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Sapan Bansriar
Nursing Officer, Cardiology
Department (ICCU), Jiwaji
University, All India Institute
of Medical Sciences Raipur,
Chhattisgarh, India

Anterior wall MI (Myocardial infarction)

Sapan Bansriar

Abstract

Anterior myocardial infarction (AMI) is a common heart disease associated with significant mortality and morbidity. Advancement in diagnosis and treatment options have led to a favourable outcome. This activity reviews the evaluation and management of anterior myocardial infarction and highlights the role of the interprofessional team in the care of patients with this condition.

Keywords: myocardial infarction, anterior myocardial infarction, anterior wall MI

Introduction

An anterior wall myocardial infarction occurs when anterior myocardial tissue usually supplied by the left anterior descending coronary artery suffers injury due to lack of blood supply. An anterior wall myocardial infarction may also be known as anterior wall MI (AWMI) or anterior ST segment elevation MI (anterior STEMI). When an AWMI extends to the septal and lateral regions as well, the culprit lesion is usually more proximal in the LAD or even in the left main coronary artery. This large anterior myocardial infarction is termed an extensive anterior.

Classification of anterior myocardial infarction is based on EKG findings as follows:

1. Anteroseptal – ST-segment elevation in leads V1 to V
2. Anteroapical (or mid-anterior) – ST-segment elevation in leads V3-V4
3. Anterolateral – ST-segment elevation in leads V3 to V6
4. Extensive anterior – ST-segment elevation in leads V1 to

The ECG findings of an acute anterior myocardial infarction wall include:

1. ST segment elevation in the anterior leads (V3 and V4) at the J point and sometimes in the septal or lateral leads, depending on the extent of the MI. This ST segment elevation is concave downward and frequently overwhelms the T wave. This is called “tombstoning” for obvious reasons; the shape is similar to that of a tombstone.
2. Reciprocal ST segment depression in the inferior leads (II, III and aVF).

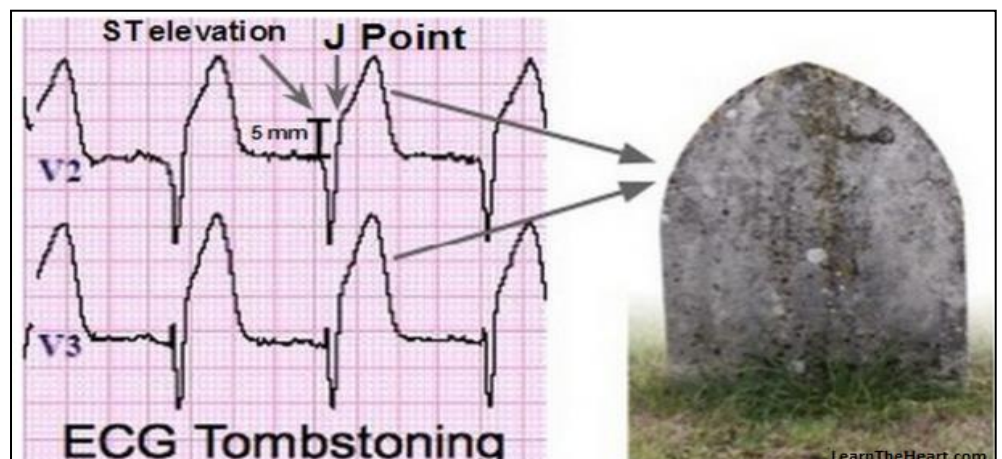


Fig 1: ECG Tombstoning

Corresponding Author:
Sapan Bansriar
Nursing Officer, Cardiology
Department (ICCU), Jiwaji
University, All India Institute
of Medical Sciences Raipur,
Chhattisgarh, India

According to the American College of Cardiology/American Heart Association guidelines for STEMI, there must be “new ST segment elevation at the J point in at least two contiguous leads of ≥ 2 mm (0.2 mV) in men or 1.5 mm (0.15 mV) in women in leads V2-V3 and/or of ≥ 1 mm (0.1 mV) in other contiguous chest leads or the limb leads.” This

means 1 millimeter in any two contiguous leads, except leads V2 or V3, where the elevation must be 2 mm in men or 1.5 mm in women.

