asthmatic, and was seen due to a 3-day history of sudden onset of non-rotatory dizziness associated with diplopia, non-projectile vomiting, numbness of the left side of the face and lingual dysarthria. She was initially admitted in a primary hospital wherein she developed chest pain, dyspnea and diaphoresis. She was transferred and was received with findings of miosis of the left eye, rotational nystagmus, left dysmetria, decreased pain and temperature sensation on the right extremities, left central facial palsy, tongue deviation to the left side, left hemiparesis and upward Babinski on the left. We localize this lesion on the left lateral medullary area with involvement of the caudal left corticospinal tract. Hence, a clinical impression of Opalski Syndrome was made. This was confirmed with cranial MRI findings of T2/FLAIR hyperintense focus involving the left lateral aspect of the medulla.

The patient's Troponin I was also elevated and she was managed as a case of acute coronary syndrome - NSTEMI. Hence, a diagnosis of Type III Cardiocerebral infarction was made. Medical intervention was started with dual antiplatelet therapy and anticoagulation with noted clinical improvement.

**Conclusion.** This case report highlights the diagnosis of Opalski Syndrome in a patient also presenting with Cardiocerebral infarction. There should be prompt recognition of the following disease entities to have an effective treatment, avoid cardiac and neurologic sequelae, and achieve an overall favorable prognosis.


**Keywords:** Opalski syndrome, cardiocerebral infarction, lateral medulla, case report

## Introduction

Lateral medullary syndrome is an acute brainstem stroke syndrome resulting from a vascular event involving the posterior inferior cerebellar artery.[1,4] This is characterized by the presence of vertigo, diplopia, dysarthria, Horner`s syndrome, and numbness (of the ipsilateral face and contralateral limb). Moreover, it is not traditionally associated with any limb weakness.[2,3] When there are signs and symptoms of lateral medullary syndrome along with signs of ipsilateral hemiparesis, this is specifically called as Opalski Syndrome – a rare variant of the lateral medullary syndrome. There is no reported data as to the prevalence of this syndrome and it has only been mentioned in case report studies throughout literature. This syndrome is mainly due to the extension of the infarct caudally involving the corticospinal fibers after the pyramidal decussation. Hence, the presentation of ipsilateral hemiparesis/hemiplegia.

Cardiocerebral infarction occurs when there is presence of acute onset of neurological deficit, indicating acute ischemic stroke and angina with evidence of elevation of cardiac enzymes and ECG changes to confirm acute myocardial infarction.[5]

The annual risk of cardiocerebral infarction is highest in the first year after the index event (1.1%), followed by a much lower annual risk in the second to fifth years (between 0.16% and 0.27%). Cardiac sympathetic overactivity from an insular cortex lesion can provoke diffuse myocardial damage, also termed as "myocytolysis," which leads to elevation of cardiac enzymes. Moreover, brain–heart axis dysregulation may

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also be an alternative pathophysiology.[5] In this case report, the index patient manifested with acute ischemic stroke initially then followed by signs and symptoms of acute myocardial infarction. Hence, prompt, and accurate clinical examination paired with diagnostic imaging to identify and manage accordingly the syndrome is of great importance.

## Case Presentation

A 48-year-old female, known asthmatic and diabetic but non-adherent to Metformin, was received in the emergency department due to a 3-day history of non-rotatory dizziness. The patient had no previous hospitalizations, stroke or myocardial infarction and heredofamilial diseases identified were hypertension and diabetes mellitus on the maternal side. The patient also presented with diplopia, dysarthria, non-projectile vomiting and numbness of the left side of the face. She was then admitted at a local primary hospital wherein she was managed as a case of urinary tract infection. However, six hours prior to admission, she suddenly developed chest pain, dyspnea and diaphoresis. Hence, she was transferred to this institution for further work-up and management.

