Myocardial Infarction in Non-obstructive Coronary Arteries (MINOCA) in the Perioperative Period can Epinephrine be Responsible?

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Citation

Koirala M, Shakya BM, Parajuli B, Ghimire B. Myocardial Infarction in Non-obstructive Coronary Arteries (MINOCA) in the Perioperative Period can Epinephrine be Responsible? *Kathmandu Univ Med J.* 2022;80(4):535-7.

ABSTRACT

Ephedrine, metaraminol, epinephrine and maneuvers like carotid sinus stimulation used during intraoperative period have been postulated to cause temporary spasm of the coronary vessels leading to decrease supply to the myocardium and precipitating myocardial infraction in non-obstructive coronary arteries (MINOCA). As an anaesthesiologists, we should be aware that even a dose as small as 25 mcg epinephrine infiltrated along with local anaesthetic in the subcutaneous plane may be responsible for coronary vessel spasm and thus myocardial infraction in non-obstructive coronary arteries.

We report a case of 45 years old female with papillary carcinoma of thyroid who developed features of non-ST elevation myocardial infarction 5 minutes after the subcutaneous infiltration of 5 ml of 2% Xylocaine with 1:200000 Epinephrine. Patient was managed for acute Myocardial Infarction. Coronary angiogram done the next day revealed normal coronary arteries, hence the diagnosis Myocardial infraction in non-obstructive coronary arteries was made.

KEY WORDS

Acute myocardial infraction, Epinephrine, Local anaesthetics

INTRODUCTION

The significant occlusion of coronary arteries is the commonest cause of myocardial ischemia and subsequent infraction. However, there are instances in which infarction has occurred with apparently normal coronary arteries. Myocardial infraction in non-obstructive coronary arteries (MINOCA) is a term used to define the patients with features of myocardial infarction without the presence of obstructed coronaries (≥ 50% stenosis) in the angiogram. MINOCA can also occur during the perioperative period with drug induced coronary artery spasm being responsible for most of the cases.

CASE REPORT

A 45 years old, 50 kgs female presented with progressively increasing swelling in the middle of the neck for one year. There was no history of shortness of breath, orthopnea, palpitation. The physical examination including airway were within normal limit. Ultrasound of neck confirmed 4.9 x 3.1 cm mixed micro-calcified and cystic mass involving the right lobe and isthmus of thyroid with normal vessels. Fine needle aspiration cytology of the same proved it to be papillary carcinoma of thyroid for which total thyroidectomy with neck dissection was planned by the ENT team. All the investigations were within normal limit

including thyroid function test. The case was planned under general anaesthesia with endotracheal intubation. Her pre induction vitals were, heart rate of 76/min and blood pressure of 110/80 mm Hg. She was induced with Intravenous Fentanyl 120 mcg, Midazolam 1 mg and titrated dose of propofol (80 mg) and Vecuronium 6 mg. Then intubated with 7 mm id cuffed flexometallic endotracheal tube. Immediately after intubation, heart rate and blood pressure increased to 90 bpm and 150/90 mm Hg respectively. Then positive pressure ventilation was instituted with a tidal volume of 400 ml and a rate of 12/min and isoflurane of 1.5% with 70% oxygen. After 5 mins the heart rate settled to 76/min and BP to 110/80 mm Hg. Then the patient was handed over to the surgeon who injected 5 ml of 2% Xylocaine with 1:200000 Adrenaline in the subcutaneous plane above platysma. After another 5 minutes, when draping was being done, frequent VPCs started appearing in the monitor and blood pressure also rose to 160/90 mm Hg. Inj. Xylocard 80 mg was given which controlled the VPCs but the blood pressure was persistently high (180-200/ 130-100 mm Hg) and the ECG showed ST depression in lead 2. Inj. Glyceryl trinitrate 100 mcg bolus doses were given multiple times and 12 lead ECG was also done which verified ST depression in V5 and V6 lead (Fig.1). In the background of new ECG changes, we postponed the surgery and extubated the patient keeping heart rate and blood pressure under control with use of Esmolol and Nitroglycerin as necessary. Post extubation BP and heart rate was 130/80 mm Hg and 90/min respectively. After regaining full consciousness, patient complained of slight heaviness in chest. In the post-operative recovery room, cardiac consultation was done and screening echo revealed global hypokinesia of left ventricle (more on LAD territory), mild LVSD (EF = 50%). Inj. Heparin 40 mg sc, Aspirin 150 mg and Atorvastatin 20 mg per oral were given. The patient was managed for acute non-ST segment elevation Myocardial infarction in the cardiac care unit. The cardiac enzymes sent after 6 hrs. had slightly raised Trop I of 0.497 ng/ml and CPK MB of 36 U/l. Coronary angiogram done on the next day revealed normal coronary arteries after which a diagnosis of MINOCA was made (Fig. 2).

