# Introduction

Lead aVR has been long neglected until recent years. This is thought to be because most cardiologists have considered that the tracing of lead aVR merely reflects reciprocal information from the lateral limb and precordial leads. However, in the last decade, evidence indicating the importance of lead aVR in the field of acute coronary syndrome (ACS) has been accumulating ***(Kosuge, et al., 2011).***

Lead aVR, an augmented and unipolar limb lead, was constructed to obtain specific information from the right upper portion of the heart, including the outflow tract of the right ventricle and the basal portion of the inter- ventricular septum, which are supplied by the main stem of the left coronary artery (LM) and/or branches from the proximal parts of the left anterior descending artery (LAD); hence culprit lesions in these coronary segments cause ST-segment deviations in lead aVR. Due to the dominance of the basal ventricular mass, this should lead to ST-segment elevation (STE) in lead aVR, as the ST-segment vector in the frontal plane points in a superior direction ***(Kimura, et al., 2005).***

The left coronary artery mostly supplies approximately 75% of the left ventricular (LV) myocardial mass, acute occlusion of the left main trunk (LMT) causes life- threatening hemodynamic deterioration and malignant arrhythmias, resulting in an adverse outcome. Therefore, a rapid diagnosis and subsequent urgent revascularization with percutaneous coronary intervention (PCI) or coronary bypass surgery is very important in acute LMT occlusion ***(Goto, et al., 2011).***

ST-segment in lead aVR observed in acute LMT occlusion is caused by transmural ischemia in the basal part of the inter-ventricular septum through impaired coronary blood flow of the first major septal branch arising from the LAD and that smaller ST-segment elevation in lead V1 is due to the counterbalance of injury currents produced by transmural ischemia in both the anterior and posterior walls ***(Wong, et al., 2011).***

In acute LMT occlusion, ST-segment elevation in lead aVR can also occur as a mirror image of ST-segment depression in the lateral limb and precordial leads. For example, global sub-endomyocardial ischemia caused by acute LMT occlusion can produce widespread ST-segment depression, especially in the lateral precordial leads, resulting

in ST-segment elevation in lead aVR. The electrocardiographic findings of acute LMT occlusion into the following patterns: (1) widespread ST-segment depression with maximal changes in lead V4-6 with inverted T waves; (2) ST-segment elevation in lead aVR; and (3) anterior (anterolateral) ST-segment elevation. Ischemia-induced conduction disturbances, including right bundle branch block, left anterior fascicular block, and intra-ventricular conduction disturbance, are also frequently observed in acute LMT occlusion ***(Alherbish et al., 2013).***

Acute occlusion in not long LAD proximal to the origin of the first septal branch can produce ST-segment elevation in lead aVR through transmural ischemia in the basal portion of the inter-ventricular septum**.** However, it should be noted that the following conditions that can cause ST-segment elevation in lead aVR may disturb the theory: concomitant ischemia in the non-LAD region caused by multi-vessel disease, LV hypertrophy with strain pattern, and some types of conduction disturbances. The current evidence suggests that in anterior wall STEMI caused by LAD occlusion, the length of the LAD and the site of occlusion of the LAD can affect ST-segment in lead aVR ***(Anttila, et al., 2011).***

# Aim of the Work

The aim of this study is to evaluate the diagnostic value of ST segment elevation in lead aVR in acute coronary syndrome patients.

# Acute coronary syndrome (ACS)

Coronary artery disease, in which atherosclerotic plaque builds up inside the coronary arteries and restricts the flow of blood (and therefore the delivery of oxygen) to the heart. One woman or man experiences a coronary artery disease event about every 25 seconds, despite the time and resources spent educating clinicians and the public on its risk factors, symptoms, and treatment ***(Lloyd-Jones, 2009).***

Coronary artery disease can lead to acute coronary syndrome (ACS), which describes any condition characterized by signs and symptoms of sudden myocardial ischemia—a sudden reduction in blood flow to the heart. The term ACS was adopted because it was believed to more clearly reflect the disease progression associated with myocardial ischemia Unstable angina and myocardial infarction (MI) both come under the ACS umbrella ***(Torres, and Moayedi, 2007)***.

The signs and symptoms of ACS constitute a continuum of intensity from unstable angina to non–ST segment elevation MI (NSTEMI) to ST-segment elevation MI (STEMI). Unstable angina and NSTEMI normally result from a partially or intermittently occluded coronary artery,

whereas STEMI results from a fully occluded coronary artery ***(Anderson, 2007)***.

## Risk factors of coronary artery disease

Non-modifiable factors that influence risk for coronary artery disease include age, sex, family history, and ethnicity or race. Men have a higher risk than women. Men older than age 45, women older than age 55, and anyone with a first-degree male or female relative who developed coronary artery disease before age 55 or 65, respectively, are also at increased risk To.

