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Case Report

Degree of Coronary Occlusions Links to The Patient Clinical Outcome: Four Cases of Double Culprits Acute ST-Segment Elevation Myocardial Infarction

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Abstract

Background: Double coronary culprit lesions in ST-segment elevation myocardial infarction (STEMI) is uncommon. Despite successful primary percutaneous coronary intervention (PPCI) in all culprit lesions, the clinical outcome remains unfavorable and the possible factors for the outcome are not fully understood.

Cases Presentation: We reported four cases of double culprit lesions STEMI underwent PPCI. Patient A, a 62 y.o. male with extensive anterior-inferior STEMI, had total occlusion (TO) at both proximal left anterior descending (LAD) and mid right coronary artery (RCA). Patient B, a 42 y.o. male with extensive anterior-inferior STEMI, had subtotal occlusion (STO) at proximal RCA and TO at proximal LAD. Both of them had RBBB ECG pattern. Patient C, a 67 y.o. male with inferior STEMI had 90% thrombus occlusion at proximal–mid LAD and TO at proximal RCA. Patient D, a 65 y.o. male with anteroseptal STEMI, had STO at proximal LAD and 80% thrombus occlusion at mid left circumflex. The cardiomyocyte infarction biomarkers increased in all patients. Although all of them underwent successful PPCI in all of culprit lesions, they suffered from acute heart failure and two of them experienced recurrent ventricular arrhythmia episodes. One of them (patient A) died two days post PPCI. He was only patient who suffering from total occlusion in LAD and RCA with TIMI thrombus 5 and experienced a total atrioventricular block post-PPCI.

Conclusion: STEMI with coronary double culprits have severe clinical outcome, regardless of the successful PCI. The degree of coronary occlusions might be linked to the patient clinical outcome

Keywords: ST-segment elevation myocardial infarction; double coronary artery culprits; clinical outcome; degree of coronary occlusion

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INTRODUCTION

ST-segment elevation myocardial infarction (STEMI) is a cardiac event caused by rupture of coronary plaque leading to activation of platelets, formation of thrombus and occlusion of the coronary culprit.[1] Typical clinical presentation of patients is chest discomfort accompanied with diaphoresis, nausea or vomitus.[1] Electrocardiogram (ECG) criteria is pivotal in diagnosing of patient with STEMI. Management of this complicated condition is challenging and time-limited. Appropriate clinical approach and rapid revascularization strategy are important to reduce mortality and morbidity in STEMI patients.

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Primary percutaneous intervention (PPCI) is the recommended strategy of reperfusion in STEMI patient within 12 hours of symptom onset.[1,2]

Incidence rate of STEMI due to a simultaneous double culprit lesion of coronary arteries is rare with a range from 1.7 to 4.8 in all PPCI.[1]Less than 30 PPCI cases have been reported. It remains unclear how coronary arteries are occluded simultaneously. Several factors may be involved in the event, including hypercoagulability, multivessel spasm, thrombocythemia, and cocaine abuse. Traditional risk factors of coronary artery disease such as diabetes mellitus (DM), cigarette use, and dyslipidemia also contribute to the occlusion.[3,4]

Clinical outcome of STEMI patients with double culprit lesions is expected to be worse than those with single culprit. An acute thrombotic occlusion of more than one coronary tree causes an extensive myocardial infarction. The later links to worse clinical outcome including a high risk of mortality.[5]

1. CASEPRESENTATION

In this report, we presented 4 cases of patients suffering from STEMI with double coronary culprit lesions underwent successful PPCI, however the outcomes remained unfavorable.

Patient A, a 62 y.o. male with a 3-hour onset of acute crushing retrosternal chest pain with coronary artery disease (CAD) risk factor was an active smoker with 3 packs a day for years. On physical examination, his respiratory rate (RR) was 22 times per minute, heart rate (HR) was 86 times per minute, and blood pressure (BP) was 140/91 mmHg. Jugular veins, pulmonary sound, and cardiac physical examination were within normal limits. Peripheral pulses were intact with no oedema. His initial electrocardiogram (ECG) showed ST-segment elevations in the inferior (II, III, and a VF) and anterior (V3–V6) leads with incomplete RBBB pattern (Figure 1a), indicated acute inferior and anterior STEMI. The patient received loading doses of dual antiplatelet (320 mg aspirin and 180 mg ticagrelor) as well as 80 mg atorvastatin. Coronary angiography procedure revealed thrombus type total occlusions (TO) at the mid right coronary artery (RCA) (Figure 1c) and proximal left artery descending (LAD) (Figure 1d). A drug eluting stent (DES) was implanted at proximal LAD (Figure 1e), and sequentially followed by RCA intervention using two DES at mid and distal part of RCA (Figure 1d). TIMI grade 3 flow was seen in both LAD and RCA, neither residual stenosis nor dissection was observed. The echocardiogram data showed a low left ventricular ejection fraction (LVEF) (Table 1) and the patient was managed with intravenous diuretic and nitrate. Two days post PPCI, during hospitalization in the intensive cardiac care unit, the patient was unconsciousness and the ECG showed total AV block (TAVB) and