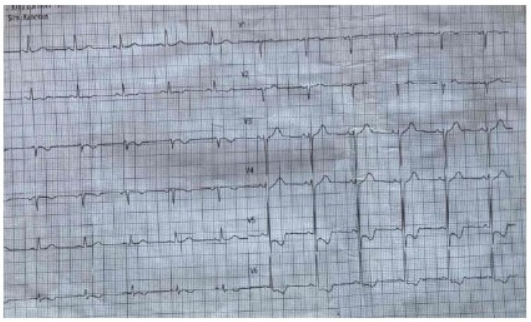

Figure 1. **ECG of the patient showing T inversion in lead V5 and V6**

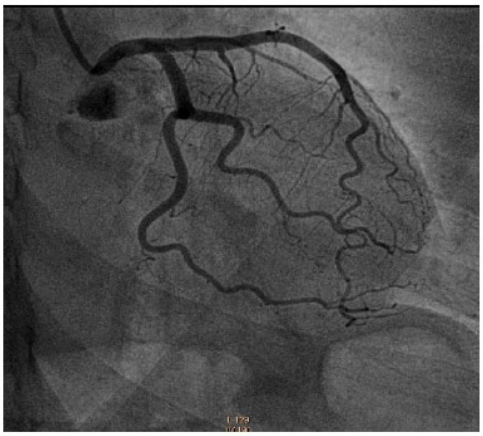

Figure 2. **Normal coronary angiogram of patient**

DISCUSSION

Pashupaty et al. in a systemic review found out that MINOCA accounted for 6 % of patients diagnosed as Acute Myocardial Infarction.[1] Safdarafdar et al. in the VIRGO Study reported that the mortality rates at 1 month and 1 year were not statistically different in MINOCA and Acute MI due to coronary artery disease.[2] This suggests that MINOCA should not be ignored and once diagnosed, treatment should be started aggressively and patient should be followed up. One of the commonest causes of MINOCA is coronary artery spasm.

Coronary artery spasm has been defined as sudden, transient, focal spasm of the endothelium of the normal coronary arteries which is likely to be induced by super sensitivity of the endothelium to various stimuli, including stimulation of carotid sinus or increased catecholamine. Perioperative coronary artery spasm has been reported to occur both during general and regional anaesthesia. Nishizaki et al. reported a case of coronary artery spasm in a conscious patient without coronary stenosis induced by carotid sinus massage which was confirmed by angiography.[3] Choi et al. reported that carotid sinus stimulation during neck surgery under general anesthesia induced coronary artery spasm in a patient without a history of coronary artery disease.[4]

Drugs such as ephedrine, metaraminol, epinephrine have been reported to cause coronary artery spasm because of their alpha-adrenergic constrictive action on large epicardial arteries. Khavandi et al reported a case of myocardial infarction in a healthy woman, after the administration of ephedrine and metaraminol for spinal anaesthesia induced hypotension.[5]

Epinephrine in the therapeutic dose given in different routes such as intravenous, intramuscular or subcutaneous have been reported to cause coronary spasm.[6-8] In all these cases, the coronary arteries were normal in the angiography.

Lee et al. have reported a case of cardiac arrest after a very low dose of epinephrine (1.5% lidocaine containing

1: 100,000 epinephrine) injected into the nasal mucosa for septoplasty.[9] The coronaries were again normal in this case too.

In our case, 2% Xylocaine with 1:200000 Adrenaline (5 ml) was injected in the subcutaneous plane. VPCs and ST-depression started to appear about 5 mins after the local infiltration. So we have assumed that it is probably due to the absorption of epinephrine from the local site into the blood vessel and resultant alpha 1 receptor mediated coronary vascular spasm.

The diagnosis of myocardial infarction was favored by rise of Trop I in subsequent blood investigation and presence of regional motion abnormality in subsequent echocardiography which resolved after a few days. Coronary angiography done on the next day confirmed the presence of normal arteries. Hence the diagnosis of MINOCA was made.

We as an anaesthesiologists should be aware that even a small dose such as 25 mcg epinephrine infiltrated along the local anaesthetic in the subcutaneous plane may be responsible for coronary vessel spasm and therefore must be vigilant during that period. Once the diagnosis is made, treatment should be started because though the coronaries are normal in the angiography the mortality rates are not different than the patients with AMI with obstructive coronaries.

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