Modifiable risk factors include elevated levels of serum cholesterol, low-density lipoprotein cholesterol, and triglycerides; lower levels of high-density lipoprotein cholesterol; and the presence of type 2 diabetes, cigarette smoking, obesity, a sedentary lifestyle, hypertension, and stress.

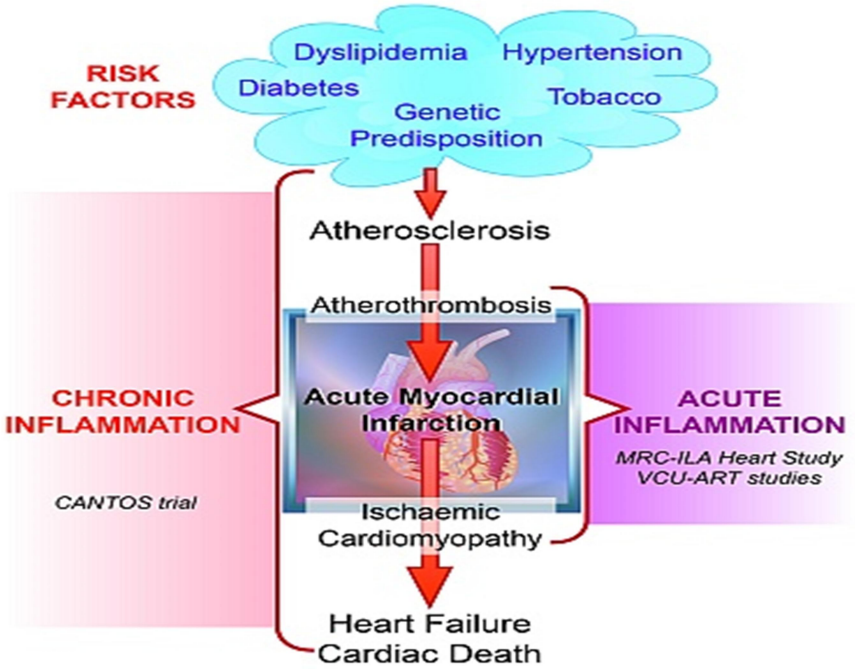


**Figure (1):** risk factors of ACS ***(Steg and James, 2012)***

## Pathophysiology of ACS

Acute Coronary Syndrome ACS begins when a disrupted atherosclerotic plaque in a coronary artery stimulates platelet aggregation and thrombus formation. It's the thrombus occluding the vessel that prevents myocardial perfusion ***(Torres, and Moayedi, 2007)***. In the past, researchers supposed that the narrowing of the coronary artery in response to thickening plaque was primarily responsible for the decreased blood flow that leads to

ischemia, but more recent data suggest that it's the rupture of an unstable, vulnerable plaque with its associated inflammatory changes.

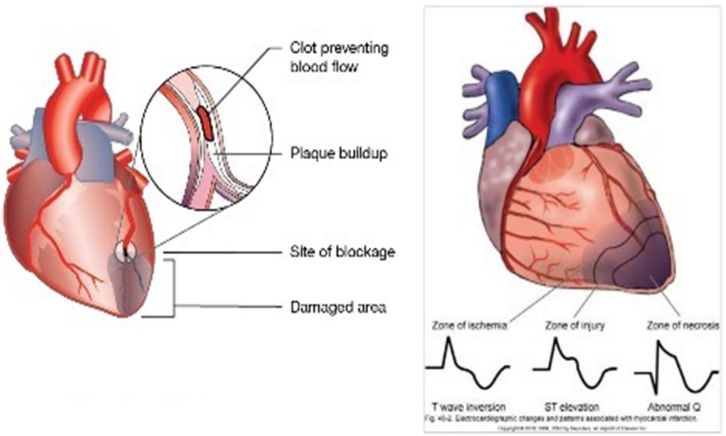


**Figure (2):** Acute coronary syndrome ***(Vale, et al., 2014)***

Myocardial cells require oxygen and adenosine triphosphate (ATP) to maintain the contractility and electrical stability needed for normal conduction. As myocardial cells are deprived of oxygen and anaerobic metabolism of glycogen takes over, less ATP is produced, leading to failure of the sodium–potassium and calcium pumps and an accumulation of hydrogen ions and lactate, resulting in acidosis ***(Woo and Schneider, 2009)***.

At this point, infarction-cell death will occur unless interventions are begun that limit or reverse the ischemia and injury. During the ischemic phase, cells exhibit both aerobic

and anaerobic metabolism. If myocardial perfusion continues to decrease, aerobic metabolism ceases and eventually anaerobic metabolism will be significantly reduced. This period is known as the injury phase***(Torres, and Moayedi, 2007)***.

