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A Rare Case Of Cardio-Cerebral Infarction Syndrome

Dr. P. Ragavi[1], Dr. A. K . Badrinath[2]
[1]Post Graduate, [2]Professor And Unit Chief,
Department Of General Medicine,
Sri Manakula Vinayagar Medical College and Hospital

***Corresponding Author:**
Dr. P. Ragavi
Post Graduate, Department Of General Medicine,
Sri Manakula Vinayagar Medical College and Hospital

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Abstract

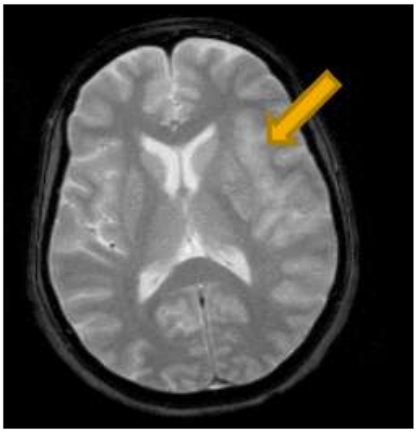
We report an extremely rare case of Cardio-cerebral infarction syndrome. Acute ischemic stroke and coronary artery disease are the major causes of death in the worldwide.(1) The prevalence of coronary artery disease has been reported in one fifth of stroke patients. Although high incidence rate of acute myocardial infarction after recent ischemic stroke and the high risk of acute ischemic stroke after recent myocardial infarction has been reported in several clinical and observational studies. So that acute or recent problem in the heart or brain that could result in an acute infarction of the other. Here we discussing a case of Cardio-cerebral infarction syndrome Type I in a 45 year old female patient.

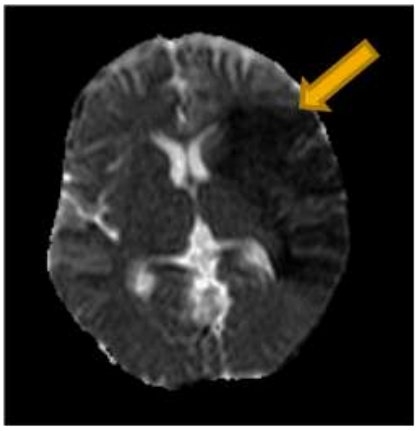
Keywords: Acute Myocardial infarction, acute ischemic stroke

Introduction

45 year old female, who is a known diabetic, presented to ER with chest pain for 3 days, radiating to left upper limb and shoulder back, associated with palpitation, sweating and breathlessness on exertion for 3 days. On clinical examination patient was vitally stable and systemic examination found to be normal. ECG showed ST elevation in V2-V4, ST depression in lead II, III, aVF. Routine biochemical investigations were done which showed elevated CK-MB (CK-MB – 72 IU/L), Troponin –I – Positive and all other parameters within normal limits. Patient was treated with loading dose, thrombolysed with INJ.STREPTOKINASE @ 5 PM and other supportive measures. Follow up serial ECG monitoring was done which showed > 50 % reduction in height of ST segment elevation in V2-V4. Patient developed weakness of right upper limb & lower limb with deviation of angle of mouth towards left side @ 3 AM. On CNS examination GCS- E4V2M5(11/15), Right sided hemiplegia with right UMN facial palsy. Optic fundus showed no papilledema. MRI Brain showed Hyperintensity in left caudate nucleus, lentiform nucleus, parietal lobe, frontal lobe, insular cortex and temporal lobe – Suggestive of Acute left MCA territory infarct with no hemorrhagic transformation, MR Angiogram showed Complete left MCA occlusion. 2D ECHO showed study consistent with CAD with Severe LV dysfunction, EF-30%. CV Doppler showed normal study. Because of the Acute ischemic stroke and coronary artery disease are the major causes of death in the world, immediate diagnosis & acute intervention is to be considered timely.

