中文題目：心臟移植靜脈阻塞引發單獨後壁心肌梗塞及 aVR 導程 ST 波段上升
英文題目：vein graft occlusion induced non-ST Elevation AMI and aVR ST elevation
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Background:

aVR ST elevation has been described as an EKG sign of left main coronary artery occlusion. Other causes of aVR
ST elevation include LAD-P occlusion , MVD, diffuse subendocardial ischemia. Vein graft occlusion has not been described as a cause of lead aVR ST elevation.

Methods and Material : case report and literature review

Results:

A 72 years-old male patient visited ER for prolonged anterior chest pain during hemodialysis. The EKG showed ST depression in leads I, II, V 456, and ST elevation in lead aVR. High sensitivity troponin -I was elevated : 486.4 pg/ ml. Chest pain improved after supportive care. He has had insulin dependent diabetes mellitus and end stage renal disease for 2 decades. He received coronary bypass surgery for 3 vessel disease 12 years ago with LIMA to LAD and vein graft to RCA PL and LCX OM branch . There was atypical vague chest discomfort 5 years ago. Thallium stress LV perfusion scan had negative result that time. For the evaluation for acute chest pain with Thallium perfusion scan was done. It showed positive myocardial ischemia over multivessel territory. Coronary angiography showed patent LIMA and subtotal occlusion of sequential vein graft at the portion that supplied LCX artery. PCI was done for vein graft occlusion with successful DES implantation. The aVR ST elevation and ST depression over leads I , II ,V 456 was absent in folllow up EKG after PCI.

Discussion:

The EKG changes of aVR ST elevation may imply left main coronary LM , LAD-P , or MV dz. The patient had received complete revascularization for MVD. There was not any EKG sign of myocardial ischemia early after CABG. The current coronary image shows patent graft to LAD and patent vein graft to RCA. Only the blood supply to LCX was blocked. It is a pathophysiology of single vessel disease involving LCX artery. There is coexistance LCX territory flow obstruction and aVR ST elevation. There was no more aVR ST elevation on EKG after correction of vein graft obstruction.

The possible mechanisms of aVR ST elevation in LCX territory flow obstruction may be myocardial ischemia or myocardial infarct. The EKG changes of LCX territorry myocardial ischemia is usually ST depression in lead V456 , I, II. There has never been report of aVR ST elevation in LCX territorry myocardial ischemia. Theoredically aVR ST elevation may be the mirror image of ST depression in precordial leads. But there ST depression in LCX territory ischemia usually is minor. The ST depression in precordial leads is marked in this case. The other possible mechanism of ST depression in precordial leads is posterior wall myocardial infarction in which the ST depression is usually present at V1-V3 and is different from the EKG changes in this case.

The TNI enzyme had more than 10 times elevation in the acute event. The parallel evolutional EKG and enzyme changes support the diagnosis of acute myocardial infarct. There was no EKG sign of acute inferior or anterior wall myocardial infarct. The absence of EKG sign of inferior wall or anterior wall ST elevation MI support the diagnosis of a non ST elevation acute myocardial infarction. The CAG of this pt confirms the subtotal occlusion of vein graft to LCX. The ST elevation in lead aVR in this case should be the mirror image of ST depression in V4-V6 secondary to non -ST elevation AMI in LCX territory.

Conclusion:

The aVR ST elevation may occur in pt with occlusion of vein graft to LCX. The ST depression in leads V456 may be the EKG manifestation of an acute lateral wall non-ST elevation AMI . The mechanism of aVR ST elevation is mirror image of ST depression in precordial leads.