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LMCA Stenosis Diagnosis in the ER Using the AVR

(Case Report and Review of Literature)

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Abstract

Electrocardiography continues to be the jugular vein of modern medicine and it continues to be the most frequently ordered test in the Emergency Medicine Department. EKG, as a diagnostic tool is immensely valuable and assists the Emergency room (ER) physicians in diagnosing variety of life threatening conditions from Acute Coronary Syndrome (ACS) to Tricyclic antidepressant poisoning. We present to you a case report of how EKG helped in diagnosing Left Main Coronary Artery (LMCA) stenosis with 100% block in the ER with help of AVR lead in ACS setting and how prompt diagnosis and activation of Cathlab decreases mortality.

Keywords - LMCA stenosis, ST elevation, AVR lead, Emergent PCI,

1. INTRODUCTION

Identification of acute LMCA stenosis in ACS setting still continues to be a diagnostic challenge in the ED. With AVR continues to be the most ignored and patronized lead by many physicians including cardiologist, the onus lies on the ER docs to promptly pick up the subtle clues on the EKG and activate the catheterization Lab thereby decreasing the time of diagnosis which unfortunately is the only predictor for survival for LMCA stenotic patients.

Case report

A 56 year old male, smoker came to the ED with complaint of severe chest pain, shortness of breath and marked sweating. The chest pain was present since 4 to 5hrs was continuous, 10/10 on pain scale, localized to the precordial region, non-radiating. It was associated with light headedness and dyspnea. The pain was not positional and it did not have any associated aggravating or relieving factors. The patient didn't complain of any nausea, vomiting, abdominal pain or cough.

Past medical history is non-contributory except he is an active smoker with 2 packs per day since last 35yrs and diabetic since 15 yrs. Non-alcoholic except binge drinker on weekends. CAGE 0/4. He denies any drug abuse history. Family history father died at early age of 50 of MI and mother is alive.

On **Physical examination** patient was febrile and tachycardic and but hemodynamically stable. On cardiac examination, S1S2 present. No murmurs, gallop. JVP was not raised. No thrill or peripheral edema present. On respiratory examination, air

entry was bilateral and no rales , ronchi or basilar crepts heard. Per abdomen examination was normal.

On Arrival, Oxygen started and sublingual GTN was given , but the pain did not subside. EKG was done .

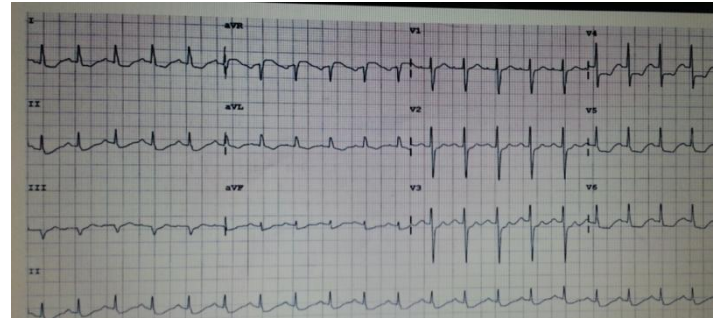


Figure 1 : EKG showing ST depression, in leads I, II and V4, V5, V6. ST elevation in AVR \geq 1mm. ST elevation in AVR \geq V1

.[Fig. 1] showed ST depression, most prominent in leads I, II and V4, V5, V6. ST elevation in AVR \geq 1mm. ST elevation in AVR \geq V1. Trop T was done and it was positive. Bed side 2D echo showed 20 % LVEF and Global hypokinesia. No sooner did the provisional diagnosis was made the patient clinical deteriorated to Systolic BP 90 and diastole not recordable cath lab was immediately activated and showed 100 % occlusion of the LMCA [Fig. 2]