If perfusion is not restored within about 20 minutes, myocardial necrosis results and the damage is irreversible. Impaired myocardial contractility, the result of scar tissue replacing healthy tissue in the damaged area, decreases cardiac output, limiting perfusion to vital organs and peripheral tissue and ultimately contributing to signs and symptoms of shock ***(Antman, 2008).***

Clinical manifestations include changes in level of consciousness; cyanosis; cool, clammy skin; hypotension; tachycardia; and decreased urine output. Patients who have experienced an MI are therefore at risk for developing cardiogenic shock.

In an attempt to support vital functions, the sympathetic nervous system responds to ischemic changes in the myocardium. Initially, both cardiac output and blood pressure decrease, stimulating the release of the hormones epinephrine and norepinephrine, which in the body's attempt

to compensate increase the heart rate, blood pressure, and afterload, ultimately increasing myocardial demand for oxygen ***(Goodacre, 2009).***

As oxygen demand increases at the same time that its supply to the heart muscle decreases, ischemic tissue can become necrotic. Low cardiac output also leads to decreased renal perfusion, which in turn stimulates the release of renin and angiotensin, resulting in further vasoconstriction.

Additionally, the release of aldosterone and antidiuretic hormone promotes sodium and water reabsorption, increasing preload and ultimately the workload of the myocardium ***(Torres, and Moayedi, 2007)***.

## Signs and symptoms

The degree to which a coronary artery is occluded typically correlates with presenting symptoms and with variations in cardiac markers and electrocardiographic findings. Angina, or chest pain, continues to be recognized as the classic symptom of ACS ***(O'Connor, et al., 2010).***

In unstable angina, chest pain normally occurs either at rest or with exertion and results in limited activity. Chest pain associated with NSTEMI is normally longer in duration and more severe than chest pain associated with unstable

angina. In both conditions, the frequency and intensity of pain can increase if not resolved with rest, nitroglycerin, or both and may last longer than 15 minutes.

Pain may occur with or without radiation to the arm, neck, back, or epigastric area. In addition to angina, patients with ACS also present with shortness of breath, diaphoresis, nausea, and light headedness. Changes in vital signs, such as tachycardia, tachypnea, hypertension, or hypotension, and decreased oxygen saturation (SaO2) or cardiac rhythm abnormalities may also be present ***(Krumholz 2008).***

Atypical ACS symptoms. Many women present with atypical symptoms, resulting in delayed diagnosis and treatment. Women frequently experience shortness of breath, fatigue, lethargy, indigestion, and anxiety prior to an acute MI and may not attribute those symptoms to heart disease.

It's also important for clinicians to realize that women tend to experience pain in the back rather than substernally or in the left side of the chest and do not characterize it as pain, but may instead report a numb, tingling, burning, or stabbing sensation ***(Torres, and Moayedi, 2007)***.

In fact, a recent study found that, when compared with men, women diagnosed with ACS more often reported

indigestion, palpitations, nausea, numbness in the hands, and atypical fatigue than chest pain.

## Diagnosing ACS:

The patient's clinical history, presenting symptoms, biomarker levels, and electrocardiographic results are all evaluated. Cardiac biomarkers. Injured myocardial cells release proteins and enzymes known as cardiac biomarkers into the blood.

These markers help practitioners determine whether the patient is having or has recently had an acute MI (either an NSTEMI or a STEMI). The utility of various biomarkers is determined by the timing and duration of their elevation as well as by the extent of their cardiac specificity ***(Blankenship and Skelding, 2008).***

The cardiac troponins, troponin T and troponin I, are the most cardiac-specific biomarkers. These structural proteins are not normally found in serum; therefore elevated serum levels may predict the degree of thrombus formation and micro-vascular embolization associated with coronary lesions.

Levels of troponins I and T increase within four to six hours of myocardial injury; troponin I levels remain elevated

for four to seven days, and troponin T levels remain elevated for 10 to 14 days.

