Review on Acute Cardio-Cerebral Infarction: a Case Report

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pISSN: 2302-1381;
eISSN: 2338-4506;
http://doi.org/10.15850/ijihs.v9n2.2178
IJIHS. 2021;9(2):84–88

Received:
October 18, 2020

Accepted:
March 30, 2021

Abstract

Objective: To describe a case of a Cardio-Cerebral Infarction (CCI) male patient presented with a history of chest pain recognized using electrocardiography, brain computed tomography, and Percutaneous Coronary Intervention (PCI).

Methods: A 69 years old man came with history of chest pain since 13 hours before to the emergency room. Electrocardiography, brain computed tomography, and PCI were performed, leading to the diagnosis of CCI.

Results: The electrocardiography showed ST Elevation in Antero-lateral, atrial fibrillation and left-sided hemipharesis, which occurred on the second day. Brain computed tomography demonstrated acute infarct stroke, while the Percutaneous Coronary Intervention (PCI) showed one vessel disease with severe stenosis in LAD and implanted stent in proximal-mid LAD. Therapy prescribed was providing antiplatelet and anticoagulation.

Discussion: Acute Myocardial Infarction (AMI) and Acute Infarct Stroke (AIS) have a narrow therapeutic time-window and a delayed intervention may results in morbidity and death. Antiplatelet and anticoagulant used in PCI for AMI increase the risk for hemorrhagic, and AIS with thrombolytic increase the risk of cardiac wall rupture in AMI. Direct Oral Anticoagulant (DOAC) treatment should reduce ischaemia and lower bleeding. The optimal time point to start anticoagulant treatment might be between 4-14 days after the onset of stroke. Duration of post-PCI triple therapy should be minimized depending on bleeding and risks of ischemia.

Keywords: Acute infarct stroke, acute myocardial infarction, Cardio-cerebral infarction

Introduction

Cardio-Cerebral Infarction (CCI) is an rare case and used to describe the simultaneous of Acute Myocardial Infarction (AMI) and Acute Ischemic Stroke (AIS). The risk of stroke in patients after acute myocardial infarction (AMI) is significantly higher in comparison with the general population. The incidence of an ischemic stroke within a month after a ST Elevation Myocardial Infarction (STEMI) has found to be 2 % and up to 1 % within 90 days after Non ST-Elevation Acute Coronary Syndrome (NSTE-ACS). [1,2]

Stroke occurs in the early postinfarction compared with occur late higher mortality. A delayed intervention, may result in permanent irreversible morbidity and mortality. Deciding when to initiate Oral Anticoagulant (OAC) in patients with non-valvular Atrial Fibrillation is a longstanding, common, and unresolved clinical challenge. Although the risk of early recurrent ischaemic stroke is high in this population, early oral anticoagulation is suspected to increase the risk of potentially harmful intracranial haemorrhage, including

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haemorrhagic transformation of the infarct.[3,4] This condition remains a challenge even today and can lead to potentially devastating complications.

Case

A 69 years old man came to the hospital with history of typical angina chest pain at rest since 13 hours before admission. Patient had history of dyspnoe on effort since 1 year, paroxysmal nocturnal dyspnoe since 6 month, and leg edema since 6 month before admission. Patient had risk factors of smoking, hypertension and stroke known since 4 years ago. Previous electrocardiography (ECG) onset 5 hours before admission showed *STEMI anterolateral.*

Physical examination showed he was compos mentis with blood pressure 100/60 mmHg, heart rate 102 beats per minute, respiratory rate 22 times per minute, temperature 36.7^0 C, oxygen saturation 98 % with canule nasal 3 liter per minute. Jugular vein pressure was 5+2 cmH20. Heart examination showed cardiomegaly without murmur. He had focal neurological deficits with left hemipharesis. National Institues of Health Stroke Scale (NIHSS) was 25. Electrocardiography showed sinus tachycardia, old myocardial infarction anterolateral, *ST Elevation Anterolateral, without evolution.* Laboratory examination showed haemoglobin 12.5 g/dL, hematocrite 47.8 g/dL, leukocyte 18.060 g/dL, thrombocyte 387.000 g/dL, ureum 40 mg/ dL, and creatinine 0.98 mg/dL. Troponin 1.35 mg/dL

On the first day of treatment the monitor showed paroxysmal atrial fibrillation (AF) and the second day of treatment the patient complained sinistra hemiplegia. Brain Computed Tomography Scan showed infarct in cortical, subcortical lobus temporo-occipital dextra, multiple infarct lacunar in basal ganglia bilateral and cerebri senillis atrophy.

Patient underwent percutaneous coronary intervention (PCI) after being approved by the neurology department on sixth day of treatment and the examination showed right coronary system dominance with severe stenosis in Left Anterior Descending (LAD) at proximal-mid portion and total occlusion in mid portion. Left Circumflex (LCx) artery has mild stenosis at proximal portion and severe stenosis in obtuse marginal 1. Right Coronary Artery (RCA) shows mild stenosis in mid portion. Patient underwent coronary stenting in LAD with 2 Drug Eluting Stents (DES) are 3.0 x 26mm and 2.75 x 38 mm deployed overlappingly. During procedure the patient is asymptomatic with stable vital signs.