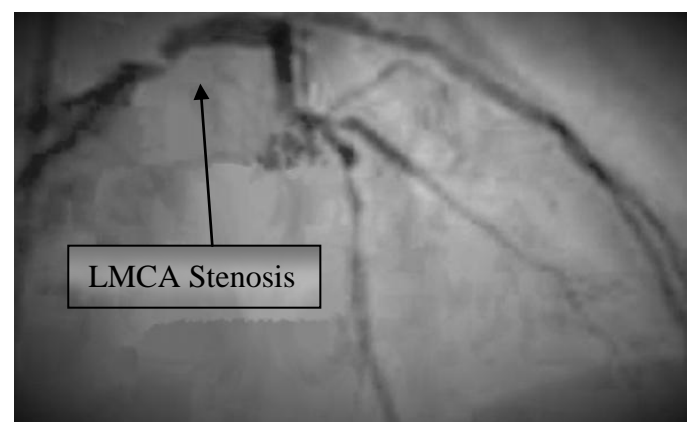


Figure 2. Coronary Angiography showing LMCA stenosis

2. DISCUSSION:

EKG plays a pivotal role in diagnosing Acute LMCA stenosis in the emergency medicine department. The AVR lead which is oftenly ignored helps in diagnosing the life threatening condition ranging from acute LMCA stenosis to PE to TCA poisoning. Lead AVR is electrically opposite to the left-sided leads I, II, aVL and V4-6; therefore ST depression in these leads will produce reciprocal ST elevation (STE) in AVR. Other theory is electrical activity from the right upper portion of the heart, including the right ventricular outflow tract and the basal portion of the interventricular septum is recorded by AVR. Infarction in this area could theoretically produce STE in AVR.

AVR lead is a patronized lead among the physicians hence they don't realise that , the predictive value of STE in AVR [Table 1] is so critical and accurate that it gives us the idea about which coronary vessel is blocked even before an Angiogram is done!! This greatly impacts the mortality and morbidity in LMCA stenosis patients. In the recent study by **Kosuge et al. (2011)**, STE in AVR ≥ 1 mm was a strong predictor of severe LMCA / 3Vessel Disease requiring CABG. Conversely, patients with < 1 mm ST elevation in AVR had a negligible risk of severe LMCA / triple vessel disease requiring CABG [1]

In the another study carried out by **Yamaji H et al** Lead AVR STE with less STE in lead V1 is an important predictor of acute LMCA obstruction and also contributes to predicting a patient's clinical outcome. The finding of lead AVR STE greater than or equal to lead V1 STE distinguished the LMCA

group from the LAD group (Left Anterior Descending coronary artery), with 81% sensitivity, 80% specificity and 81% accuracy [2]. Although thorough epidemiological studies are yet to be carried out in to know the prevalence and risk factors of association of LMCA stenosis and Ischemic heart diseases, a small study done in tehran indicates that the patients with LMCA disease were more likely to be male, elderly, and have diabetes mellitus or dyslipidemia, whereas cigarette smoking was found as an independent predictor of isolated LMCA. There was a strong correlation between the severity of LMCA stenosis and coexistent diseases in the rest of the coronary arteries [3]. LMCA stenosis can occur in isolation as in a case reported where the stenosis was due to anatomic anomaly [4] or in association with ST segment elevated myocardial infarction (STEMI) [5], [6].

Although in ACS setting , STE in AVR prompts us to think about the coronary artery stenosis , there's however two small caveats; first is that this rule isn't applicable in case of Supraventricular tachycardia (SVT) since ST changes are common in SVT and has no clinical relevance and secondly if the patient is asymptomatic.

Prompt diagnosis and Activation of Catheterization Lab is the only predictor for survival [1]. In the yet another study done by **Rokos et al** has proposed update for EKG criteria that enhance the rate of appropriate CathLab activation for acute MI **Table 2**. [10] Medical therapy including thrombolytics don't work. In the setting of ACS with Acute LMCA obstruction 70 % mortality without

immediate PCI. Emergent PCI may decrease the mortality to 40%.

Primary revascularization includes CABG and PCI. CABG is still considered to be gold standard.the two conduits for CABG is internal thoracic mammary artery (superior/long term patency) and Saphenous vein. **Kang et al** described similar mortality following LMCA PCI with drug-eluting stents (DES) compared to CABG although the risk of repeat procedures remains higher following PCI [7]

Coronary computed tomography (CT) angiography is being increasingly employed for the evaluation of coronary artery disease (CAD). Recent publications using 64-slice CT (1-2), and dual-source CT (3-4) have demonstrated high accuracy for the detection of coronary stenosis in comparison with invasive

angiography. [9]The most common PCI techniques are percutaneous transluminal coronary angioplasty and coronary stenting. PCI with bare metal stent or PCI with drug eluting stent. Although medically managed Unprotected LMCA have a 3yr mortality of 50 % , surgery still considered to be the treatment of choice. The 2 largest studies in the U.S. were the Emory Angioplasty versus Surgery Trial (EAST) and the Bypass Angioplasty Revascularization Investigation (BARI).Both trials have shown that CABG is superior to PCI in relieving angina and obviating the need for repeat revascularization procedures.