General examination, respiratory and abdominal examination were unremarkable. Pertinent systemic physical examination findings included normotension at 120/90 mmHg, tachycardia at 102 bpm with irregular rhythm and no murmurs. Detailed neurologic examination revealed a GCS of 15, and was oriented to three spheres. Patient had 4/5 manual motor strength examination in her left extremities. There was 50% decreased pain and temperature sensation on the right extremities as well as Horner's Syndrome on the left.

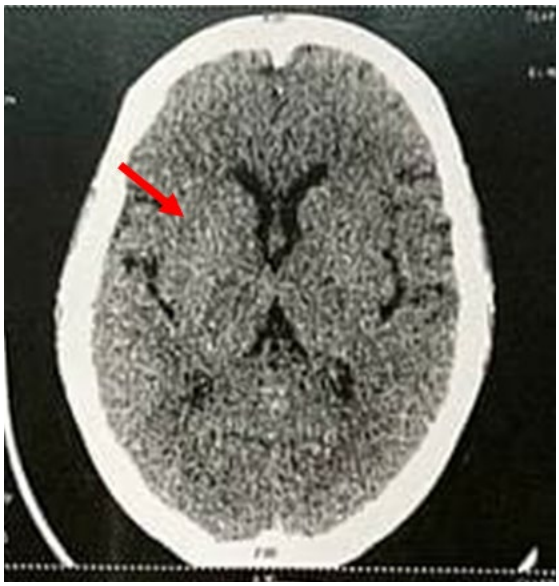

**Figure 1: Cranial CT scan plain, Axial view, at the level of the 3rd ventricle revealing a tiny lacunar infarct at the right lentiform nucleus (red arrow).**

Moreover, rotational nystagmus on left lateral gaze, left dysmetria, tongue deviation to the left, left facial central nerve palsy and mild lingual dysarthria were appreciated. Deep tendon reflexes were intact with upward Babinski on the left.

At the emergency room, the NIHSS was 9. Capillary blood glucose was 127mg/dL and 12 lead ECG revealed sinus rhythm with frequent premature atrial complexes occurring in atrial bigeminy. Chest radiograph showed a magnified heart and atheromatous thoracic aorta while plain cranial CT scan showed a consideration of a tiny lacunar infarct at the right lentiform nucleus and bilateral mastoiditis as shown in *Figure 1*.

Hematologic examination revealed: normal complete blood count, serum creatinine and electrolytes were normal except for mild hypokalemia at 3.4mg/dL. FBS was high at 147.21 mg/dL with an elevated HbA1c at 7.45%. Total cholesterol was normal at 164.86 mg/dL, Triglycerides was 117.7 mg/dL, LDL was high at 108.89 mg/dL, HDL was low at 32.43 mg/dL and VLDL was 23.54 mg/dL. Cholesterol ratio (Total Cholesterol/HDL) was calculated at 5.08 which is elevated (optimal TC/HDL ratio is <3.0 for females).

The patient had a Bamford Stroke Classification of Posterior Circulation Syndrome specifically a lateral medullary syndrome with caudal corticospinal tract involvement.

Referral to a neurologist was done. Cranial MRI with stroke series confirmed the clinical localization of lateral medullary syndrome with involvement of the caudal part of the medulla consistent with Opalski Syndrome. Specific cranial MRI findings were as follows: T2/Fluid-Attenuated Inversion Recovery (FLAIR) hyperintense focus involving the left lateral aspect of the visualized medulla measuring 1.0 x 0.8 x 0.9 cm as shown in *Figure 2*. It is hyperintense in the diffusion-weighted imaging (DWI) scans with equivocal signal in the Apparent Diffusion Coefficient (ADC) images as shown in *Figure 3* hence consistent with the diagnosis of Lateral Medullary Syndrome. However, the finding of ipsilateral hemiparesis is not consistent with the classic findings of Lateral Medullary Syndrome but this is more in line with the diagnosis of Opalski Syndrome, a rare variant of the Lateral Medullary Syndrome or Wallenberg's Syndrome.

