DOI: http://dx.doi.org/10.25239/SHJ/Vol6/No1/CaseReport2

Case Report

Two Consecutive Acute Myocardial Infarctions in a Thrombolysed patient

Author(s): Dr. Mohamed A Abdelmalik^, Dr. Nosyba M Hussein*, Hamoda A Ali**

*Ahmed Gasim Hospital, Khartoum, Sudan
^Corresponding Author, Mo.malikomer3@gmail.com

Abstract

A second episode of ST segment elevation myocardial infarction (STEMI) in a patient who had been thrombolysed for a first infarct in less than 48 hours is rare. We report a case of acute inferior STEMI in a 70-year-old patient who was thrombolysed with Streptokinase 32 hours earlier for anterior STEMI.

Introduction:

STEMI is usually caused by rupture of a vulnerable plaque and involves a single coronary artery, in spite of the presence of multiple complex plaques lesions. Many intrinsic and extrinsic factors can precipitate fibrous cap weakness and finally result in plaque rupture of non-culprit lesions (1). Furthermore, the occurrence of a second Myocardial Infarction (MI) after receiving antiplatelet and fibrinolytic therapy is extremely rare. We report, herein, a case of consecutive anterior and inferior MI in the same patient, occurring within less than 48 hrs.

Case presentation:

A 70-year-old male, with past history of hypertension on treatment, developed sudden chest pain at rest. On physical examination, his pulse was 69 beats/min, blood pressure 107/70 mmHg. Initial (ECG) showed ST elevation in leads V2, V3, V4, V5, V6, (Figure 1).

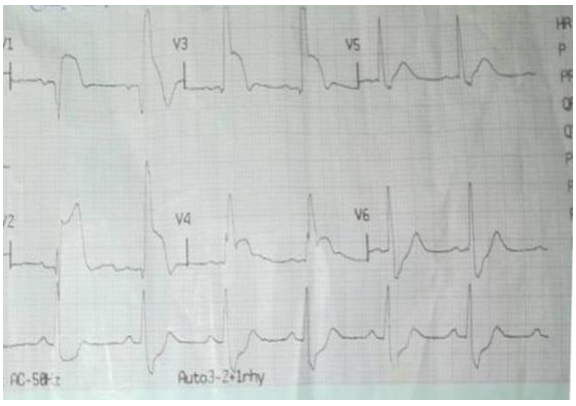

Figure 1. ST elevation in precordial leads

DOI: http://dx.doi.org/10.25239/SHJ/Vol6/No1/CaseReport2

Loading doses of Aspirin and Clopidogrel, in addition to intravenous Streptokinase were administered within 30 minutes of the onset of chest pain. 18 hours later, he was transferred to a tertiary cardiac center. Laboratory testing revealed an elevated Creatinine at 2.8mg/d which delayed cardiac catheterization. 32hrs from symptom onset, he developed further chest pain and cardiogenic shock ensued. ECG showed inferior STEMI complicated with complete heart block. Intubation and mechanical ventilation were performed, followed by cardiac catheterization (Figure 2). It revealed subtotal occlusion of the proximal Left Anterior Descending (LAD) Artery, and total occlusion of the proximal Right Coronary Artery (RCA). Percutaneous coronary intervention (PCI) to both vessels was performed. Direct stenting with a 3.0x28 Drug Eluting stent was performed in the proximal LAD. Pre-dilatation followed by stenting with a 2.75x28mm Drug Eluting stent was performed in the proximal RCA. There was TIMI 3 flow in both vessels at the end of the procedure. After PCI, there was a dramatic hemodynamic response with resolution of complete heart block, normalization of BP and appearance of an idioventricular rhythm on the monitor.