HYPERINTENSITY LESION **MRI BRAIN DWI IMAGE**





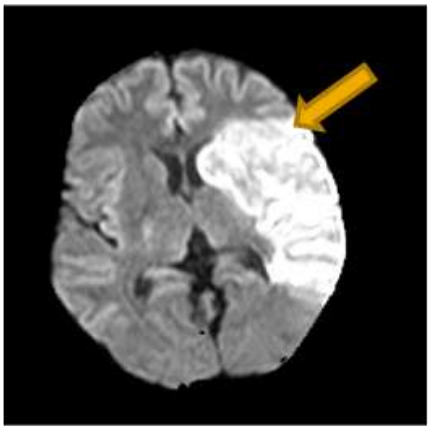
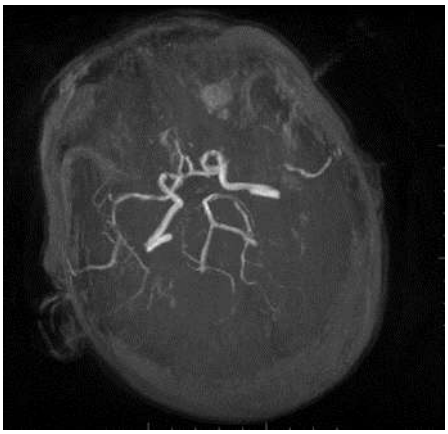


Fig 1 (Left): MRI-BRAIN - Area of Hyperintensity lesion in left caudate nucleus, lentiform nucleus, parietal lobe, frontal lobe, insular cortex, temporal lobe Suggestive of Acute left MCA territory infarct with no hemorrhagic transformation.

Fig 2 A, 2B (Right): MRI BRAIN (DWI image) showed Restricted Diffusion

Fig 3: MR ANGIOGGRAM – Complete Left MCA Occlusion



There was no involvement of Sensory system, autonomic system or cerebellar system clinically. Bowel and bladder habit was intact. Other Systemic examination was unremarkable. Routine blood investigations – complete blood count, renal function test, Liver function test, electrolytes, thyroid function test, urine routine, diabetic profile were all normal. Patient was started on anti-edema measures, statins, physiotherapy and anti-coagulants were withheld in view of massive infarct in the brain.

During the course of stay, patient GCS was not improved and repeat CT Brain was done which showed Left acute MCA territory infarct with no hemorrhagic transformation, causing midline shift of 9mm to the right with subfalcine, left uncal and descending transtentorial herniation. Patient was planned for Emergency craniectomy.

Volume 6, Issue 6; November-December 2023; Page No 75-80

Ct Brain

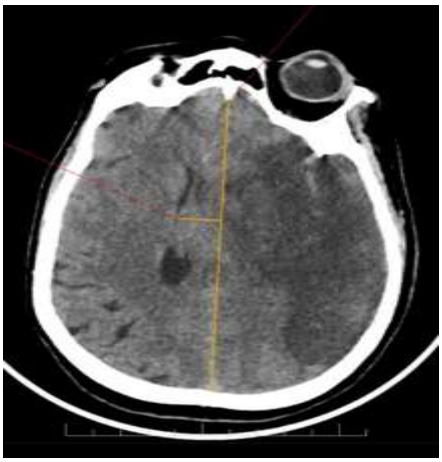
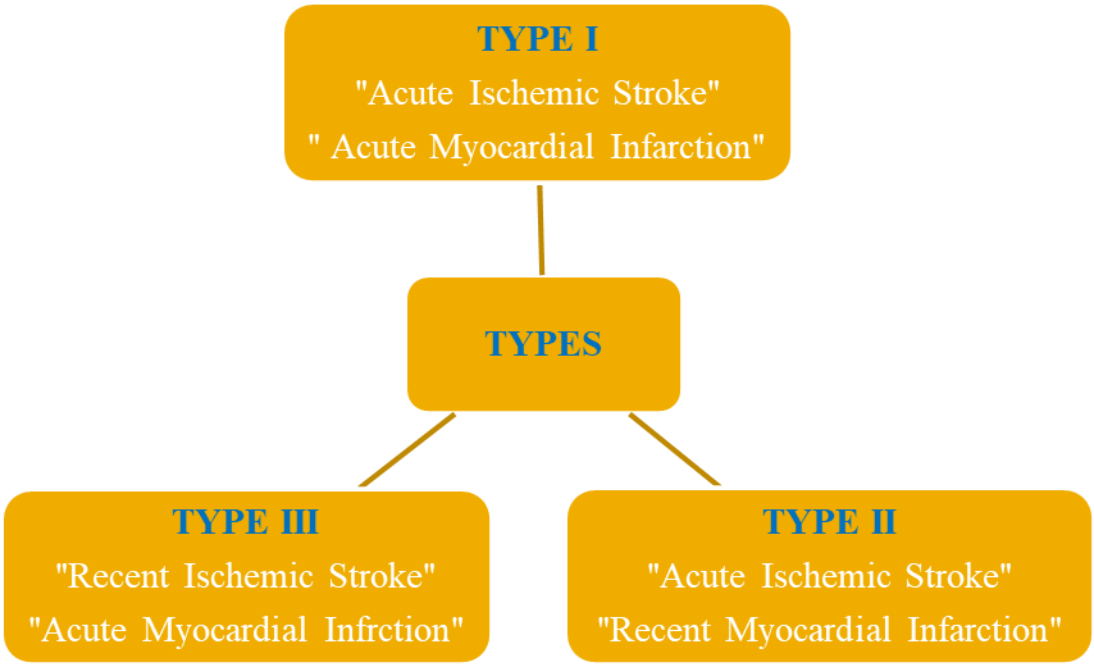