Observation during this period shows normal vital sign. Our evaluation using ECG doesn't show any changes in anterolateral leads. The patient was discharged after 8 days of treatment in hospital without any symptoms. During hospitalization patient gets therapy was Aspirin 81 mg once daily (OD), clopidogrel 75 mg (OD), Warfarin 2 mg OD, Carvedilol 6.25 mg twice daily, Ramipril 2.5 OD, Furosemide 40 mg OD, spironolactone 25 mg OD , Nitrat 2.5 mg twice daily, Atorvastatin 40 mg OD. This case report presents a 69-year-old patient presenting with typical angina chest pain who is later diagnosed with *Recent*

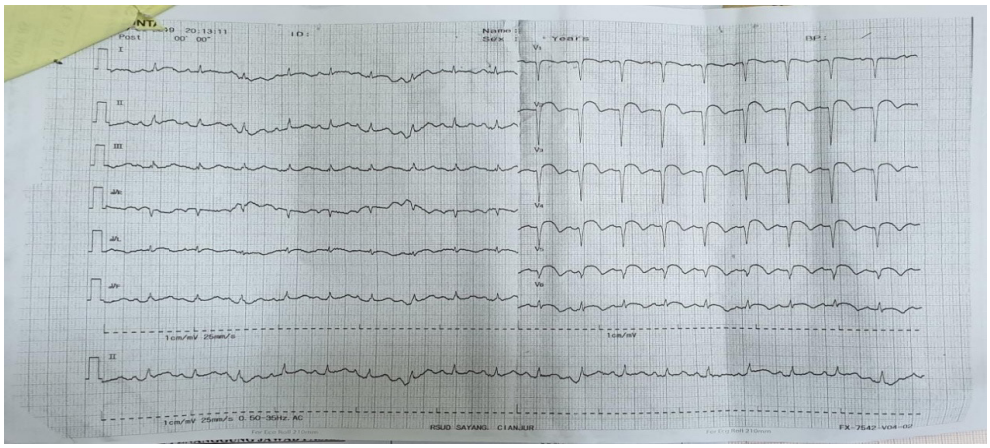

Fig. 1 Electrocardiography Showed Sinus Tachycardia, Old Myocardial Infarction Anterolateral, *St Elevation Anterolateral*

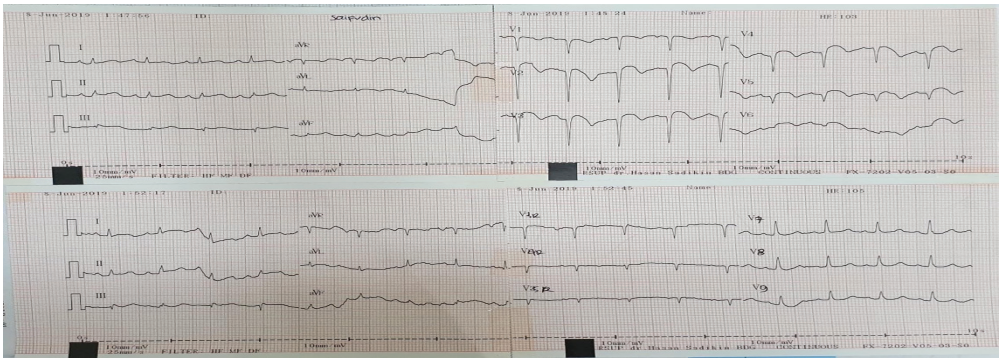

Fig. 2 Electrocardiography Showed Sinus Tachycardia, Old Myocardial Infarction Anterolateral, Evolution (-)

STEMI Anterolateral and in hospital treatment the patient complains of left hemiparesis who is later diagnosed with ischemic stroke.

Discussion

A 69 years old man came with typical angina chest pain and history of left hemipharesis on second day treatment was diagnosed as STEMI Anterolateral and infarct Stroke. Patient has electrocardiography showed Sinus tachycardia, old myocardial infarction, *ST Elevation Anterolateral.* Brain CT demonstrated acute infarct stroke. Angiography showed showed right coronary system dominance with severe stenosis in Left Anterior Descending (LAD) at proximal-mid portion and total occlusion in mid portion.

