Regression of plaque burden after primary percutaneous coronary intervention (PCI) in a patient with TVD: 4 years follow-up

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Abstract
Coronary Artery disease (CAD) is an important cause of mortality and morbidity in the developed world as well as in Bangladesh. Treatment of Acute Myocardial Infarction (AMI) patient either by Streptokinase (STK) or Primary Percutaneous Coronary Intervention (pPCI) has increased the survival outcome and reduced the mortality. Several studies have documented the significant beneficial role pPCI in terms of in-hospital survival outcome over thrombolysis. Our patient, who had Anterior MI in 2004 and his CAG revealed TVD. pPCI of the culprit mid LAD lesion with Bare Metal Stent (BMS) was done immediately after hospitalization. He was later referred for CABG, but decision was postponed because of asymptomatic status. His re-look CAG on 20-02-2008 (i.e., 4 yrs after the original procedure), revealed patent LAD stent with the regression of atherosclerotic plaque in Ostio-Proximal LAD and proximal LCX. Our findings indicated that pPCI with rigid control of CAD risk factors and modification of lifestyle plays a key role in the regression of atherosclerotic plaque and maintenance of stent patency.

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Introduction: Acute coronary syndrome (ACS) is one of the important causes of death in the developing as well as developed world. Managing the patient either by STK or pPCI can reduce the mortality and improve 30 days in-hospital survival outcome. Primary angioplasty is the preferred treatment modality for AMI in centers with available cath lab facilities and well-trained personnel.1 The main advantage of pPCI is the achievement of a higher rate of coronary recanalization with a lower risk of intracranial bleeding.2 Now a days pPCI has become the choice of revascularization procedure compared to thrombolysis, for establishing TIMI III distal flow. It has already been established that pPCI is superior to thrombolytic therapy for the treatment of patient with STEMI. Many studies have demonstrated that pPCI within 2hrs of onset of symptoms has lower mortality and higher rate of complete reperfusion.3 Our present case shows the survival outcome in patient with acute Ant MI with TVD who had pPCI in Mid LAD in the year 2004.

Case: 61 yr Bangladeshi gentleman admitted to a local hospital with the onset of sudden chest pain for one and half hrs. His admission ECG revealed acute ST elevation in anterior chest leads (Fig.1). He is a known hypertensive on medication and active smoker with no known positive family history for CAD. He was taken to the cardiac cath lab and CAG revealed TVD (Ostial + mid LAD, tight distal LCX and 100% RCA) with the culprit lesion in the mid LAD (Fig. 2).

Figure 1: ECG before Primary PCI in 2004 during chest pain showing Acute ST elevation in Anterior Chest leads

Figure 2a: Culprit lesion in Mid LAD

Figure 2b: Stenting of mid LAD lesion
Regression of plaque

He was discharged in a stable condition and given beta blocker, losartan plus HTZ, atorvastatin with nicotinic acid, Clopidogrel plus aspirin. He was followed-up in the OPD with the continuation of medication, regular exercise, and diet control. He stopped smoking. He remained asymptomatic throughout. He did not attend OPD regularly, but otherwise remained compliant. In 2007, he was advised for SPECT perfusion scan for routine follow-up. SPECT perfusion revealed MI involving apical wall with a strip of per-infarct hibernation and also showed myocardial hibernation in septum (Fig.4).

Emergency pPCI to culprit lesion was done with a BMS of 3 x 19 mm size deployed at 14ATM and post dilated with 3.5 x 12 mm balloon at 12ATM. Final CAG showed no residual stenosis with achievement of TIMI III distal flow. Post procedural ECG showed almost normalization of Elevated ST in the anterior chest leads (Fig.3).

He was planned for a re-look CAG to evaluate his coronary status. This revealed, a patent LAD stent and regression of Ostio-proximal LAD plaque (Fig.5)(Fig.6).
Slow flow in the circ and diffuse small vessel disease was noted, explaining SPECT perfusion findings. He was discharged in a stable condition with the advice of continuing medication, lifestyle modification and regular follow-up.

**Discussion:** It has been well established that the survival outcome of patients with Acute Myocardial Infarction by primary PCI is superior to conventional thrombolytic therapy. Weaver et al., demonstrated that pPCI results in reduced rates of mortality, reinfarction, and stroke. It has been well documented that PTCA alone in AMI, recurrence of ischaemia or re-infarction occurs in 37-49% and late infarct related artery re-occlusion in 9-14%. Stone has demonstrated that pPCI (stenting) has low rates of in-hospital death (0.8%), re-infarction (1.7%), recurrent Ischaemia 3.8% and pre-discharge target vessel revascularization for ischaemia (3.8%). In patient undergoing pPCI, procedural success provides significant prognostic values. Patient selection, lesion size, vessel caliber and plaque burden, overall door-to balloon time are the key factors in the procedural success and in-hospital 30 days survival outcome. Many have demonstrated that pPCI reduces the risk of left ventricular free wall rupture. Many have demonstrated that the better survival outcome among patients treated with pPCI, even when need to be transported to a center with cath lab facilities. Considering the better survival outcome and less procedural complications and decreasing morbidity, the UK government is considering to establish a national primary angioplasty service for patients with acute myocardial infarction. Our present patient demonstrated that the rigid control of CAD risk factors by regular exercise, complete withdrawal of smoking and anti-platelet and lipid lowering agents are not only the key factors in the maintenance of stent patency, but can reduce plaque burden. Nicholls et al. has described male gender, diabetes, and a history of prior revascularization are strong independent predictors of atherosclerotic burden in coronary disease patients. Birgelen CV et al. documented that positive linear relation between LDL cholesterol and annual changes in plaque size, with an LDL value of 75 mg/dL predicting, on average, no plaque progression. HDL cholesterol shows an inverse relation with annual changes in plaque size. In the landmark COURAGE study of patient with Stable Angina, revealed that PCI in addition to optimal medical therapy reduced the prevalence of angina. Therefore, our present patient’s strong compliance to lifestyle modification and rigid cholesterol control by lipid lowering agent, may have caused regression of plaque burden.

**References:**
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**Figure 5:** Arrow indicated the regression of Ostio-Proximal LAD plaque

**Figure 5b:** Regression of same territory in 2008

**Figure 6a:** Re-look CAG 2008

**Figure 6b:** Re-look CAG 2008

**Figure 6:** Arrow indicated patent Proximal LAD Stent with good distal runoff

Regression of plaque
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