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(RESEARCH ARTICLE)

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Double trouble in a day!

Ng Kwang How [1,*], Muhamad Ali SK Abdul Kader [2] and Gurudevan a/l Mahadevan [1]

[1] *Department of Cardiology, Hospital Sultanah Aminah, Johor, Malaysia.*
[2] *Department of Cardiology, Hospital Serdang, Selangor, Malaysia.*

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Abstract

The no-reflow phenomenon is a complication of percutaneous coronary intervention resulting in poor myocardial perfusion and is associated with significant morbidity and mortality. It is vital for cardiologist to recognise this phenomenon as it is clinically significant. We reported two cases of no reflow phenomenon occurred in the same day and discussed its predisposing factors, possible aetiology, managements and its successful outcome.

Keywords: Percutaneous coronary intervention; No-reflow phenomenon; TIMI flow; Microvascular dysfunction

1. Introduction

As the technology of coronary angiogram and angioplasty advanced, they are now widely and increasingly performed. The common indications are acute coronary syndrome, stable angina, positive screening test and anginal equivalent. The increasing percutaneous coronary intervention (PCI) rate is also associated with increased risk of procedure-related complications. One of the complications is acute no reflow-phenomenon, which is associated with poor functional and clinical outcomes, potentially causing ventricular arrhythmia, congestive heart failure, myocardial infarction or death. As the perfusion at the tissue level can be impaired even when the angiographic epicardial coronary artery appears to be patent. The no-reflow phenomenon is defined as inadequate myocardial perfusion through a given segment of the coronary circulation without angiographic evidence of mechanical obstruction [1]. Angiographic epicardial coronary flow is evaluated by referring to the Thrombolysis In Myocardial Infarction risk score (TIMI) flow grading system, TIMI frame count and myocardial blush grading [2]. Slow-reflow or no-reflow is confirmed if the TIMI flow grades is less than 3, presence of high TIMI frame count, or abnormal myocardial blush [3]. Clinical features of acute no reflow include angina, persistence or increase ST segment elevation or haemodynamic instability. We presented two cases of acute no reflow phenomenon post PCI that had occurred in a single day, and discussed some practical approaches to the no-reflow phenomenon.

2. Case presentation

The first case was a 54-year-old male smoker, with underlying hypertension without proper follow up, presented with typical angina for one day prior to admission. It was associated with hypertensive crisis. ECG revealed ST depression over lateral leads (Figure 1) and he had raised cardiac markers. Otherwise renal profile, full blood count and fasting blood sugar were unremarkable. He was diagnosed to have Non-ST Elevation Myocardial Infarction (NSTEMI) and started with anticoagulant and Dual Antiplatelet Therapy (DAPT). He was then scheduled for inpatient angiogram on day 3 of admission. Angiogram showed three vessels disease, which were 80-90% stenosis at proximal to mid Left anterior descending (LAD), 70% stenosis at mid circumflex (LCX), 50-60% stenosis at proximal and distal Right Coronary Artery (RCA) (Figure 2). Adhoc PCI to LAD was done. IV heparin 6000unit (100unit/kg) stat was given. Left coronary artery was engaged with EBU 3.0 6Fr and Runthrough intermediate wire was negotiated to the distal LAD.

* Corresponding author: Ng Kwang How

Lesion was adequately prepared with semi-compliant balloon Pantera 2.0mm X 15mm. PCI to mid LAD with Drug-eluting balloon (DEB) Pantera Lux 3.0mm X 30mm and subsequently PCI to mid-proximal LAD with Drug-eluting stent (DES) Orsiro 3.0mm X 35mm and DES Orsiro 3.5mm X 35mm were done (Figure 3). Acute no reflow phenomenon was observed immediately post PCI (Figure 4). Intracoronary (IC) Glyceryl Trinitrate (GTN) 200mcg was then given but acute no reflow persisted. Subsequently repeated IC adenosine 200mcg and IC Tirofiban loading dose were given. Haemodynamic was monitored closely when the medications were administered. The final angiogram demonstrated the improvement of TIMI 0 to TIMI 3 flow without dissection, perforation or thrombus (Figure 5).