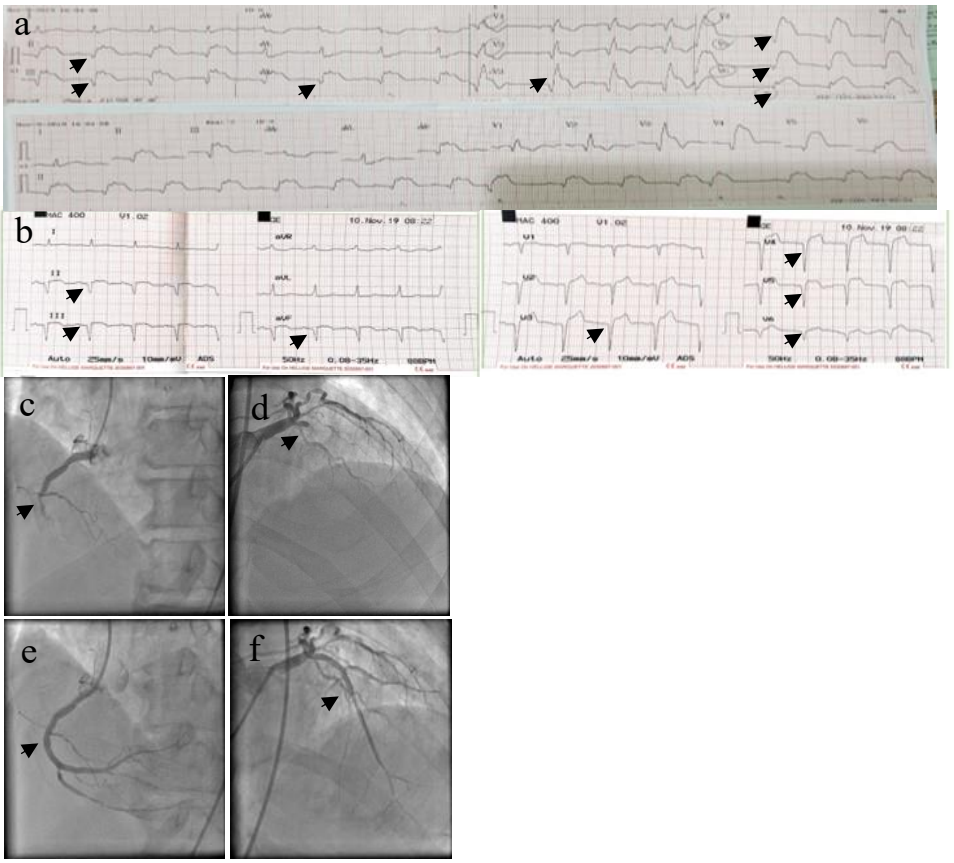

Figure 1. Electrocardiogram (ECG) and coronary angiogram of patient A. (a) Initial ECG pre-primary percutaneous intervention (PPCI) showed ST-segment elevation in the inferior (II, III, and a VF) and anterior (V3–V6) leads with incomplete right bundle branch block (RBBB) pattern, (b) the ECG post PPCI, (c) and (d) the respective angiograms of right coronary artery (RCA) and left anterior descending artery (LAD) pre PPCI, (e) and (f) the respective angiograms of RCA and LAD post PPCI. Arrowheads in (a) and (b) indicate the abnormality of ECG and in (c) and (d) point to the occluded coronary flow with corresponding antegrade flow recovery post stenting in (e) and (f).

