Lead aVR Saves the Day in the Diagnosis of Severe Multivessel and Left Main Coronary Artery Disease

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History of Presentation:

A 68-year-old one pack per day tobacco female smoker with Hypertension, Diabetes Mellitus, Coronary Artery Disease with previous stents, and Peripheral Arterial Disease with previous stents presents to the Emergency Department with severe constant retrosternal chest pain. Blood pressure on admission was 64/35mmHg, heart rate of 72bpm, and oxygen saturation of 97%. Cardiovascular exam revealed no JVD, no carotid bruit, no murmurs, S1, S2 regular, no edema, no cyanosis. No crackles or wheezes on lung auscultation.

Diagnostic tests included elevated serum troponin I at 16.300ng/ml (normal 0-.045ng/ml) on hospital admission. Elevated D-dimer 2.35mg (FEU)/L (normal <.5) on admission. Complete Blood Count with differential included low Hemoglobin:10.3, low Hematocrit: 33.0, mean corpuscular volume: 97.1, and platelet count: 167. Comprehensive metabolic panel included elevated Creatinine: 2.3, and elevated BUN :28.

Electrocardiogram (ECG) on hospital admission showed slight ST-segment elevation in lead aVR. Diffuse ST-depression in remaining leads II, III,V4,V5,V6 (**Figure1**).

Chest Radiograph on admission revealed evidence of cardiomegaly **(Figure2**).

Management:

On admission to the Emergency Room the patient was given Norepinephrine infusion due to hypotension. Due to the interesting ECG findings and elevated troponins, we became very suspicious of Left Main Coronary Artery Disease. Thus, the patient was brought urgently into the catheterization lab for cardiac support.

Cardiac coronary angiogram was performed less than 24 hours from hospital admission. Angiogram showed severe diffuse proximal, mid and distal left main coronary artery disease. In addition, there was both severe ostial Left Anterior Descending artery (LAD) stenosis and severe ostial left circumflex artery stenosis (**Figure 3a,b,c**).

She underwent an intra-aortic balloon pump procedure in the catheterization lab to stabilize the left ventricle heart and prevent further progression into cardiogenic shock. Patient was then transferred to the coronary care unit and started on Heparin treatment due to insertion of the intra-aortic balloon pump. Her blood pressure was 125/67mmHg, and heart rate of 93bpm with intra-aortic balloon pump. She will undergo cardiothoracic surgical evaluation for Coronary Artery Bypass Surgery (CABG).

Imaging:

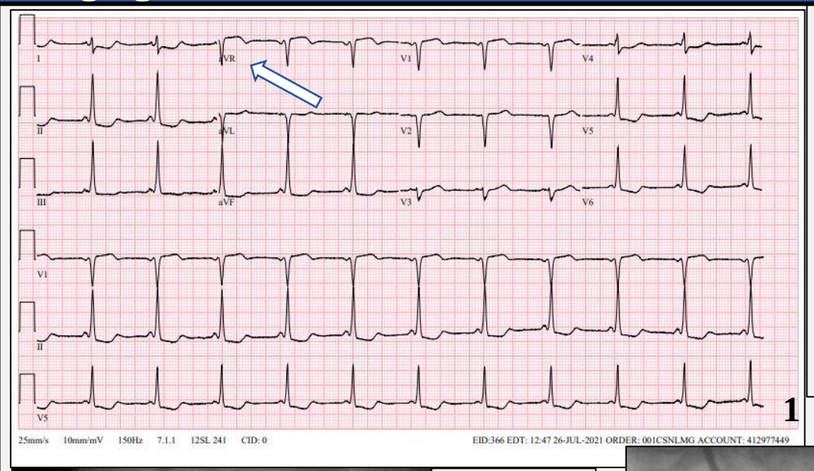


Figure 1: Electrocardiogram: showed slight ST-segment elevation in lead aVR. Diffuse ST-depression in remaining leads II, III,V4,V5,V6 **.** Electrocardiogram showed sinus rhythm with PR interval of 110ms, QRS duration of 94ms.