Transthoracic echocardiography revealed concentric left ventricular hypertrophy with adequate contractility and systolic function (Simpson's Ejection Fraction of 74%), normal left atrium, mitral sclerosis, trivial tricuspid regurgitation and small pericardial effusion without tamponade. Troponin I was elevated at 0.605 ng/mL (normal value is less than 0.3 ng/mL) hence ACS-NSTEMI was confirmed. proBNP was also elevated at 668 pg/mL (normal value is less than 300 pg/mL). Coronary angiogram was ordered but was not done due to financial constraints.

The TOAST classification of cardio-embolism was highly suspected because of the ongoing myocardial infarction hence anticoagulation was started with Enoxaparin 40mg

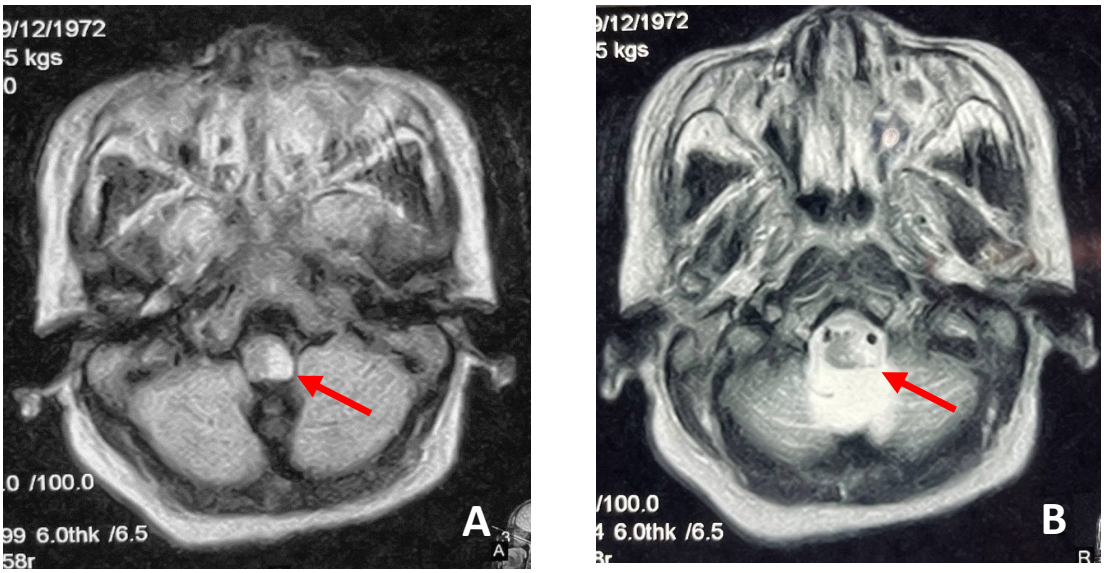


**Figure 2: (A) Cranial MRI T1-weighted imaging (T1WI) Axial view at the level of the Medulla revealing a hyperintense lesion involving the left lateral aspect of the medulla oblongata (red arrow); (B) Cranial MRI Fluid-Attenuated Inversion Recovery (FLAIR) Axial view with hyperintense lesion at the left lateral medulla (red arrow).**