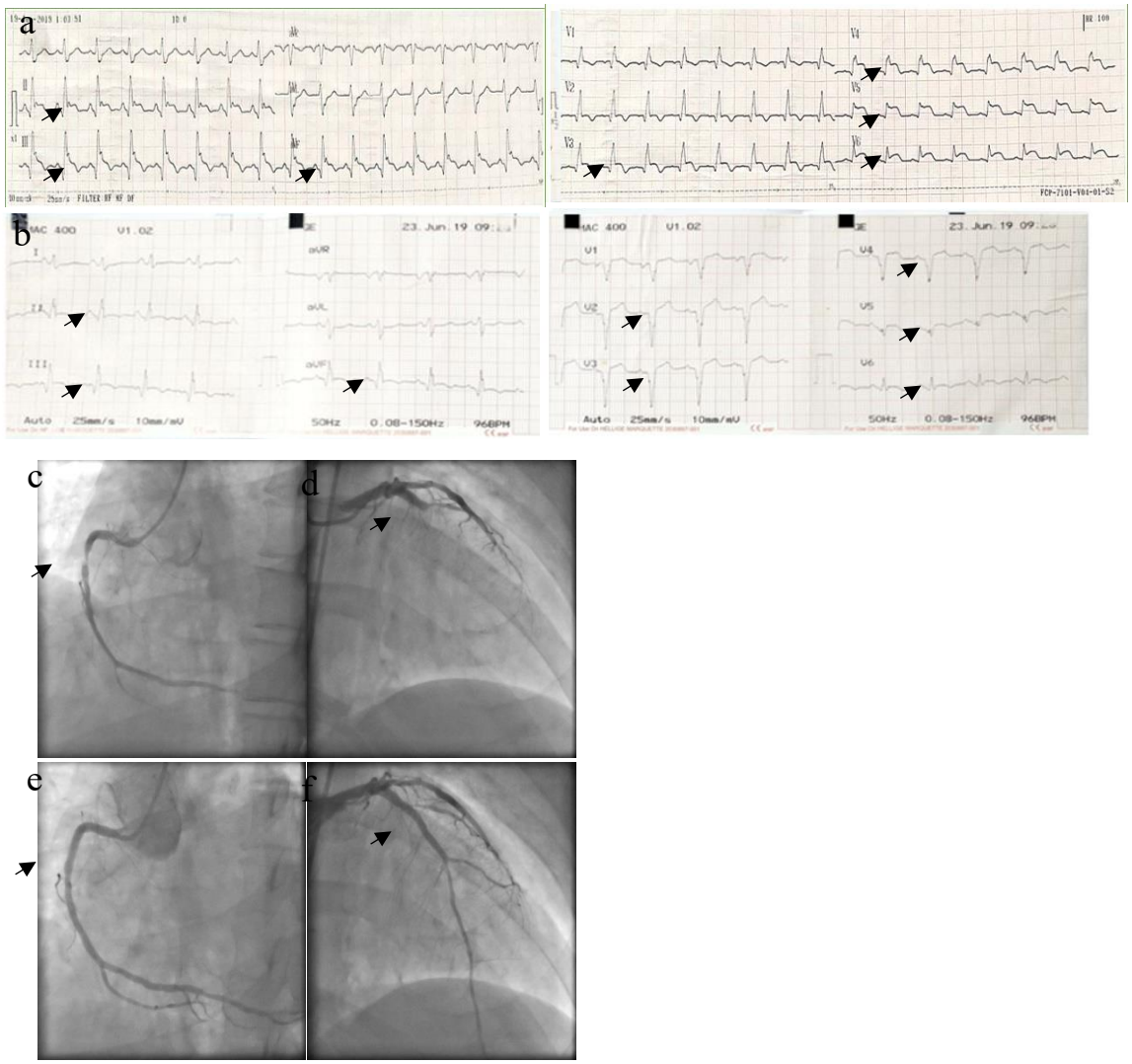

Figure 2. ECG and coronary angiogram of patient B. (a) Initial ECG pre-PPCI showed ST-segment elevation in the inferior (II, III, and a VF) and anterior (V3–V6) leads with incomplete RBBB pattern, (b) the ECG post PPCI, (c)–(e) the respective angiograms of RCA and LAD: (c) and (d) pre-PPCI, (e) and (f) post PPCI. Please see Figure 1 for the abbreviations and annotation of arrowheads.

ventricular tachycardia (VT) that unresponsive to both medical and electrical treatments. After 45 minutes cardiopulmonary resuscitation, the patient passed away. His laboratory result showed increase level of CKMB 471 unit/L and troponin I>40ug/L, with a slight hypokalemia 3.4mmol/L (Table 1). Although the cause of deterioration remained unclear, the event of TAVB and increase of troponin level suggested that the patient experienced an acute coronary thrombosis again.