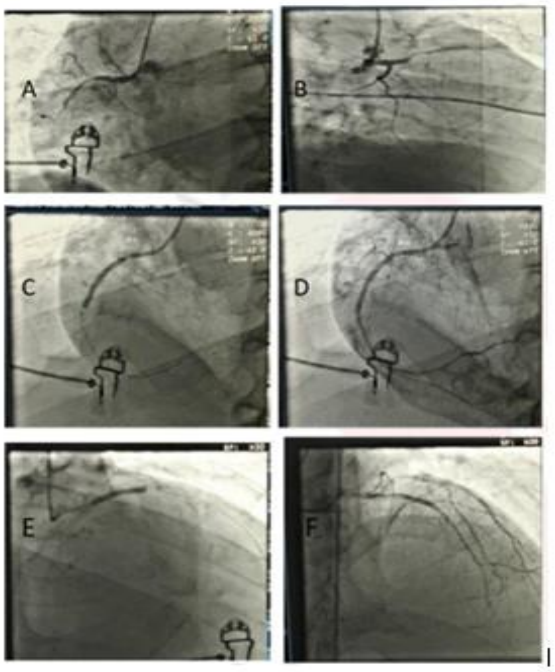

Figure 2
A. totally occluded proximal RCA,
B. sub-totally occluded proximal LAD,
C. Stent deployment in RCA,
D. Final result of PCI to RCA,
E. Stent deployment in LAD,
F. Final result of PCI to proximal LAD.

Post procedure, the patient returned to the Cardiac Care Unit. Unfortunately he passed away six hours later, due to severe metabolic acidosis, pH 6.8.

Discussion:

Presence of thrombosis in multiple arteries is rare in patients who undergo primary PCI (1-3); this may be due to sudden cardiac death as the initial presentation precluding the performance

DOI: http://dx.doi.org/10.25239/SHJ/Vol6/No1/CaseReport2

of coronary angiography and PCI. This is supported by autopsy data; which demonstrated that more than 50% of patients who died from acute myocardial infarction had thrombosis in more than 1 major epicardial coronary arteries. Our patient is unique in that two arteries were clinically and angiographically documented to be acutely thrombosed in a consecutive manner. Moreover, the second MI occurred after receiving loading doses of dual antiplatelet therapy and streptokinase. This supports the fact that plaques in a non-culprit coronary artery can rapidly progress and lead to total occlusion of another territory within a few hours after the first STEMI episode. Chang et al. reported a case of consecutive STEMI recurring in a different coronary artery within one day. Additionally, evaluation by IVUS of the 3-vessels for the incidence and predictors of single and multiple plaques rupture in Acute MI and stable angina, reported by Hong et al, found plaque rupture of non-infarct-related artery in 17% of patients with acute MI (4). Many intrinsic and extrinsic factors can precipitate fibrous cap weakness and results in plaque rupture. Those extrinsic factors may include conditions such as progression of inflammatory process related to disseminated intravascular coagulopathy, intraluminal mechanical forces modulated by sympathetic tone and catecholamines. In our case, we postulate that the likely mechanism of rapid coronary thrombosis in non-culprit vessel was progressive metabolic acidosis due to ongoing cardiogenic shock.

Conclusion:

Consecutive STEMI in the same patient within a short duration can have severe complications

References:

1. Davies MJ, Thomas A. Thrombosis and acute coronary-artery lesions in sudden cardiac ischemic death. N Engl J Med. 1984 May 3;310(18):1137-40.
2. Chang CT, Lu YH, Chu CY, Sheu SH, Hsu PC. Two Consecutive Episodes of Acute Myocardial Infarction Occurring in Different Coronary Arteries of a Single Patient with Sepsis. Acta Cardiol Sin. 2014 Nov;30(6):578-81.
3. Lee SG, Lee CW, Hong MK, Kim JJ, Park SW, Park SJ. Change of multiple complex coronary plaques in patients with acute myocardial infarction: a study with coronary angiography. Am Heart J. 2004 Feb;147(2):281-6.
4. Hong MK, Mintz GS, Lee CW, et al. Comparison of coronary plaque rupture between stable angina and acute myocardial infarction: a three-vessel intravascular ultrasound study in 235 patients. Circulation 2004; 110:928-33.29.