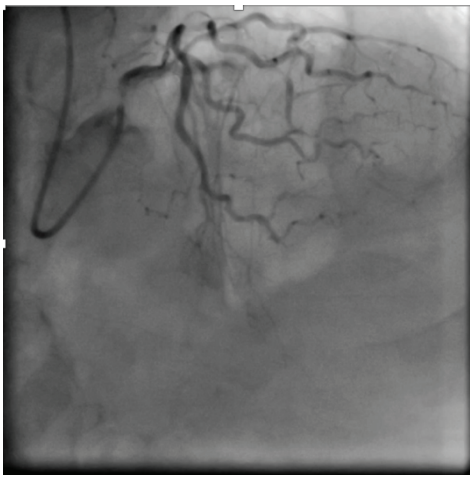

Fig. 3 Angiography Showed Severe Stenosis in Left Anterior Descending (LAD) at Proximal-Mid Portion and Total Occlusion In Mid Portion

Left Circumflex (LCx) artery has mild stenosis at proximal portion and severe stenosis in obtuse marginal 1. Right Coronary Artery (RCA) shows mild stenosis in mid portion. Patient underwent coronary stenting in LAD with 2 Drug Eluting Stents (DES) are 3.0x26 mm and 2.75x 38 mm deployed overlappingly.

Cardio-cerebral infarction (CCI) is used to describe the simultaneous occurrence of Acute ischemic stroke (AIS) and Acute Myocardial Infarction (AMI). AIS and AIS that coincidence usually referred CCI type 1. The risk of stroke in patients after AMI is significantly higher in comparison with the general population. The incidence of stroke among AMI survivors can approach 1.2% during a 1-month observation, whereas long-term observational data show that more than 2% have stroke within 1 year. One of the cardiac condition coincidence AIS and AMI in this patient is paroxysmal atrial fibrillation suggested caused dislodged LV thrombus. Although the result of echocardiography was no found thrombus, but there's decrease LV systolic function (Biplane's 35%). Stroke following a myocardial infarction remains a challenge even today and can lead to potentially devastating complications. Furthermore, strokes occurring in the early postinfarction period are associated with excessive mortality compared with those that occur late.[1,2]

The management of a simultaneous presentation of both stroke and AMI is unclear. A delayed intervention of one infarcted territory for the other may result in permanent irreversible morbidity or disability, and even death. Both conditions have a narrow therapeutic time-window, such that acute management of one at the expense of the other may result in permanent irreversible disability from the infarcted area that received

delayed intervention. In addition, the use of antiplatelet and anticoagulants that are inherently part of a percutaneous coronary intervention (PCI) for AMI may increase the risk for hemorrhagic conversion associated with intravenous thrombolysis and the use of a thrombolytic in AIS increases the risk of cardiac wall rupture in the setting of AMI.[3,4]

There are no clinical trials that have addressed this dilemma likely due to its rarity, and there are also no evidenced-based societal guidelines on the sequence of approach to management. The ideal management of simultaneous CCI is a treatment strategy that benefits both vascular territories. An important deciding factor in approach to management is the presentation of AMI. Simultaneous CCI with STEMI poses the greatest management challenge, and the management options suggested in present article will be more beneficial for simultaneous presentation of AIS and STEMI. Intravenous thrombolysis, approved for the acute management of both conditions has been suggested as the best approach to the treatment of simultaneous CCI if there is no contraindication, and both presentations are within the time. A combined endovascular approach with the use of Percutaneus Transluminal Coronary Angiplasty for AMI and thrombectomy devices for AIS have been suggested.The statement from the American Heart Association/ American Stroke Association on the scientific rationale for the inclusion and exclusion criteria for IV-tPA in AIS recommended treatment with IV-tPA at the dose appropriate for AIS, followed by PTCA and stenting if indicated (*Class IIa*, *Level of Evidence C*), based on the fact that pretreatment with IV-tPA does not decrease the coronary benefit of PTCA and stenting.[5,7]

In patients with atrial fibrillation and a recent ischaemic stroke (who are at high risk for both recurrent ischaemia, DOAC treatment which should reduce ischaemia and has a lower bleeding risk than VKAs is a promising strategy. However, individual net clinical benefit will vary according to the absolute risk of these events occurring in such patients, since the risk might depending on the timing of treatment (with early treatment being likely to reduce the risk of ischaemic stroke but potentially increasing the risk of intracranial haemorrhage). The AHA/ASA guidelines recommend that starting oral anticoagulation within 4–14 days after ischaemic stroke onset is reasonable for most patients. Moreover, early (2–3 days after stroke onset) initiation of VKA administration was associated with fewer recurrent ischaemic strokes than late (>3 days after stroke onset) initiation of VKA treatment in the VISTA prospective cohort study of 1644 patients with ischaemic stroke and atrial fibrillation.[8,9]

PRECISE-DAPT score for predicting bleeding events in patients with acute coronary syndrome undergoing percutaneous coronary intervention (PC) was 3 (low criteria) Patients with an indication for oral anticoagulation undergoing PCI should concern about ischaemic risk and bleeding risk prevailing. Follow-up this patient was triple therapy with OAC, clopidogrel, and aspirin 80 were continued for 1 month after BMS placement and for 1 year after DES placement. Duration of triple therapy should be minimized depending on bleeding and ischaemic risks.[10,11]

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