Fig 4: Left MCA infarct with midline shift (9 mm) to right with subfalcine, left uncal & descending transtentorial herniation

Types Of Cardio-Cerebral Infarction



Type I : Concurrent cardio-cerebral infarction syndrome

Acute myocardial infarction (< 12 hours) with acute ischemic stroke (<4.5 hours)

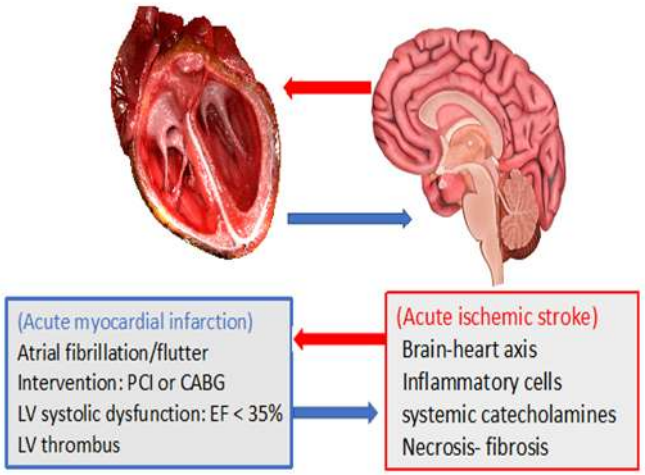
3 Subtypes:

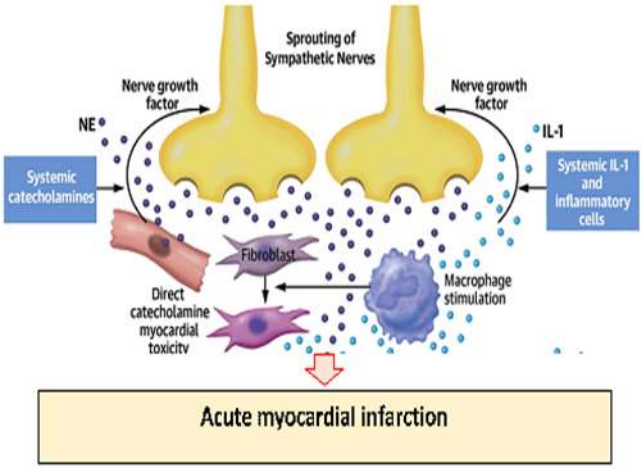
Type IA : Cardiac causes

Type IB : Brain causes

Volume 6, Issue 6; November-December 2023; Page No 75-80

Type IC : Non-cardiac & Non-brain causes





Phathophysiology :

1) Cardiac causes – Type 1A:

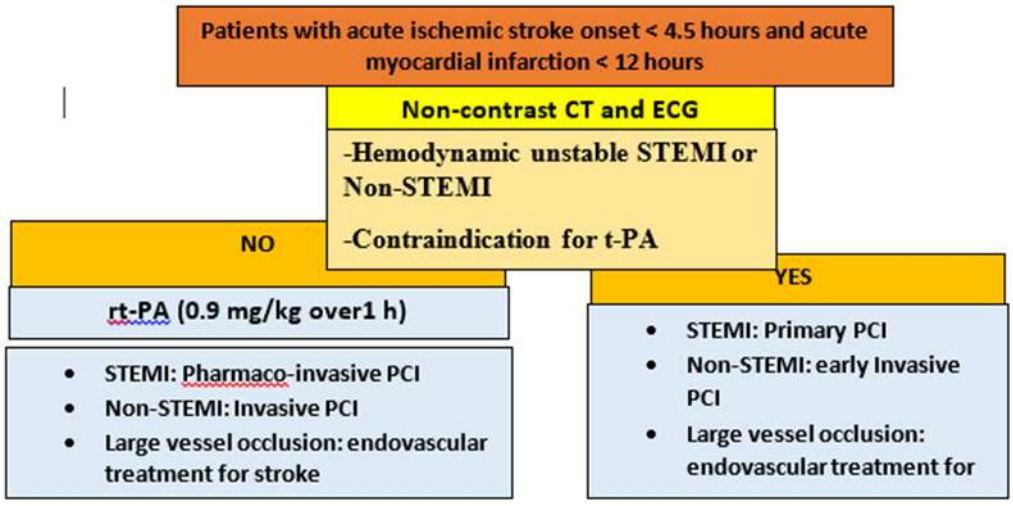
1. Atrial fibrillation – common source of both cerebral & coronary embolism[4]
2. Type I acute aortic dissection with dissection flap extending to coronary & Common carotid origin[5]
3. Electrical injury – Concurrent cerebral & coronary vasospasm[6]
4. Pre-existing intra-cardiac thrombus from poor LV ejection fraction lead to simultaneous coronary & vascular occlusion[7]
5. Thrombus formed in the right ventricle in acute right ventricular infarction with right ventricular failure in combination with patent foramen ovale can embolize to both vascular territories
6. Severe hypotension following Acute MI leads to hemodynamic stroke[8]
7. Prosthetic valve thrombosis
8. Intracardiac masses (Myxoma, papillary fibroelastoma)
9. Infective endocarditis

2) Brain causes (Brain-heart axis) – Type 1B: Brain-heart axis dysregulation is an alternative pathophysiology of simultaneous cardio-cerebral infarction syndrome. Insular cortex plays a critical role in central autonomic system regulation. AIS in perietoinsular region leads to have higher risk of developing arrhythmias such as Atrial fibrillation.[9] Cardiac sympathetic overactivity from an insular cortex lesion can provoke diffuse myocardial damage "myocytolysis", which leads to elevation of cardiac enzymes.[10]

3) Non cardiac & non-brain causes – Type 1C: Recent studies suggested that Coronavirus disease 2019 (COVID-19) infection can be increased the risk of AIS & AMI. The evidence that COVID-19 may increase the risk of acute ischemic cardiovascular events, the underlying mechanisms may cytokine mediated hypercoagulability and plaque destabilization.[11] Severe hypotension can be causes infarction in brain and myocardial infarction.

Volume 6, Issue 6; November-December 2023; Page No 75-80

Treatment:



Anti-platelet & Anti-coagulant treatment

(Triple 1 week ; Dual 6 months ; Single OAC or Aspirin life long)

TRIPLE 1 week

Aspirin(75-100mg) + Clopidogrel(75mg) + OAC (Rivaroxaban 2.5 mg twice or Warfarin : INR 2-3 & TTR > 70%

DUAL (N)OAC + Antiplatelets (1week – 6 months)

AF : Clopidogrel (75mg)+ OAC (Rivaroxaban 15mg OD (GFR <60: 10mg) or Warfarin INR 2-3 & TTR > 70%

LVT : First 3 months - Clopidogrel (75mg)+ OAC (Rivaroxaban 15mg OD (GFR <60: 10mg) or Warfarin INR 2-3 & TTR > 70%. Last 3 months : Aspirin(75-100mg) + Clopidogrel(75mg)

