Simultaneous thromboses of multiple coronary arteries in acute myocardial infarction

Tan I L, Tan H C, Teo S G, Lim Y T

ABSTRACT

Simultaneous thrombotic occlusion of multiple coronary arteries in acute myocardial infarction is a well-recognised phenomenon. Studies have reported diffuse destabilisation of atherosclerotic plaques in patients with acute myocardial infarction, leading to the concept of "pan-coronaritis". The putative mechanism is attributed to a systemic thrombophilic and inflammatory state. We report the occurrence of this phenomenon in two middle-aged male patients.

Keywords: acute myocardial infarction, coronary thromboses, myocardial infarction, simultaneous coronary occlusion

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INTRODUCTION

Acute myocardial infarction (AMI) results from thrombosis at the site of coronary plaque rupture or erosion with cessation of blood flow. Although uncommon, multiple coronary thromboses occurring simultaneously in patients with AMI have been reported. We report two patients in our centre who were found to have more than one site of acute thrombosis at the time of their AMI presentation.

CASE REPORT

Case One

A previously healthy 46-year-old Chinese man with only cardiovascular risk factor of dyslipidaemia presented to the Emergency Department with sudden onset of severe crushing retrosternal chest pain at rest, associated with diaphoresis and nausea. His haemodynamics were stable and cardiovascular examination was normal. His resting 12-lead electrocardiogram (ECG) showed hyperacute ST-segment elevation in the anterior leads. Emergent coronary angiography showed an occluded proximal left anterior descending artery, the infarct-related artery, and a thrombus-laden right coronary artery (Fig. 1).

Both arteries were treated with X-Sizer thrombectomy followed by coronary stent

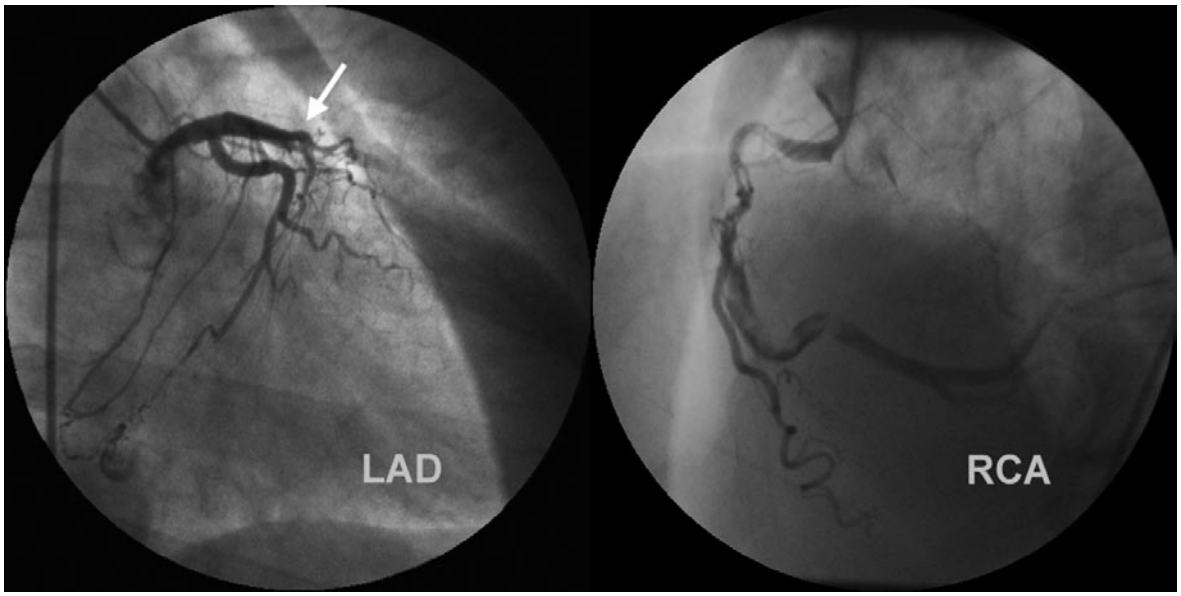

Fig. 1 Case 1. Coronary angiogram shows a completely occluded LAD artery (arrow) and thrombus-laden RCA.