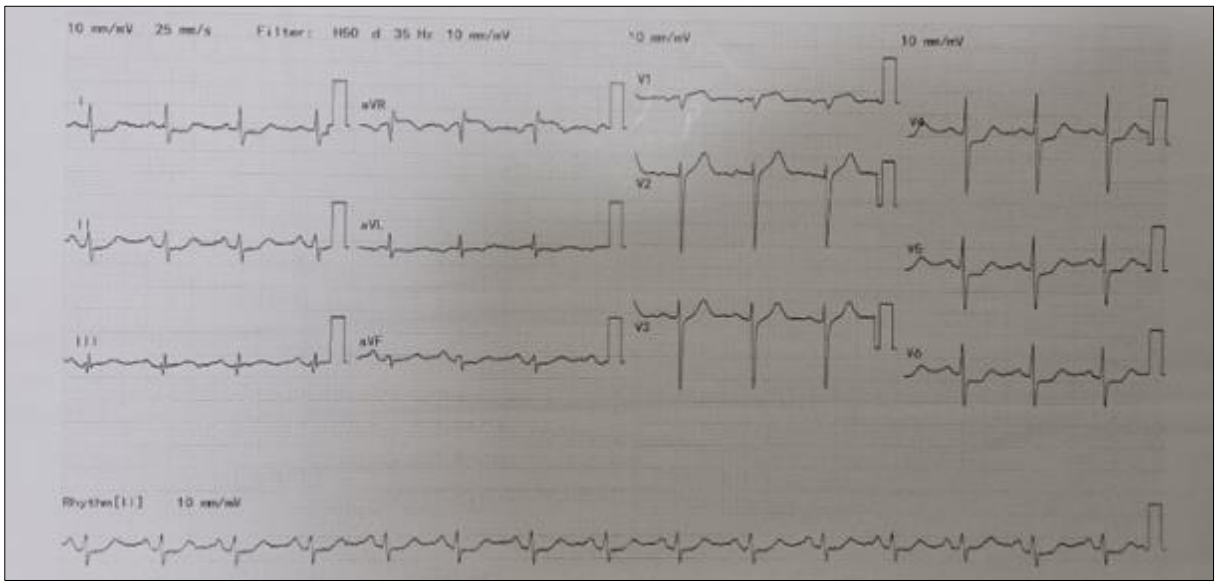
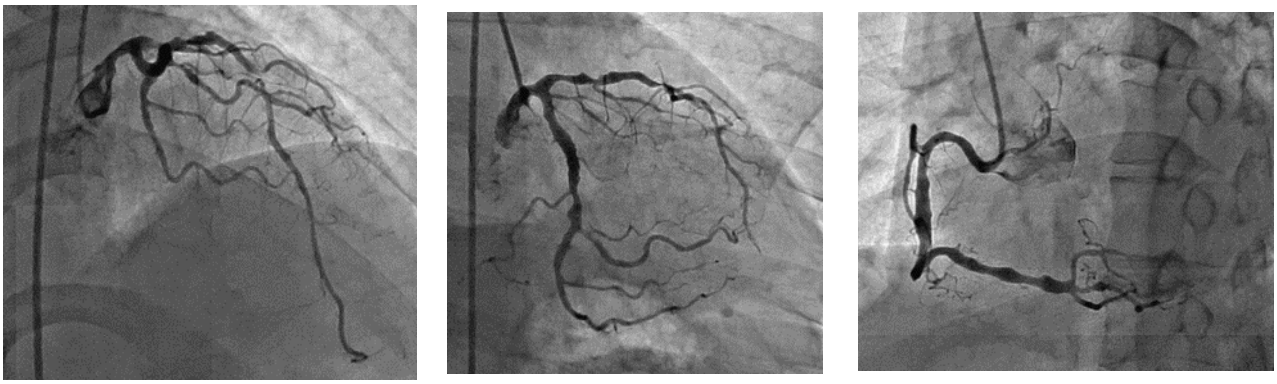
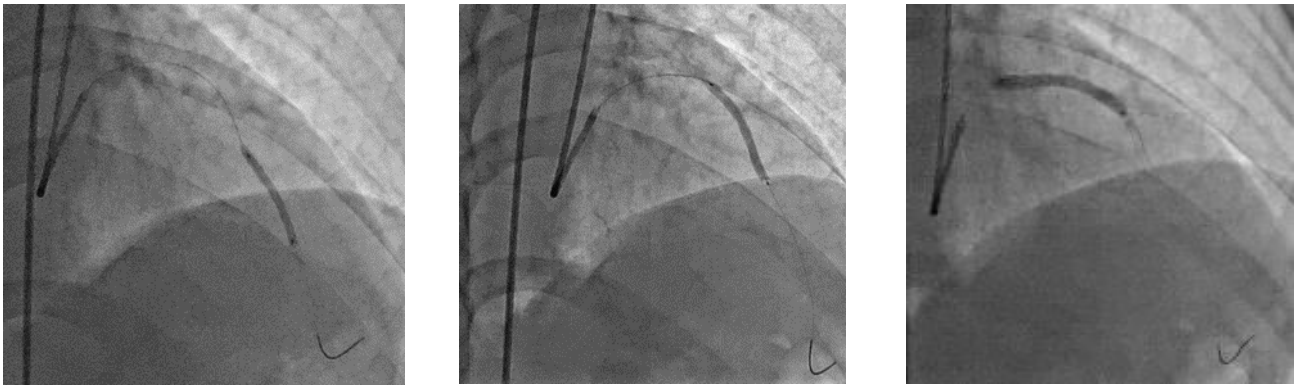