SINGLE AFTER 6 MONTHS : (N) OAC OR Anti-platelet

AF : Rivaroxaban 20mg OD (GFR <60: 15mg) or Warfarin INR 2-3 & TTR > 70%

LVT : Aspirin 100mg once daily

Discussion:

DEFINITION: CCIS is a primary disorders (infarction or its complications) of 1 of these 2 organs (Heart or Brain) often result in secondary infarction/injury to the other or to both organs.(2) The incidence of acute ischemic stroke (AIS) after recent myocardial infarction (MI) during hospital stay ranges from 0.7% to 2.2%, usually occurred in the first days after Acute MI. Brandi Witt et al (3), suggested that during hospitalization for MI 11.1 the AIS occurred per 1000 MI compared with 12.2 at one month and 21.4 at one year. The most positive predictors of ischemic stroke after MI : Older age, hypertension, diabetes, history of previous stroke,

Volume 6, Issue 6; November-December 2023; Page No 75-80

history of anterior location MI, atrial fibrillation, heart failure. This patient fit into Type I cardio-cerebral infarction syndrome.

Conclusion:

Although uncommon, simultaneous cardio-cerebral infarction is among one of the most challenging medical emergency condition and requires timely management. Due to the rarity of the condition, the management of these patients is very challenging and there is no ideal recommendations. Balanced management should be a trade-off between early rescuing the brain or the heart.(12)

References:

1. Habib M. Cardio-cerebral infarction syndrome: definition, diagnosis, pathophysiology, and treatment. Journal of Integrative Cardiology. 2021;7:1-6.
2. Al Suwaidi J, Al Habib K, Asaad N, et al. Immediate and one-year outcome of patients presenting with Acute Coronary Syndrome complicated by stroke: Findings from the 2ndGulf Registry of Acute Coronary Events (Gulf RACE-2). BMC Cardiovascular Disorders. 2012 Dec;12(1):1-9.
3. Witt BJ, Ballman KV, Brown Jr RD, et al. The incidence of stroke after myocardial infarction: a meta-analysis. The American journal of medicine. 2006 Apr 1;119(4):354-e1.
4. Tokuda K, Shindo S, Yamada K, et al. Acute embolic cerebral infarction and coronary artery embolism in a patient with atrial fibrillation caused by similar thrombi. Journal of Stroke and Cerebrovascular Diseases. 2016 Jul 1;25(7):1797-9.
5. Le Nguyen T, Rajaratnam R. Dissecting out the cause: a case of concurrent acute myocardial infarction and stroke. Case Reports. 2011 Jun 3;2011:bcr0220113824.
6. Verma GC, Jain G, Wahid A, et al. Acute ischaemic stroke and acute myocardial infarction occurring together in domestic low-voltage (220-240V) electrical injury: a rare complication. J Assoc Physicians India. 2014 Jul 1;62(7):620-3.
7. Yeo LL, Andersson T, Yee KW, et al. Synchronous cardiocerebral infarction in the era of endovascular therapy: which to treat first?. Journal of Thrombosis and Thrombolysis. 2017 Jul;44:104-11.
8. Omar HR, Fathy A, Rashad R, et al. Concomitant acute right ventricular infarction and ischemic cerebrovascular stroke; possible explanations. International archives of medicine. 2010 Dec;3(1):1-4.
9. Vingerhoets F, Bogousslavsky J, Regli F, et al. Atrial fibrillation after acute stroke. Stroke. 1993 Jan;24(1):26-30.
10. Cheshire WP, Saper CB. The insular cortex and cardiac response to stroke. Neurology. 2006 May 9;66(9):1296-7.
11. Modin D, Claggett B, Sindet-Pedersen C, et al. Acute COVID-19 and the incidence of ischemic stroke and acute myocardial infarction. Circulation. 2020 Nov 24;142(21):2080-2.
12. Kijpaisalratana N, Chutinet A, Suwanwela NC. Hyperacute simultaneous cardiocerebral infarction: rescuing the brain or the heart first?. Frontiers in neurology. 2017 Dec 7;8:664.

Volume 6, Issue 6; November-December 2023; Page No 75-80