Patient B, a 42 y.o. male presented with a typical angina pectoris,4.5 hours onset, accompanied by diaphoresis and nausea. His CAD risk factors were hypertension and active smoker with one pack cigarettes per day for the past 10 years. The physical examination showed RR 24 times per minute, HR 100 times per minute, and BP 114/91 mmHg. Cardiac and pulmonary physical examination were within normal limits. His ECG revealed elevation of ST-segment in the inferior (II, III, and a VF) and anterior (V3–V6) leads with incomplete RBBB pattern (Figure 2a). Loading doses of aspirin 320 mg, ticagrelor 180 mg, and 80 mg atorvastatin were administrated. PPCI was performed and coronary angiogram showed a thrombus type lesion at the proximal portion of RCA (Figure 2c) and totally occluded of proximal portion of LAD (Figure 2d). Proximal LAD was implanted one DES (Figure 2f) and middle part RCA was implanted one DES (Figure 2e) with TIMI grade 3 flow and no residual stenosis. Post PPCI procedure, the patient got pulmonary edema managed with nitrate and diuretic. The patient's symptoms and the ECG (Figure 2b) were significantly improved and there was no further orthopnea. Patient was transferred into cardiac intensive care unit and received dual antiplatelet aspirin and ticagrelor, ACE-inhibitor, and atorvastatin 40 mg once daily. Fondaparinux was given until the patient was discharged from hospital.

Patient C, a 67 y.o. male with CAD risk factors of active smoker two pack cigarettes per day for the last 8 years and dyslipidemia. He came to emergency room with 3 hours onset of retrosternal chest pain, diaphoresis, and vomitus. The physical examination showed RR was 18 times per minute, HR was 87 times per minute, and BP was 124/95 mmHg. Cardiopulmonary physical examination was normal. His initial ECG showed ST-segment elevation in the inferior (II, III, and a VF) and anterior (V4–V6) leads (Figure 3a), indicated acute