Figure 1 ECG



a) b) c)

Figure 2 Angiogram a) 80-90% stenosis at proximal to mid LAD; b) 70% stenosis at mid -distal LCX, c) 50-60% at proximal and distal RCA



a) b) c)

Figure 3 PCI to LAD; a) PCI to mid LAD with Pantera Lux 3.0mm X 30mm; b) PCI to mid-proximal LAD with Orsiro 3.0mm X 35mm; c) PCI to proximal-ostial LAD with Orsiro 3.5mm X 35mm

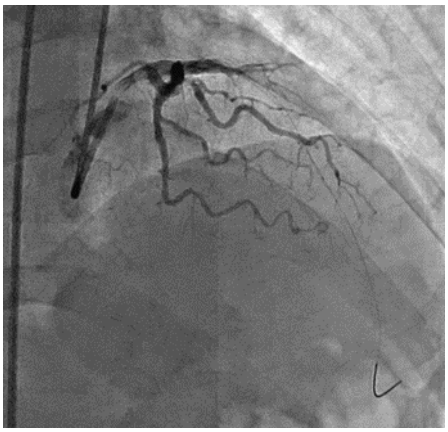


Figure 4 No reflow phenomenon

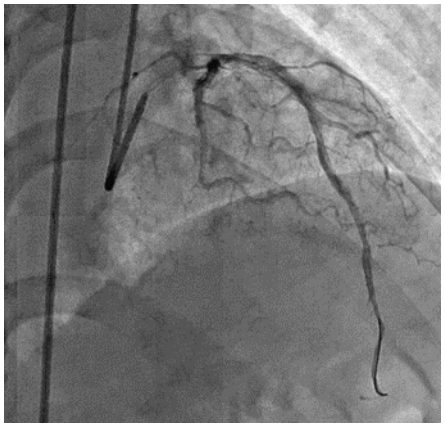


Figure 5 TIMI 3 flow

The second case was a 56-year-old male smoker, with underlying diabetes and hypertension, previously defaulted medications and follow up, presented to a non-PCI capable centre, diagnosed with late presentation inferior STEMI complicated with cardiogenic shock and uncontrolled diabetes. His ECG revealed ST elevation and deep Q wave over inferior leads (Figure 6). Echocardiography revealed impaired left ventricular ejection fraction of 35-40% with the presence of hypokinesia. He was admitted to coronary care unit, started with anticoagulation and DAPT, stabilised with fluid challenge and Intravenous dobutamine infusion. He was transferred to our centre for urgent angiogram. Angiogram revealed acute total occlusion at proximal RCA and diffuse severe stenosis of 80-90% at the LAD and LCX (Figure 7). Adhoc PCI to RCA was performed. IV heparin 5000unit (100unit/kg) stat was given. RCA was engaged with JR 4.0 6 Fr and Runthrough Floppy wire was negotiated to the distal RCA. Pre-dilatation at mid to ostial RCA with semi-compliant balloon Pantera 2.5mm X 15mm was done. PCI with DES Orsiro 3.0mm X 35mm at mid RCA and DES Orsiro 3.5mm X 35mm at proximal to ostial RCA were performed (Figure 8). Unfortunately, no reflow phenomenon was observed again immediately post PCI (Figure 9). IC GTN 200mcg, repeated IC adenosine 200mcg and IC Tirofiban loading dose were administered with close haemodynamic monitoring. Final angiogram demonstrated TIMI 3 flow without dissection, perforation or thrombus (Figure 10).