Cardiac Department
National University Hospital
5 Lower Kent Ridge Road
Singapore 119074

Tan I L, MBBS, MRCP
Resident

Tan H C, M Med, FRCP, FACC,
Senior Consultant and Chief

Teo S G, BSc, MBBS, MRCP
Associate Consultant

Lim Y T, MMed, FRCP, FACC
Senior Consultant

Correspondence to:
Dr Tan Huay Cheem
Tel: (65) 6772 5213
Fax: (65) 6777 1684
Email: tanhc@nuh.com.sg

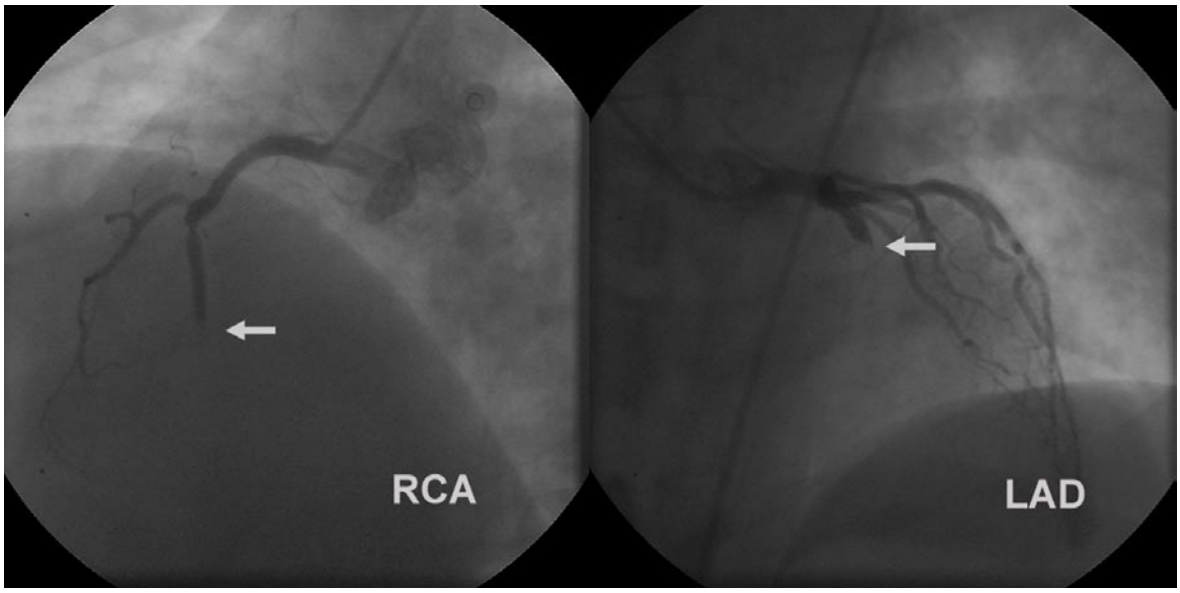

Fig. 2 Case 2. Coronary angiogram shows complete occlusion of the RCA (arrow) and the previously-stented LAD artery (arrow).

implantation with good results. His convalescence was uneventful and he was discharged five days later. Two-dimensional echocardiogram performed seven months afterwards showed a left ventricular ejection fraction (LVEF) of 38%, and scintigraphic perfusion scan showed a fixed defect involving the apex, inferior and inferolateral wall segments.

Case Two

A 35-year-old Indian man, with cardiovascular risk factors of type II diabetes mellitus, hypertension, positive family history of ischaemic heart disease and previous tobacco abuse, presented with exertional chest pain. He had prior coronary stenting to his mid-left anterior descending (LAD) artery five months ago. While undergoing a treadmill exercise stress test, he developed chest pain and collapsed. His ECG showed junctional bradycardia (50 beats/minute) and hyperacute ST-segment elevation in the anterior and inferior leads. Systolic blood pressure was low at 60 mmHg. Immediate coronary angiography performed showed complete thrombotic occlusion of the mid-right coronary artery and in the previously stented LAD artery (Fig. 2).