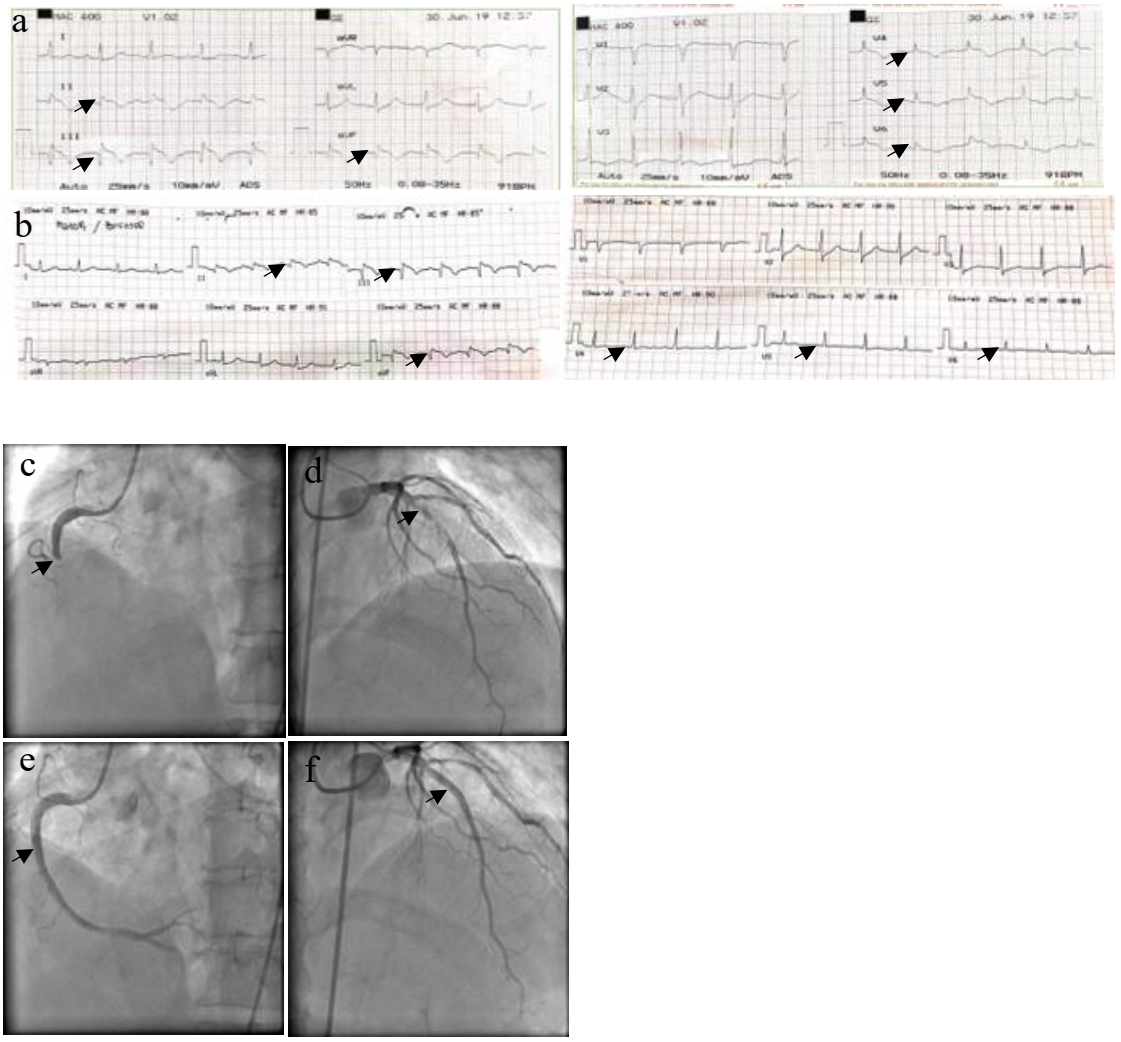

Figure 3. ECG and coronary angiogram of patient C. (a) Initial ECG pre-PPCI showed ST-segment elevation in the inferior (II, III, and a VF) and anterior (V4–V6) leads, (b) the ECG post PPCI, (c)–(e) the respective angiograms of RCA and LAD: (c) and (d) pre-PPCI, (e) and (f) post PPCI. Please see Figure 1 for the abbreviations and annotation of arrowheads.

inferior and anterior STEMI. Aspirin 320mg, ticagrelor180mg, and atorvastatin 80mg were given before PPCI. Coronary angiogram revealed a thrombus type total occlusion in proximal RCA(Figure3c) and 90% stenosis in proximal LAD with a hazy filling defect (Figure3d). During PPCI, the patient developed several episodes of ventricular fibrillation. PPCI was performed, each of the RCA (Figure3e) and LAD (Figure 3f) was stented with one DES. In the final angiogram, TIMI grade flow in LAD was 3, but in the RCA was 2. Echocardiographic examination was performed and the LVEF was low (Table 1) and thus an intravenous diuretic and nitrate were given. He was discharged on day 5th with aspirin 80 mg once daily, ticagrelor 90 mg twice a day as well as ACE-inhibitor, beta blocker and atorvastatin 40 mg once daily.

Patient D, a 65 y.o. male with history of type II diabetes mellitus, hypertension, and dyslipidemia came with 24 hours onset of typical angina pectoris, diaphoresis, and nausea. Physical examination showed RR 22 times per minute, HR 78 times per minute, and blood pressure was 98/87 mmHg. No abnormality was found in the cardiopulmonary physical examination. Peripheral pulses were intact with no oedema. His initial ECG showed ST segment elevation in anterior (V1-V4) leads (Figure 4a). The patient was loaded with aspirin 320 mg, ticagrelor 180 mg, and atorvastatin 80 mg and immediately sent to catheterization laboratory for PPCI. Coronary angiography procedure showed a thrombus type subtotal occlusion in proximal LAD (Figure 4c) and in mid left circumflex (LCx) artery (Figure 4d). The patient underwent successful PPCI, TIMI grade 3 flow, with one DES was implanted in each of LAD and LCx (Figure 4e and f). Although the patient experienced pulmonary edema, his symptoms and the ECG (Figure 4b) were significantly improved and patient transferred to intensive care unit. His LVEF was 48%. Then, he was regularly managed with dual antiplatelets aspirin and ticagrelor, atorvastatin, and ACE-inhibitor. Fondaparinux was given for 5 days. The patient was discharge after 7 days hospitalization.

