Rescue in situ thrombolysis for acute coronary thromboembolism in an angiographically normal coronary artery

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ABSTRACT

Coronary thromboembolism in an angiographically normal coronary artery is extremely uncommon. There are few instances where normal coronary arteries have been documented just prior to an episode of acute thromboembolic insult. We now report such a case of acute coronary thromboembolism in a patient with widely patent coronary vessels documented just prior to the event during preoperative screening angiogram with successful in situ revascularization.

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Acute coronary thromboembolism in the absence of significant coronary atherosclerosis is a rare, but distinct clinical entity.[1,2] In most of the previous reports, the diagnosis of coronary embolus was made mostly by presumption instead of being documented by the presence of in situ embolus in the coronary tree. The common associations are valvular heart disease, prosthetic heart valves, infective endocarditis and cardiomyopathies.[3] It is known that the nonfatal coronary embolism is under diagnosed resulting from atypical presentation. There are not many instances where normal coronary arteries were documented just prior to an episode of an embolic insult. We report such a case of acute coronary thromboembolic event in a patient with widely patent coronary arteries.

Case Report. A 57-year old man was admitted for elective cardiac catheterization. The patient's medical history included rheumatic heart disease, previous balloon dilatation of the mitral valve, progressive mitral regurgitation, atrial septal defect and chronic atrial fibrillation. The patient had progressive shortness of breath, and his functional capacity was noted as class III (New York Heart Association) at the time of admission. He was found to have moderate mitral stenosis, moderately severe mitral regurgitation and acquired atrial septal defect with significant left to right shunt. With this background, the patient was accepted for mitral valve surgery and atrial septal defect closure. He underwent right and left heart catheterization (using non-ionic contrast media Iohexol-Omnipaque 300). The study confirmed normal coronaries (**Figure 1**), moderate mitral stenosis and severe mitral regurgitation with significant left to right shunt across the atrial septal defect. Left ventricular function was moderately impaired with an ejection fraction of 40%. The procedure was uncomplicated, and he was transferred to the cardiac holding unit. An hour later he reported central chest pains. An electrocardiogram revealed acute ST-segment

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elevation in the anterior leads suggestive of acute occlusion of the left anterior descending artery. The patient was taken back to the cardiac catheterization laboratory. Selective coronary angiogram revealed abrupt occlusion of the mid left anterior descending artery with typical crescent like filling defect caused by a huge thrombus (**Figure 2**). The rest of the coronary tree was found to be normal. Successful recanalization and restoration of coronary flow to grade TIMI III (**Figure 3**) was obtained with intracoronary Eptifibatide (IIb IIIa glycoprotein inhibitor) bolus (180 micg/kg) and heparin (5000 units) through catheter manipulation. Serial electrocardiograms, cardiac enzymes and a repeat echocardiography were suggestive of a very small non-Q wave myocardial infarction involving a small distal segment of the left ventricular apex. Transesophageal echocardiogram (TEE) and Duplex scan did not reveal any cardiac, aortic or peripheral clot source. He made an uneventful recovery and proceeded to mitral valve replacement with Carbomedics mechanical valve and atrial septal closure one week following. No mural thrombus was noted during surgery and the patient tolerated the procedure very well. He has been keeping well for the last one year.

Discussion. Most of the previous reports including angiographic and autopsy studies confirmed the crucial pathologic role of coronary thrombosis in acute myocardial infarction. A coronary thrombus formation is usually associated with a significant coronary artery stenosis[4] and is uncommon in a normal coronary artery. One of the well-known causes of thrombus formation without apparent atherosclerotic stenosis has been coronary spasm.[5] A coagulation abnormality causing in situ thrombosis must also be considered. The precise mechanism of thrombus formation in our patient could not be identified and remained speculative. Angiographically undetected intimal damage, such as a small atheromatous plaque rupture, might have occurred during contrast injection and caused a predisposition to platelet activation and subsequent thrombosis (probably a pale thrombus, however, no angioscopy was available to prove it). The successful and complete lysis of this coronary thrombus with in situ glycoprotein IIb IIIa platelet inhibitor supports this theory. Despite negative post event TEE study for clots, the possibility of left atrial or appendage origin may not also be excluded with certainty. There was mild elevation of cardiac biological markers probably caused by fragmentation and downstream embolization causing distal micro infarction.

In summary, this case illustrates the importance of early recognition and intervention in acute coronary event to minimize myocardial damage, the

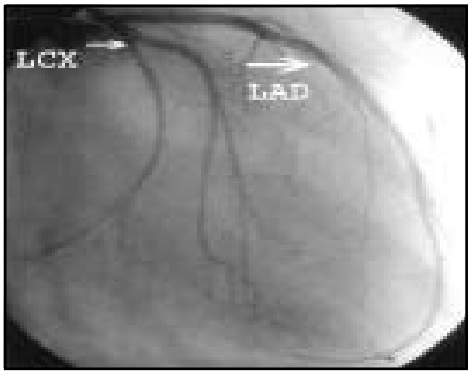

Figure 1 - Normal left coronary arteries. LAD - left anterior descending artery. LCX - left coronary artery

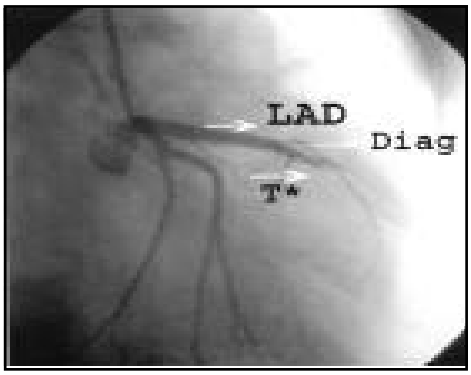

Figure 2 - Thrombus (T*) occluding the mid left anterior descending artery (LAD). Diag - diagonal

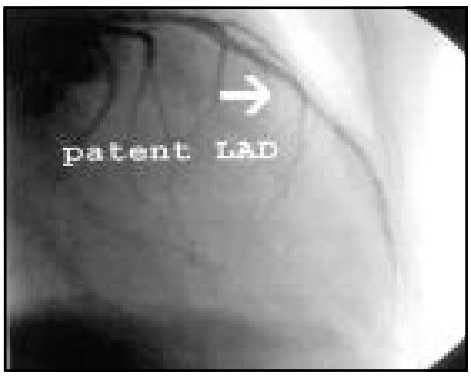

Figure 3 - Left anterior descending artery (LAD) after recanalization.

successful role of direct clot lysis with platelet's glycoprotein inhibitor and more importantly, how to minimize such occurrences via careful screening in atrial fibrillation, adequate anticoagulation and minimize contrast and catheter related intimal injury.

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