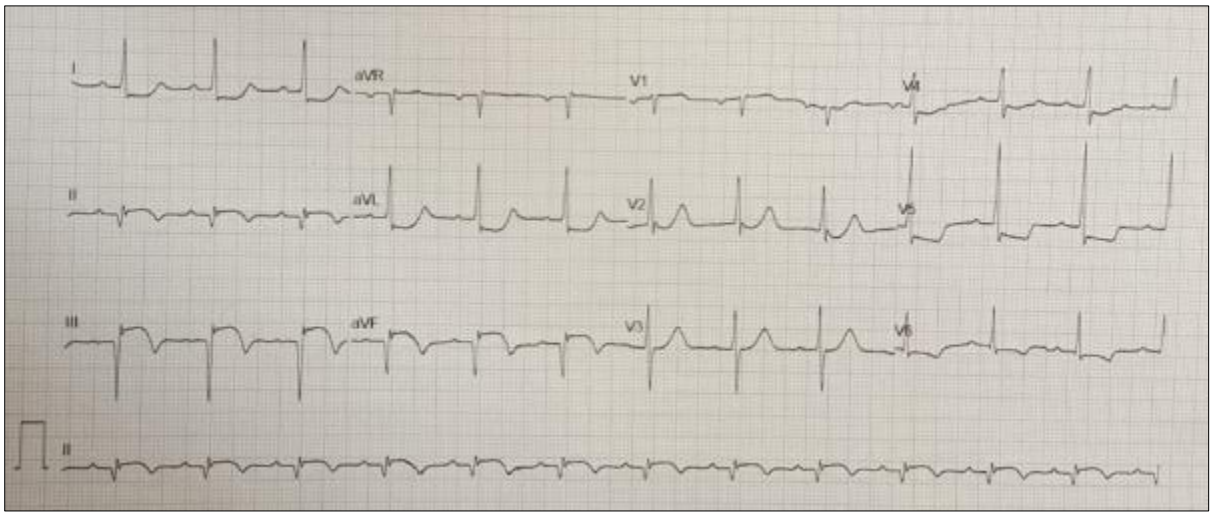


Figure 6 ECG

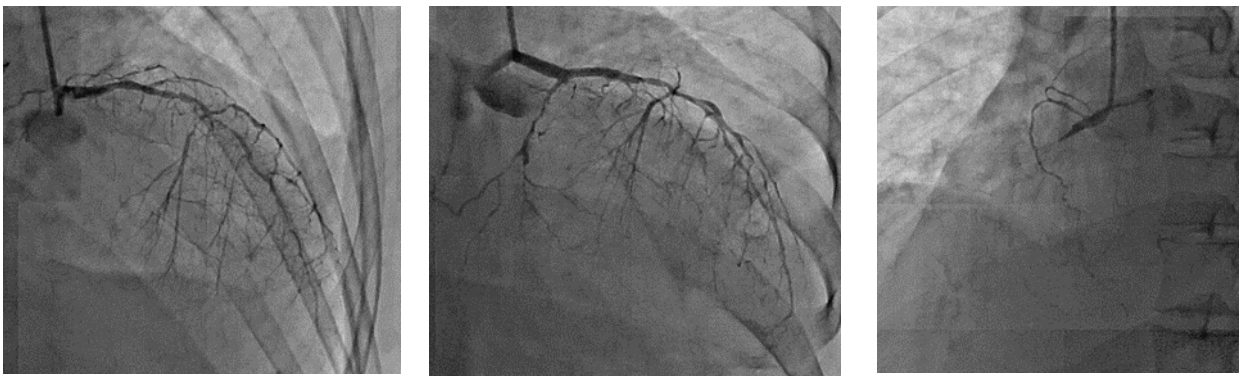


Figure 7 Angiogram

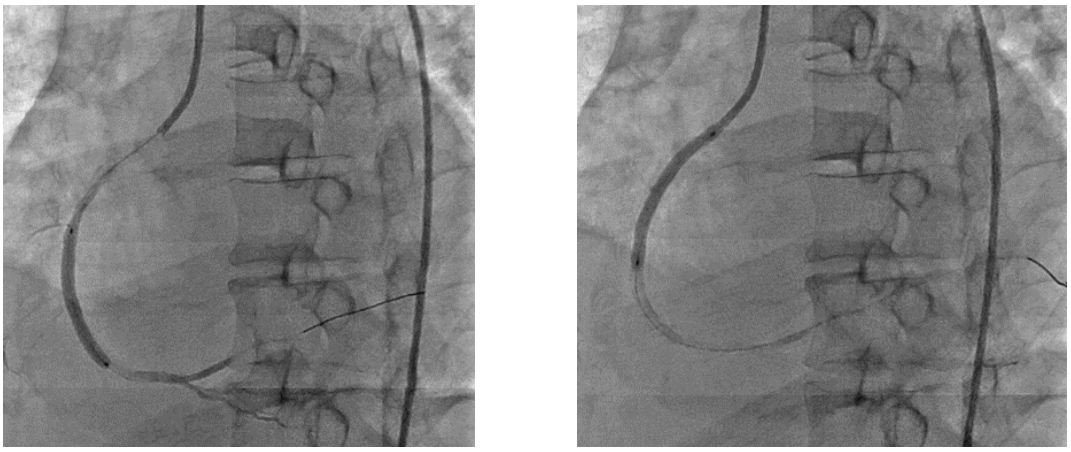


Figure 8 PCI to RCA

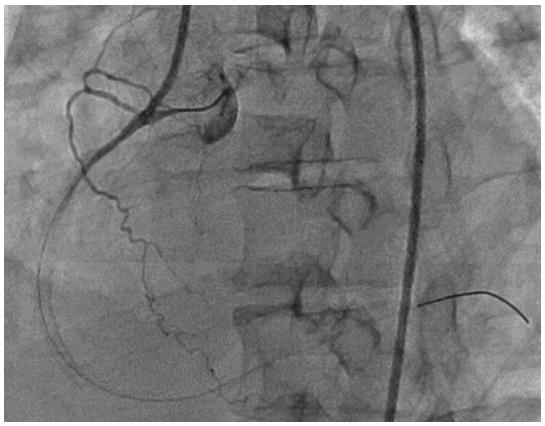


Figure 9 No reflow phenomenon

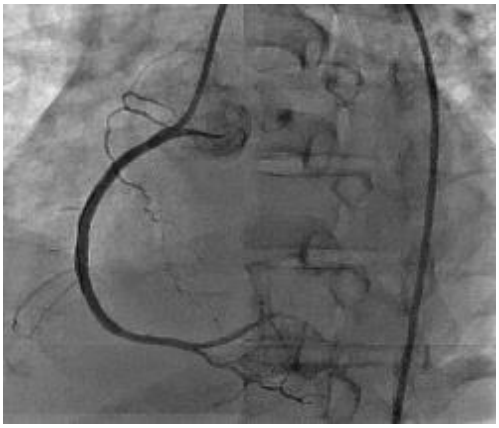


Figure 10 TIMI 3 flow

3. Discussion

The incidence of acute no reflow phenomenon is approximately 1-3% in overall PCI cases [4]. It is more common in certain conditions such as tobacco use, late presentation of acute coronary syndrome, longer door-to-balloon time, high thrombus burden, venous bypass graft PCI, use of rotational atherectomy, low left ventricular ejection fraction, high Killip classification and also uncontrolled diabetes and hypertension [4,5]. In our cases, both patients were smokers, and had the risks factors of diabetes and hypertension. Besides that, our second patient had impaired LVEF and delayed door to balloon time. These risk factors may explain the development of acute no reflow phenomenon. Optimisation of the modifiable pre-procedural and intraoperative risk factors are important to prevent this phenomenon. Before procedure, risk factors modifications include satisfactory control of diabetes and hypertension. Intensive statin reduces the incidence by 4.2% [6]. Double antiplatelet is also beneficial in reducing complications such as acute stent thrombosis, periprocedural myocardial infarction and acute no-reflow [4]. Intraoperatively, patient should be adequately anticoagulated with unfractionated heparin and monitored with activated clotting time [4]. The dose of heparin required is 70–100 UI/Kg. Intracoronary nitrates also reduce the risks. Besides, using appropriate catheter can prevents catheter induced obstruction and subsequent thrombus formation. Thrombus and air emboli can also be avoided by regular catheters flushing [4]. Rotational atherectomy