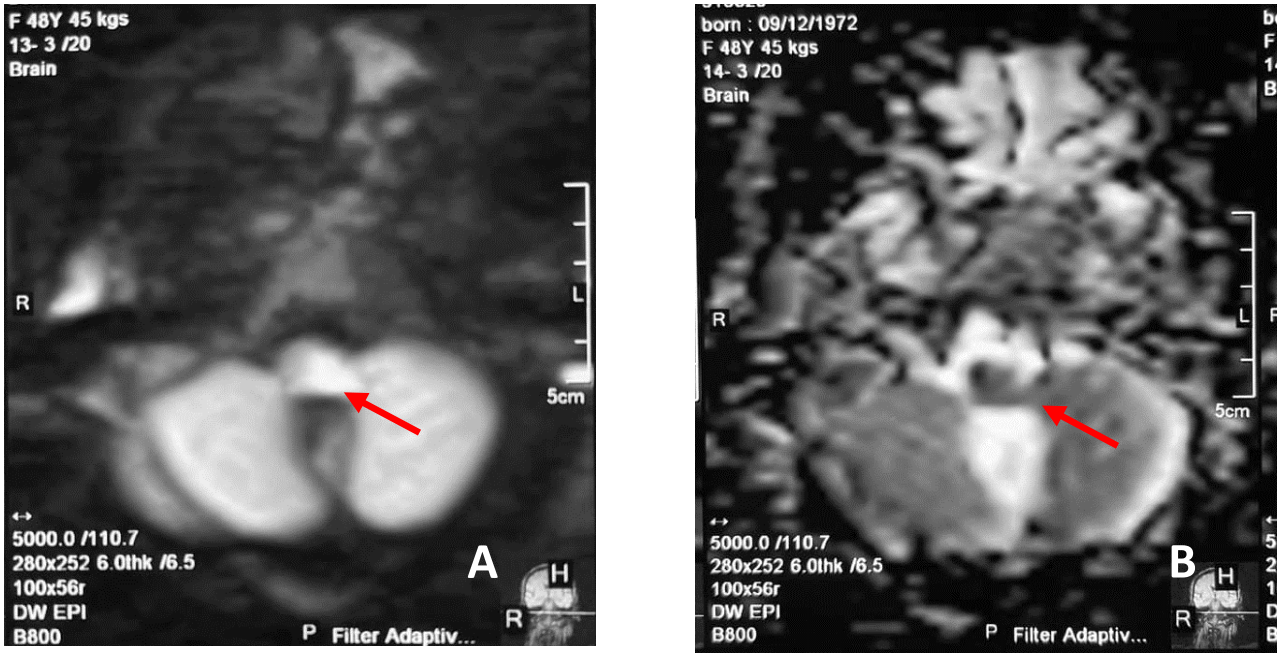


**Figure 3: (A) Cranial MRI Diffusion-weighted imaging (DWI) Axial view showing a hyperintense lesion in the left lateral medulla oblongata signifying an acute stroke (red arrow). (B) Cranial MRI ADC Images with equivocal signal in the left lateral medulla (red arrow).**

subcutaneously every 12 hours. Dual antiplatelet therapy was started with aspirin 100mg/tab 2 tablets given as loading dose then 100mg/tablet 1 tablet once daily, clopidogrel 75mg/tab 1 tablet once daily, citicholine 1 gm IV every 12 hours, atorvastatin 80mg/tab 1 tablet once daily, piracetam 1.2 gram/tablet 1 tablet three times daily, cinnarizine + dimenhydrinate 20mg/40 mg/tablet 1 tablet three times daily, ondansetron 2mg IV every 8 hours as needed for nausea and vomiting and omeprazole 40 mg IV once daily. Other medications started were the following: trimetazidine 35mg/tab 1 tab twice daily, carvedilol 6.25mg/tab 1 tab twice daily and cefuroxime 750mg IV every 8 hours which was given for the urinary tract infection. Over the course of the patient's hospitalization, the patient was also referred to