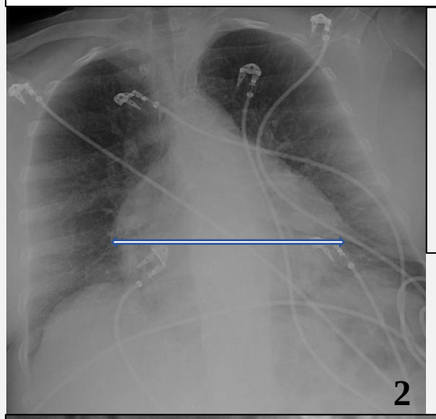
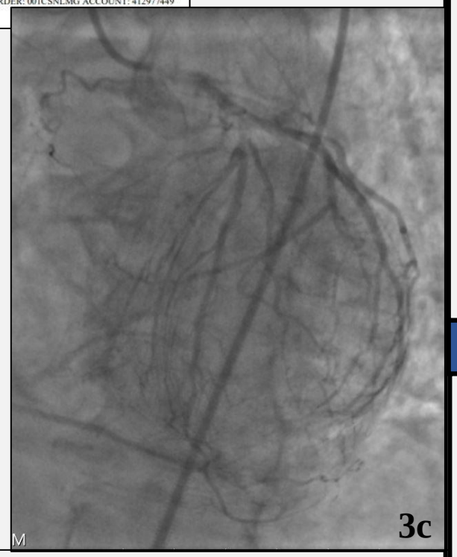
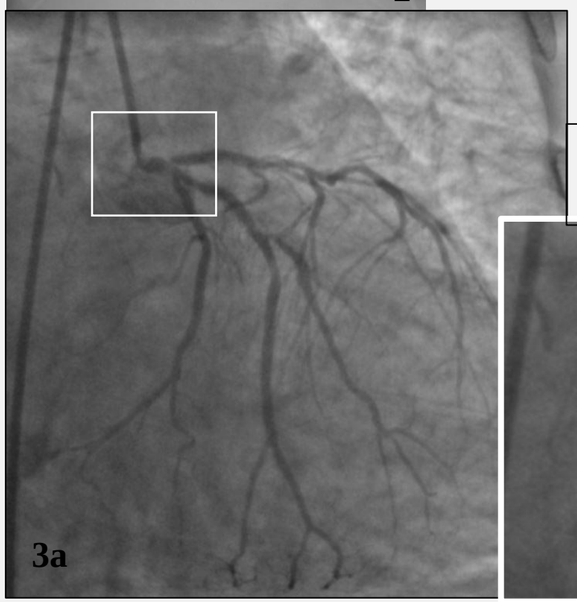


Figure 2: Chest Radiograph: On Hospital admission showed evidence of cardiomegaly.





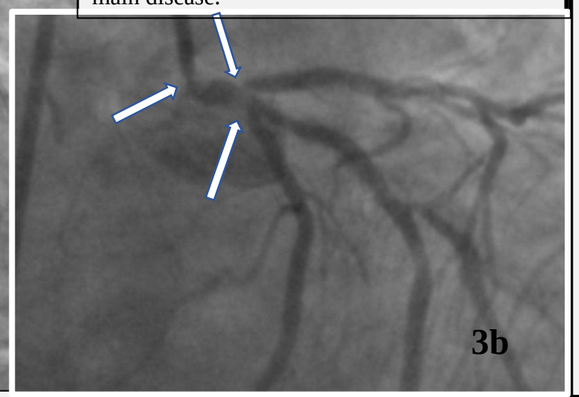


Figure 3a,b,c Cardiac coronary angiogram: Severe diffuse proximal, mid and distal left main disease.

Discussion:

On the electrocardiogram, the slight ST elevation changes in the aVR lead can lead to pertinent early clinical diagnosis of severe multivessel coronary artery disease.

A review by Ching[1] demonstrates the importance of lead aVR as an electrocardiographic predictor of left main or multi-vessel disease. Studies have found that the degree of ST elevation in lead aVR was the strongest predictor of severe Left Main/Three vessel disease, followed by elevated troponins[2]. ST segment elevation in lead aVR >/= 1.0mm had 80% sensitive and 93% specific for left main or triple-vessel disease in non-ST-segment elevation acute coronary syndrome patients [2]. In our patient, because of the evidence of slight ST elevations in lead aVR followed by elevated troponins, we elected to perform an urgent left heart catheterization and coronary angiogram. The patient's coronary angiogram reflected several multivessel coronary artery disease.

References:

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2. An early and simple predictor of severe left main and/or three-vessel disease in patients with non-ST-segment elevation acute coronary syndrome.
Kosuge M., Ebina T., Hibi K., et al.
Am J Cardiol. 2011; 107: 495-500
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