is a procedure used to treat coronary calcification. It has a burr rotating at high speed which will generates friction and heat. This will lead to microembolization of debris and thermal injury and subsequently microvascular obstruction and no-reflow phenomenon [4,7]. Appropriate manipulation of the rotational atherectomy is important to reduce acute no-reflow. Recommended techniques are using single burr with burr-to-artery ratio of 0.5 to 0.6, rotational speed of 140,000 to 150,000 rpm, gradual burr advancement with a pecking motion, short ablation runs of 15 seconds, and avoid decelerations >5,000 rpm [7].

Once the no-reflow phenomenon is observed, first we need to exclude mechanical causes such as coronary spasm, coronary haematoma, coronary dissection, thrombus formation and distal embolization [4]. Studies suggested that no flow phenomenon might be caused by microvascular dysfunction in the ischemic area. Postulated mechanisms for microvascular dysfunction are free radicals, cardiac sympathetic reflexes causing vasoconstriction or interactions between activated polymorphonuclear leukocytes and the endothelium [8]. Patient haemodynamic stability needs to be ensured and supportive measures such as fluid administration or inotropic support might be needed to stabilise the haemodynamic if necessary before pharmacological therapy is administered. Several agents are beneficial in treating acute no reflow phenomenon. Adenosine is one of the most frequently used agents in treating acute no reflow. It is an endogenous short-acting nucleoside. It has a potent vasodilatory effect. It also has antiplatelet effect, which can inhibit platelet aggregation, thrombo-emboli formation and restores endothelial function. [9,10]. However, it has a short half-life, and potential complication of blocking atrioventricular nodal and hypotension. Sodium nitroprusside and other nitrates