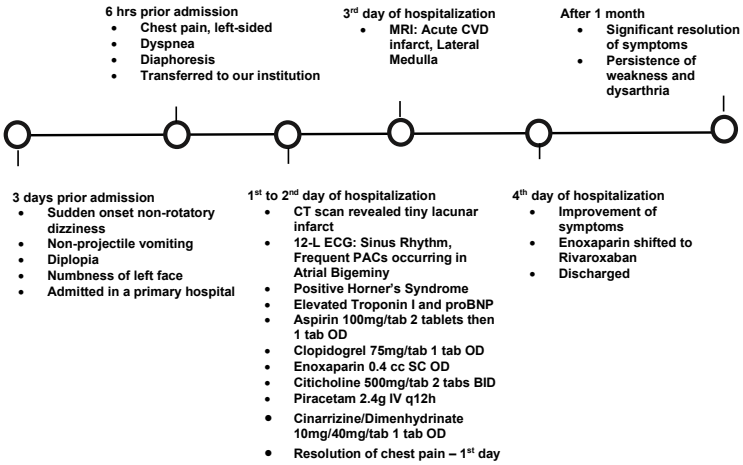


**Figure 4: Timeline of the patient's course from onset of symptoms until follow-up.**

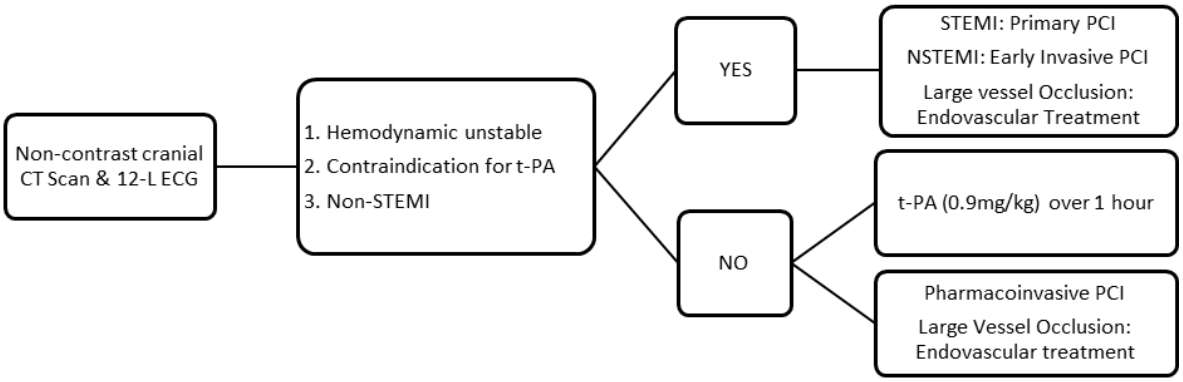


**Figure 5: Flowchart for the management of Cardiocerebral Infarction (Taken from: Habib, et. al. Cardio-Cerebral infarction syndrome: definition, diagnosis, pathophysiology, and treatment. J Integr Cardiol. 2021; 7)**

Rehabilitation Medicine and underwent passive limb exercises and mobilization of the extremities.

On the 4th day of hospitalization, the patient's vital signs were stable with blood pressure ranging from 110-130/70-80 mmHg and heart rate of 62-77 bpm. The condition of the patient improved with conservative medical treatment. Neurological examination at discharge revealed slight weakness on the left lateral extremity (4/5 manual motor strength) and the patient was able to ambulate with assistance. However, the patient still had dysarthria, diplopia, miosis and ptosis of the left eye. She was then discharged with home medications of triple antithrombotic therapy of rivaroxaban 10mg/tab 1 tablet once daily, aspirin 100mg/tab 1 tablet once daily, and clopidogrel 75mg/tab 1 tablet once daily. Metformin, carvedilol, atorvastatin and trimetazidine were also continued.

After one month, the patient had resolution of her dizziness and diplopia but still had slight weakness of the left extremities and lingual dysarthria. There was also no recurrence of any chest pain nor any shortness of breathing. The patient was adherent to the discharge medications but unable to have a formal follow up consult with her physicians thereafter.

*Figure 4* shows a summary of the timeline and course of hospitalization of the patient. Informed consent was provided by the patient.