Table No 1:
Predictive Value of STE in AVR

1	STE in AVR \geq 1mm indicates proximal LAD / LMCA occlusion or severe 3VD
2	STE in AVR \geq V1 differentiates LMCA from proximal LAD occlusion
3	Absence of ST elevation in AVR almost entirely excludes a significant LMCA lesion

Table 2. Comparison of 2004 ACC/AHA guidelines and Rokos et al proposed update for EKG criteria that enhance the rate of appropriate CathLab activation for**Acute MI**

Indications for appropriate Cath Lab activation	Diagnostic criteria for patients with symptoms <12 h	2004 ACC /AHA guideline	Proposed update vs. ACC/ AHA guidelines	Comment
Classic STEMI	ST-elevation ≥ 1 mm in 2 contiguous leads V1-V4	Class I-A	Agree	ST-elevation ≥ 2 mm (men) and ≥ 1.5 mm (women) improves diagnostic specificity. ¹⁵ Presence of reciprocal changes (ST-depression in opposite leads) improves diagnostic specificity.
Inferior	ST-elevation ≥ 1 mm in 2 contiguous leads (II, III, or AVF)	Class I-A	Agree	Same as above
Lateral	ST-elevation ≥ 1 mm in 2 contiguous leads (I, AVL, V5, or V6)	Class I-A	Agree	Same as above
New or presumed new-onset LBBB	Presumed new” LBBB assumed when prior EKG unavailable” “New” LBBB when prior EKG available	Class I-A	Proposed demotion in future ACC/AHA guidelines	Unless clinically unstable, most LBBB should be evaluated with biomarkers and non-emergent angiography if indicated. An “old” EKG without LBBB does not necessarily confirm
Preexisting LBBB with Sgarbossa concordance	Concordance noted between QRS complex and ST/T-wave complex, with ST elevation ≥ 1 mm in ≥ 1 lead	None	Proposed addition to future ACC/AHA guidelines	Use of these decision criteria provides N95% specificity and avoids the need to find prior EKG for comparison. Discordant ST-elevation ≥ 5 mm is also a Sgarbossa criteria, but some studies found it a weak predictor
Posterior MI (isolated)	ST-depression ≥ 0.5 mm in leads V1-V3 Associated T-waves	Fibrinolytics: class IIa-C Primary PCI:	Proposed clarification in future ACC/AHA	Recent data ³⁴ demonstrated that most posterior MIs are currently evaluated with

	are either upright or inverted. Appearance of tall R-waves in V1-V2 may be delayed.	class I-A implied	guidelines	urgent (rather than emergent) angiography, but this delay is associated with worse clinical outcomes
Left Main coronary occlusion	ST-depression ≥ 1 mm in 6 or more leads Lead aVR with ST-elevation ≥ 1 mm ST-elevation in lead aVR \geq V1	None	Proposed addition to future ACC/AHA guidelines	Most relevant in any EKG with diffuse ST-depression ≥ 1 mm that does not meet classic STEMI criteria, thus providing a subtle clue that emergency angiography may be warranted
de Winter ST/T-wave complex	ST depression ≥ 1 mm up-sloping at the J-point in leads V1-V6 Precordial T waves are tall, upright, symmetric Normal QRS duration	None	Proposed addition to future ACC/AHA guidelines	Tall T waves and up-sloping ST depression are persistent, not transient. Precordial T waves are tall, upright, symmetric
Hyper-acute T-waves	Tall peaked T waves immediately follow symptom onset may represent acute ischemia	None	Proposed addition to future ACC/AHA guidelines	Generally prudent to perform serial EKGs, because true HATW generally morph quickly into a classic STEMI pattern

3. CONCLUSION

Acute LMCA stenosis in the setting of ACS is life threatening, when quickly picked up on EKG by ED physicians and prompt activation of Catheterization Lab is perhaps the only way of reducing the mortality and morbidity. EKG is the oldest pal of physicians without any ignorance and giving due importance to Lead AVR helps us in diagnosing Acute LMCA Stenosis and prevent a catastrophe.

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