Follow up data during hospitalization showed that all patients had acute heart failure (Table 1) as the complication of acute STEMI regardless of successful coronary revascularization by primary percutaneous coronary intervention.

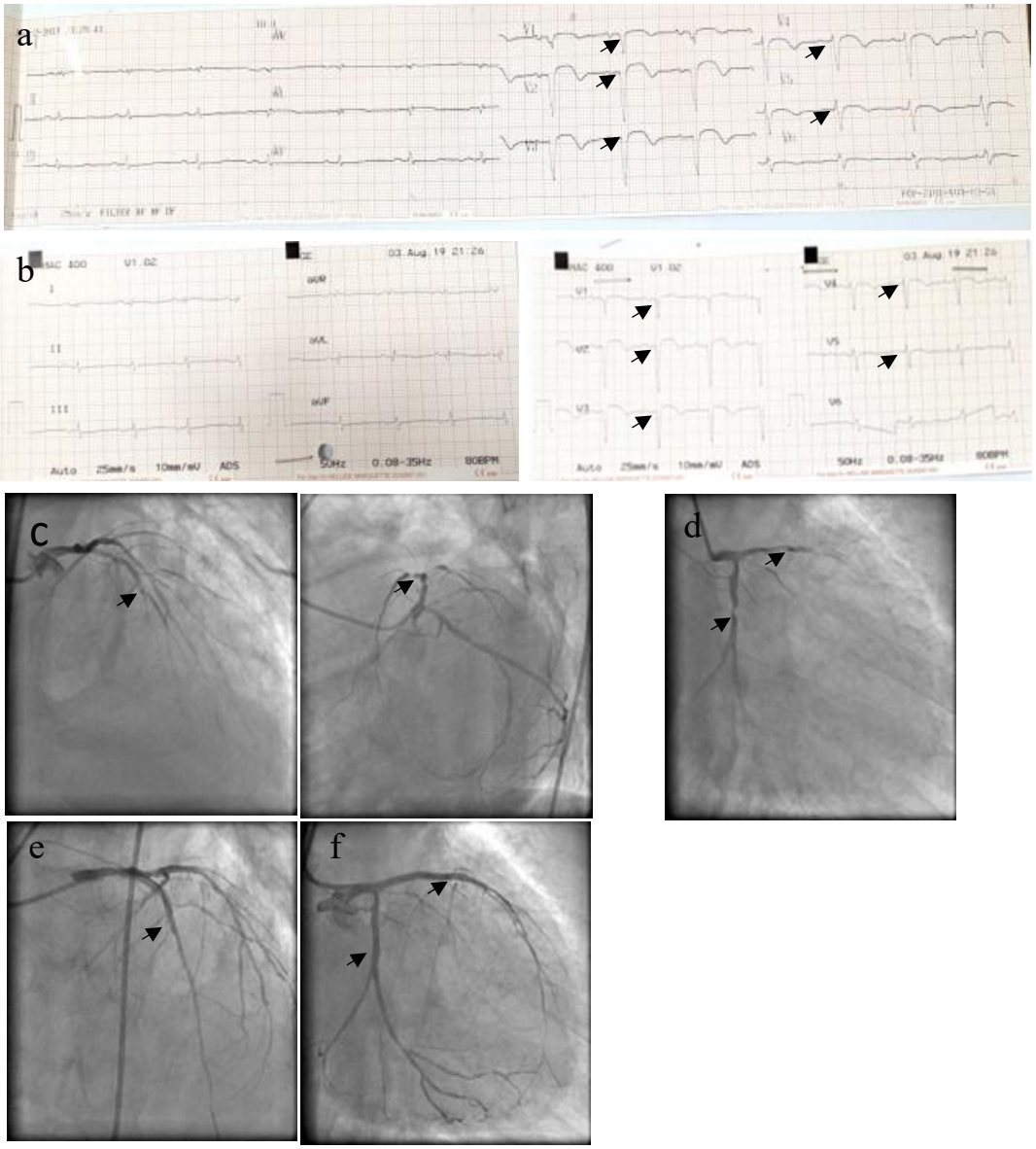

Figure 4. ECG and coronary angiogram of patient D. (a) Initial ECG pre-PPCI showed ST-segment elevation in the anterior (V1–V4) leads, (b) the ECG post PPCI, (c)–(e) the respective angiograms of LAD and left circumflex artery (LCx): (c) and (d) pre-PPCI, (e) and (f) post PPCI. Please see Figure 1 for the abbreviations and annotation of arrowheads.