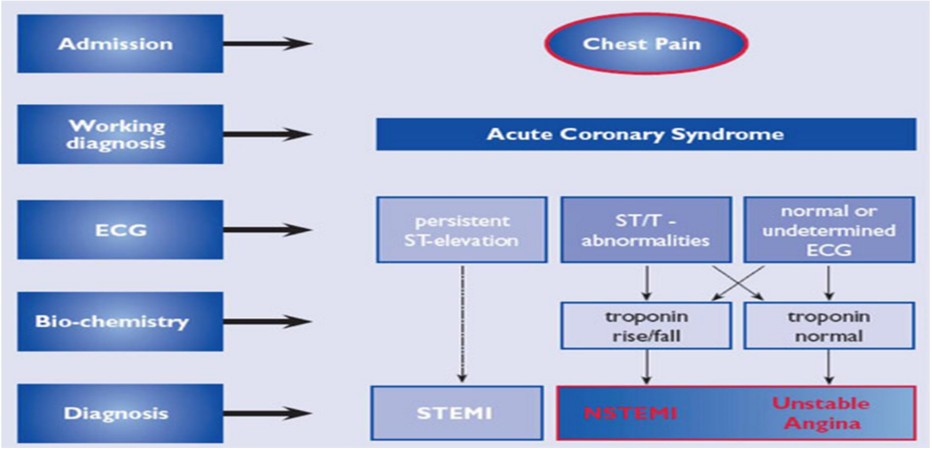


**Figure (3):** Diagnosis of acute coronary syndrome ***(Shinozaki , Tamura and Kadota, 2011)***

Normal reference ranges for cardiac biomarkers vary among laboratories; in order to diagnose myocardial necrosis a single troponin elevation greater than the 99$^{th}$ percentile of an agreed-upon reference control group is required.

Cardiac troponins are the preferred biomarkers for diagnosing acute MI because elevated levels correlate with a more accurate diagnosis, predict a high risk of future cardiac events even when levels of the myocardium-specific biomarker creatine kinase-MB (CK-MB) are normal or only mildly elevated, and elicit fewer false positives when concurrent skeletal muscle injury is present (after trauma or

surgery, for example). But if a laboratory is unable to process troponins, CK-MB is considered a reasonable alternative ***(Janda, 2009)***.

CK-MB is a cardiac-specific enzyme that's released within four to six hours of injury and remains elevated for 48 to 72 hours after injury.

Two consecutive levels of CK-MB greater than the 99th percentile of a reference control group contribute to the diagnosis of acute MI. Myoglobin, a heme protein, is not cardiac specific, yet it's still considered a valuable biomarker because it's the first to rise after myocardial damage**.**

If a patient presents with ACS symptoms that started less than three hours earlier, CK-MB and troponin levels may not yet be elevated. In such a case, myoglobin can rule out or lead to an early diagnosis of acute MI and prompt decisive therapy ***(Montalescot G, 2009)*.**

## Electrocardiographic findings

The AHA and the ACC recommend that a 12-lead electrocardiogram (ECG) be performed in patients with symptoms consistent with ACS and interpreted by an experienced physician within 10 minutes of ED arrival.

Findings on a 12-lead ECG help the practitioner to differentiate between myocardial ischemia, injury, and infarction; locate the affected area; and assess related conduction abnormalities ***(Vale, N, 2014).***

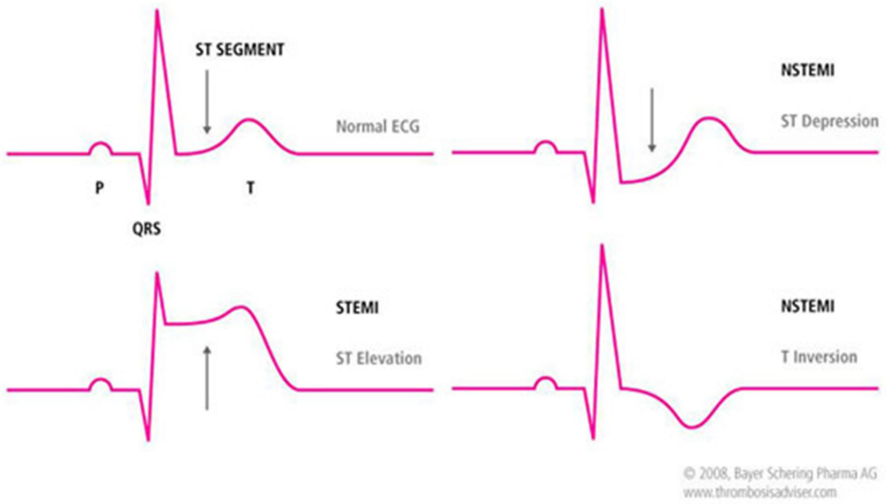


**Figure (4):** Electrocardiographic ***(Popma. Coronary arteriography. In: Bonow , Mann, Zipes, Libby and Braunwald, 2012)***

Electrocardiographic findings reflective of unstable angina or NSTEMI include ST-segment depression and inverted T waves. ST depression will normally resolve when the ischemia or pain has resolved, although T-wave inversion may persist.Providers should review electrocardiographic findings as well as levels of cardiac biomarkers to distinguish between unstable angina and NSTEMI ***(Hansson GK, 2005).***