## Discussion

Opalski Syndrome is a rare variant of the lateral medullary syndrome with ipsilateral hemiplegia that was first described by Opalski in 1946.[6] As previously discussed, neuroradiological findings obtained by conventional MRI have shown that the lesion causing this syndrome is located lower than in the Lateral Medullary Syndrome (Wallenberg's syndrome) and involves the corticospinal fibers caudal to the pyramidal decussation hence the presentation of ipsilateral hemiparesis.[7] Another consideration is that the ischemia may be due to additional implication of the posterior spinal artery. The other possible explanations have been given by *Liu et al*, who considered that the motor deficit may be due to

compromised medullary penetrating arteries which arise from the distal vertebral artery or the anterior spinal artery and supply the pyramidal fibers below the decussation and this may also be the reason for the ischemia in a very specific location in the medulla.[8]

This was clearly seen in this case due to the lateral caudal medullary location of the hyperintense lesion seen in MRI DWI axial sections. In addition, in order to confirm that the lower medullary infarction is responsible for this acute stroke syndrome, DWI was done which demonstrated temporal association between clinical signs and infarction as seen *in Figure 3*

Lateral medullary syndrome may vary in its manifestations/symptomatology since the damage may involve either of the following: the inferior cerebellar peduncle and dorsolateral medulla, descending spinal tract, the nucleus of the trigeminal nerve, nuclei, and fibers of the vagus nerves and glossopharyngeal, descending sympathetic tract fibers, spinothalamic tract, and vestibular nuclei.[1,2]

The etiology of lateral medullary syndrome and possibly in Opalski Syndrome includes the following: atherosclerotic disease; hypertension; dissection of vertebral arteries; cardiogenic embolism: cardiac diseases with risk for embolism including atrial fibrillation, mechanical prosthetic valves, left atrial or ventricular thrombus, dilated cardiomyopathy, myocardial infarction, and infective endocarditis; small vessel disease; hypoplastic vertebral artery; and Moya-moya disease.[1] For the index patient, the identifiable risk factors may be accelerated atherosclerotic disease due to uncontrolled diabetes mellitus and possible cardio-embolism due to ongoing simultaneous myocardial infarction and also the infratentorial location of the stroke is also claimed to be a feature suggestive of cardioembolic stroke as stated in the Stroke Society of the Philippines Handbook of Stroke.[9]

For this index case, Opalski syndrome was the presenting disease entity of her cardiocerebral infarction. Currently, this is the first reported case of the occurrence of Opalski Syndrome together with cardiocerebral infarction. Cardiocerebral infarction is defined as the occurrence of acute ischemic stroke (AIS) and acute myocardial infarction (AMI), either at the same time (simultaneous or synchronous) or one after the other (metachronous). In a single institutional series done at the Philippine General Hospital from April 2017 to April 2020, the prevalence rate was noted to be at 0.79% (0.55% for metachronous and 0.25% for synchronous).[10] Moreover, in the said study, the primary outcome of all-cause mortality was 45%, the majority of which were cardiovascular deaths (69%) and only one patient (8%) died of brain herniation. Five patients (17%) had a good functional outcome on discharge and at 30 days post-stroke, only 6 (21%) patients had good functional outcome.[10] Thus, highlighting the need for further improvement and knowledge in the management of these patients in order to minimize morbidity and mortality.

The Al-Shifa Hospital also provided a Classification of Cardiocerebral Infarction Syndrome (CCIS) which divided it into three types: Type I is defined as an acute myocardial infarction occurring less than 12 hours with acute ischemic stroke occurring less than 4.5 hours from last known well or if patients awake with stroke symptoms or if patients have unclear time of onset > 4.5 hours from last known well, or MRI findings with diffusion-positive FLAIR negative lesions. Type 2 CCIS is defined as acute ischemic stroke occurring less than 4.5 hours from last known well, or if patients awake with stroke symptoms or have unclear time of onset > 4.5 hours from last known well, MRI with diffusion-positive FLAIR negative lesions) after recent myocardial infarction (myocardial infarction in the previous 3 months but more than 12 hours). Lastly, Type 3 CCIS is defined as an acute myocardial infarction occurring less than 12 hours after recent ischemic stroke (ischemic stroke in the previous 3 months but more than 4.5 hours).[5] For this case, the patient is classified as Type 3 cardiocerebral infarction since the patient had a three-day history of neurologic symptoms followed by a six-hour history of angina, dyspnea, and diaphoresis upon presentation at the emergency room.