2. DISCUSSION

STEMI is resulted from erosion or rupture of atheromatous plaque followed by thrombotic occlusion of coronary artery.[1,2] In the daily clinical practice, spontaneous occlusion of two coronary arteries is rare, with the estimated prevalence around 2.5%.[6] But the actual number of the prevalence could be underestimated because of bias of selection associated with fatal outcomes.[2] In STEMI patients with culprits in two vessels, the most frequent finding in coronary angiogram is occlusions of two arteries originating from different sides of a coronary tree.[2,7] In a case series of 18 patients, nine patients showed double occlusion in both RCA and LCx,[6] however after adding 29 cases, most of the double occlusion was in the RCA and LAD.[1,2,7] In our current case series, 3 patients presented concomitant thrombosis in LAD and RCA, while one patient in LAD and LCx. A simultaneous occlusion of LAD and LCx in patients with STEMI is very rare.[8]

The underlying mechanism responsible for multiple vessel culprits remains unclear. A proposed mechanism is linked to inflammatory response inducing diffuse destabilization of plaques in more than coronary vessels.[4,7] Since acute coronary syndrome is an exacerbation of chronic inflammatory disease in atherosclerosis, the term "pancoronaritis" is then established. A study reported that more than thirty percent of patients with acute coronary syndrome had multiple plaque ruptures with overlying thrombi.[4] This was supported by another report from an autopsy of STEMI patients which found multiple thromboses in the coronarytree.[9]The multiple plaque ruptures can be not only came from simultaneous, but also sequential thrombotic occlusion of more than one coronary arteries.[11,12]Further possible mechanism is that the occlusion of a coronary artery may induce hemodynamic instability and hypotension resulting in stasis of blood inducing occlusion in another diseased coronary artery.[6,13]

Table 1. Clinical, laboratory, electrocardiographic, and angiographic characteristics of patients

	Patient A	Patient B	Patient C	Patient D	Normal Value
Clinical data					
Age (years old)	62	42	67	65	
Sex	Male	Male	Male	Male	
Onset of angina (hours)	3	4.5	3	12	
CAD risk factor	Active smoker	Active smoker, hypertension	Active smoker, dyslipidaemia	Diabetes, hypertension, dyslipidaemia	
Electrocardiogram at admission	Anterior-inferior ST elevation, RBBB	Anterior-inferior ST elevation, RBBB	Anterior-inferior ST elevation	Anterior ST elevation	
Laboratory data					
CKMB (U/L)	471	88	378	21	7-25
Troponin I (ug/L)	0.449	>50	0.014	>40	0.015-0.038
Haemoglobin (g/dL)	14.8	16.6	13.5	11	13-16
Leucocyte (10^3/uL)	15	20.9	10.8	11.1	3.8-10.6
Thrombocyte (10^3/uL)	301	335	380	243	150-400
Ureum (mg/dL)	35	24	28	73	15-39
Creatinine (mg/dL)	1.14	0.7	0.89	1.12	0.6-1.3
Magnesium (mmol/L)	0.92	0.8	0.78	1.0	0.74-0.99
Calcium (mmol/L)	2.04	2.2	2.37	1.93	2.12-2.52
Sodium (mmol/L)	145	139	132	127	136-145
Potassium (mmol/L)	3.4	4.8	3.8	3.5	3.5-5.1
Chloride (mmol/L)	103	106	98	96	98-107
Blood glucose (mg/dL)	154	244	316	111	80-160
Fasting glucose (mg/dL)	133	158	189	127	≤125
Total cholesterol (mg/dL)	170	209	160	199	<200
Triglyceride (mg/dL)	150	173	75	227	<150
HDL cholesterol (mg/dL)	31	32	47	19	40-60
LDL cholesterol (mg/dL)	127	157	96	140	0-100
HbA1C (%)	5.2	6.6	10.4	8.8	6-8
Coronary intervention data					
Degree of occlusion at the culprit lesions	100% pLAD 100% mRCA	100% pLAD 99% pRCA	90% pLAD 100% pRCA	99% pLAD 80% mLCx	
TIMI thrombus	LAD 5 RCA 5	LAD 5 RCA 3-4	LAD 2-3 RCA 5	LAD 2-3 LCx 1-2	
TIMI flow post stenting	LAD III RCA III	LAD III RCA III	LAD III RCA II	LAD III LCx III	
TIMI frame count post stenting					
RCA	13 frames	19 frames	29 frames	18 frames	
LCx	5 frames	11 frames	8 frames	10 frames	
LAD (corrected)	22.36 frames	11.18 frames	17.65 frames	7.06 frames	
Post PPCI electrocardiogram	QS at anterior-inferior leads	QS at anterior leads	QS at inferior leads	QS at anteroseptal leads	
Echocardiogram					
LVEF (%)	35	42	36	48	>50
Clinical outcome	AHF, TAVB, ventricular arrhythmia	AHF	AHF, ventricular arrhythmia	AHF	