agents are also commonly used. They have greater and longer vasodilatory and myocardial hyperaemic effects [11]. Intracoronary calcium channel blockers such as verapamil, diltiazem and nicardipine are other effective medication in preventing and treating this phenomenon [12]. Glycoprotein IIb/IIIa platelet receptor inhibitor can reduce platelet aggregation, thrombus burden and distal thrombus embolization and thus improve epicardial and myocardial flow [4].

We reported two cases of acute no reflow phenomenon immediately post PCI that had occurred in the same day. Both were straight forward fast procedures and were adequately heparinized. The lesions were treated and covered adequately with PCI. Both cases of acute no reflow resolved with medical therapy. In the absence of obvious thrombus,

dissection, spasm, or residual stenosis, we postulated that the most likely aetiology was microvascular dysfunction. Both patients remained asymptomatic and discharged well the next day.

4. Conclusion

Our intention of presenting these two cases is to emphasize the importance of cardiologist recognising the acute no reflow phenomenon which can results in increased mortality and morbidity. Besides, early detection and treating the modifiable risk factors, high index of suspicion, risks and complications awareness, timely intervention are vital in treating this phenomenon and improving patient prognosis.

Compliance with ethical standards

Acknowledgments

We wish to thank all the staffs of the Invasive Cardiac Laboratory for their contribution in the cases.

Disclosure of conflict of interest

The authors declare no conflicts of interest.

Statement of ethical approval

The present research work does not contain any studies performed on humans' subjects by any of the authors.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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