According to the scientific statement from the American Heart Association/American Stroke Association (AHA/ASA) guideline last 2018: for patients presenting with concurrent acute ischemic stroke and acute MI, treatment with IV alteplase at the dose appropriate for cerebral ischemia, followed by percutaneous coronary intervention (PCI) and stenting if indicated, is reasonable but currently there are no specific recommendation for patient with non-STEMI or for patients with STEMI and with contraindication for thrombolytic therapy such as in the case of our index patient.[5] Intravenous alteplase was not given for this case because the patient was received beyond the golden period of IV thrombolysis as well. PCI is necessary according to the treatment pathway shown in *Figure 5* however it was not done due to financial constraints. The timing of an invasive strategy such as PCI should be based on the patient's risk factors. The current ESC guidelines recommend an immediate invasive strategy in patients with 'very-high characteristics', such as hemodynamic instability, refractory angina, life threatening arrhythmias, acute heart failure with refractory angina or ST deviation, recurrent dynamic ST- or T-wave changes. Furthermore, an early (< 24 h) invasive strategy is recommended in patients with a global registry of acute coronary events (GRACE) risk score > 140, dynamic ST-segment and T-wave (ST-T) changes or a typical rise-and-fall in cardiac troponin. In addition, an invasive strategy is recommended in patients with at least one of the following intermediate-risk criteria such as: diabetes mellitus, renal insufficiency, congestive heart failure, early post-infarction angina, recent PCI, prior CABG, GRACE score >109 and <140.[11] For the index patient, there was absence of high-risk characteristics, and the GRACE score is only at 91. However, there was rise in Troponin I, a high-risk characteristic, and the patient also has Diabetes Mellitus, an intermediate risk characteristic. Hence, PCI is an ideal medical intervention in this case.

However, since PCI was not done, medical intervention was employed for this case. As stated in an article by *Habib et. al* on cardiocerebral infarction, the suggested medical treatment is triple antithrombotic therapy of aspirin (75-100 mg), clopidogrel (75 mg), and oral anticoagulant, rivaroxaban 2.5 mg twice a day or warfarin, given for one month followed by double therapy of a single antiplatelet and anticoagulant for one year.[5]

In addition, the use of thrombolytics is considered to be contraindicated and primary PCI for STEMI and early invasive PCI strategy for non-STEMI patient is recommended.[5] However, this was not done in the patient due to financial constraints. While our institution has access to a multidisciplinary team of neurologists, cardiologists, ICU specialists, & rehabilitation medicine that are needed to handle such complicated case, limited financial support, thrombolysis and PCI are major gaps to stroke and coronary care that were identified and should be addressed accordingly.

Furthermore, according to the study of *de Castillo, et. al*, dual antiplatelet therapy plus anticoagulation was employed in 66% of the patients with either synchronous or metachronous cardiocerebral infarctions which is similar to the intervention employed in this case.[10] Aside from cardiocerebral infarction, other indications for triple antithrombotic therapy are as follows: patients diagnosed with concomitant acute coronary syndrome (ACS) and atrial fibrillation & patients diagnosed with ACS and who underwent PCI.[12]

## Conclusion

This case report highlights the importance of an astute localization of rare stroke presentations such as Opalski Syndrome that thorough neurologic examination is of equal and great importance as with appropriate imaging technique. In this presented case, Opalski syndrome was accurately diagnosed clinically and confirmed with Cranial MRI. Furthermore, this was managed with medical treatment with triple antithrombotic therapy and thus contributes to the previously reported literature on cardiocerebral infarction.[5] With a concurrent myocardial infarction, a deeper understanding of the vascular lesions of both the heart and brain warrants an efficient and safe treatment administered by a multidisciplinary team in order to avoid further cardiac and neurologic sequelae and achieve an overall favorable prognosis.

**Conflict of Interest.** The authors declares that there is no conflict of interest regarding the publication of this case report.

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