Intravenous abciximab was administered and intra-aortic balloon pump was inserted for mechanical support. Intravenous dopamine was also commenced for inotropic support. Both arteries were successfully revascularised with coronary stenting procedures. No obvious re-stenosis was noted in the LAD artery. The patient was discharged 11 days later. The latest echocardiogram performed five years later showed a mildly-impaired left ventricular systolic function with LVEF of 40%.

DISCUSSION

In treating AMI, a single culprit lesion is often stipulated to explain the phenomenon. However, multivessel simultaneous acute thromboses and occlusion resulting in multiple territorial infarctions can occur, and are found in our two patients. The occurrence, albeit uncommon, have been reported by several other investigators. Burke and Virmani found, in autopsy examination of patients who died of AMI, that 25-50% had multiple acute coronary thromboses[1]. Using coronary angiography, Goldstein et al found that many AMI patients have multiple coronary plaques with overlying thrombi. There were additional unstable lesions, defined as total or as stenosis with ulceration, fissuring and/or intraluminal filling defects in 21% of his patient cohort, and up to 32.8% and 10.0% of patients with acute AMI and unstable angina, respectively, had thrombi present in the non-culprit arteries. These multiple thrombi were observed equally in the proximal and distal segments of the arteries[2] and are associated with adverse clinical outcomes[3].

It is also well known that AMI is frequently caused by angiographically normal to mildly stenotic lesions that cannot be identified by coronary angiography. These so-called vulnerable plaques consist of cholesterol-rich lesions with thin fibrous cap that are prone to rupture or erosions. Angioscopy has been used to detect these vulnerable "yellow" plaques. Asakura et al showed that in patients with AMI, all three major coronary arteries were widely diseased and had multiple yellow plaques, though undisrupted[4]. These suggest that plaque instability

may not represent a more random "vascular accident" but perhaps reflect a "pan coronary" process.

Further evidence supporting a more generalised pathophysiological process comes from a coronary blood flow study by Hirayama et al[5]. He found that coronary blood flow is impaired, even in the non-infarct related artery (IRA) after mechanical revascularisation of the culprit artery in the setting of AMI. Coronary blood flow, measured by frame count, in the non-IRA remained high at over 30 frames count compared to the normal level of 21 frames count. The impaired coronary flow may indicate distal circulatory perturbations occurring as a result of microthrombi embolisation even in a non-IRA. The underlying putative mechanism for simultaneous multiple coronary occlusion is postulated to be a systemic inflammatory phase precipitating thrombi deposition on thrombosis-prone plaques[6,7]. The activation of inflammatory cytokines and the release of certain haematological or atherosclerotic factors following AMI result in a systemic thrombophilic state. These cause simultaneous triggering of rupture-prone plaques and consequent multivessel thrombosis.

What is the implication of this phenomenon in our clinical practice? Firstly, the simultaneous involvement of multiple coronary vessels in AMI results in injury to extensive areas of myocardium. Aggressive reperfusion therapy, preferably primary angioplasty, is crucial in salvaging the myocardium and to preserve left ventricular function. Both our patients suffered significant myocardial damage despite timely intervention. Intravenous glycoprotein IIb/IIIa platelet receptor inhibitors will be most useful in these situations in preventing further thrombus formation during percutaneous coronary intervention. Secondly, the concept of single-culprit vessel is challenged. We have to consider the possibility of multivessel simultaneous infarction readily, especially in patients who showed multiple areas of infarction on electrocardiogram, and who failed to improve clinically even after flow to the presumed "culprit vessel" is restored. The priority of vessel needed to be revascularised during primary coronary intervention procedure in AMI may be guided by the ECG changes. In general, the area of ischaemia recorded on the ECG should be treated first. However, in the event that ECG is not helpful, IRA subtending a larger myocardial territory may be revascularised first.

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