RBBB, right bundle branch block; PPCI, primary percutaneous intervention, TO, total occlusion; STO, sub-total occlusion; pLAD, proximal left anterior descending artery; mRCA mid right coronary artery; mLCx, mid left circumflex artery; TAVB, total atrioventricular block; AHF, acute heart failure

In the current case series, all patients had acute heart failure following myocardial infarction and two of them, patients A and C, experienced a malignant arrhythmia. In addition to ventricular arrythmia, patient A suffered from total AV block and thus had an unfavorable outcome ended with a mortality. The possible risk factors for having these catastrophic complications were hypertension, history of smoking, poorly controlled lipid profile and diabetes mellitus. Other possible mechanisms that may involve in clinical outcome severity are hypercoagulable state and autoimmune diseases including in-

creased frequency of factor V Leiden, prothrombin variant 20210A, and elevated levels of lipoprotein(a).[7]In patients A and C, these factors may be considered to be checked.

Numerous factors could affect the clinical status, including degree of coronary occlusions, vascular tonus, diameter of vessel, and adrenergic surge.[8] The degree of coronary occlusions refers to the angiographic thrombus burden classification published elsewhere.[9] The existence of new right bundle branch block in patient with anterior STEMI and a high level of biomarker of cardiac myocyte infarction (Figure 1 and 3 and Table 1) indicates a wide area of myocardial infarction and link to poor prognosis as well as high mortality. Thrombus type total occlusion of two major coronary arteries was considered as the cause of mortality of patient A in this series, as can be seen in Figure 1 to 4 and Table 1 that only patient A had thrombus type total occlusion in two coronary arteries. Furthermore, TIMI frame count of LAD post stenting was higher in the patient A and C (Table 1) suggesting that there was a microcirculation impairment of the myocardium in those patients. The deteriorated condition in patient A may related with the impairment. This is supported by a report by Gibson et al. that increasing of TIMI frame count is related with worst clinical outcome.[10] Thus, taken together, the degree of coronary occlusions might be linked to the clinical outcome of the patients.

All coronary culprit lesions of patients in these cases were revascularized during PPCI. The number of reported cases with simultaneous thrombotic occlusions of multiple coronary arteries remained small and thus the management and outcome of patients with this characteristic are still not fully discussed in the recent STEMI guidelines. Recent studies find a benefit of complete revascularization in patients with STEMI,[13,15] and thus support the idea of complete revascularization during acute phase of patient with two culprit `arteries.[1,2,4]

Anticoagulant prescription post primary PCI remains controversial.[1,16] However, in all patients in this series received anticoagulant therapy after revascularization to prevent further thrombosis. Benefit of anticoagulant therapy in a high thrombus burden patient should be considered carefully while the risk of bleeding must be calculated as well. Therefore, a routine anticoagulation post PPCI should be avoided unless an indication is present and should consider benefit-risk profile.[16] Although all patients in this case series did not treated with a glycoprotein IIb/IIIa inhibitor, the use of this intravenous antiplatelet during PPCI should be considered if there is evidence of no-reflow or a thrombotic complication.[1]Therefore, in the case high thrombus burden with high risk of recurrent thrombosis, intravenous anticoagulant or antiplatelet should be given post PPCI during hospitalization with causation of bleeding risk.

3. CONCLUSION

In the conclusion, we reported 4 cases of patients suffering from STEMI with double coronary culprit lesions underwent successful percutaneous coronary intervention, however the clinical outcome remained unfavorable. The degree of coronary occlusions might be linked to the patient clinical outcome.

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