See the full 12-lead ECG example below and a few more at the bottom.

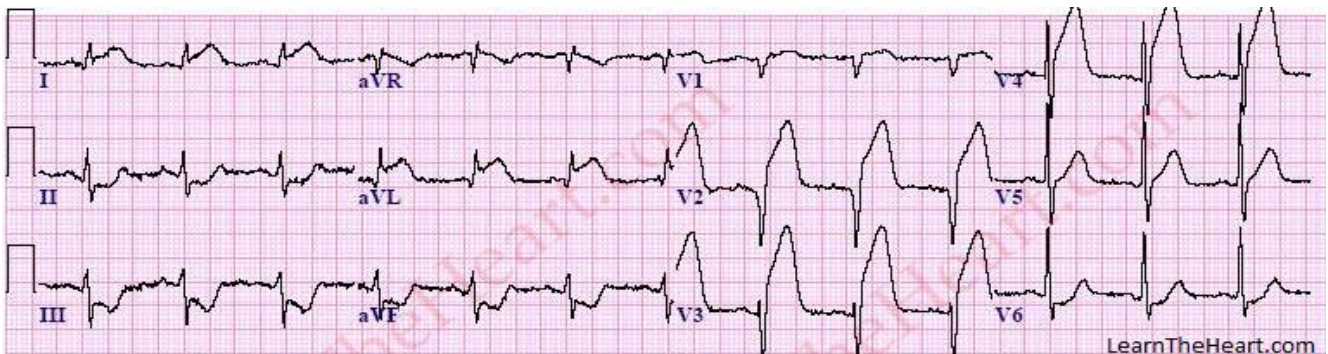


Fig 2: ECG Old anterior wall MI with poor R wave progression

The ECG findings of an old anterior wall MI include the loss of anterior forces, leaving Q waves in leads V1 and V2.

This is a cause of poor R wave progression, or PRWP. Here is an example of an old anterior wall MI:

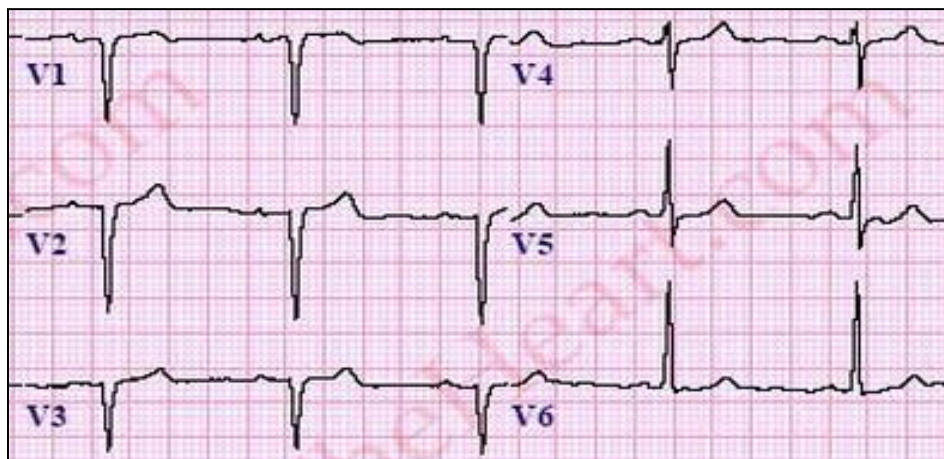


Fig 3: ECG Old anterior wall MI with poor R wave progression

Note: To distinctly say that an old anterior wall MI is present on the ECG, there must be no identifiable R wave in lead V1 — and usually V2, as well. If there is an R wave in V1 or V2, the term poor R wave progression, but not old anterior wall MI, can be used.

seen in V1 and/or V2, indicating a ventricular aneurysm — a known complication of a myocardial infarction. Visit Left Ventricular Aneurysm ECG Review or Left Ventricular Aneurysm Topic Review. An example of an old anterior myocardial infarction with a left ventricular aneurysm is below.

On rare occasions, persistent ST segment elevation may be

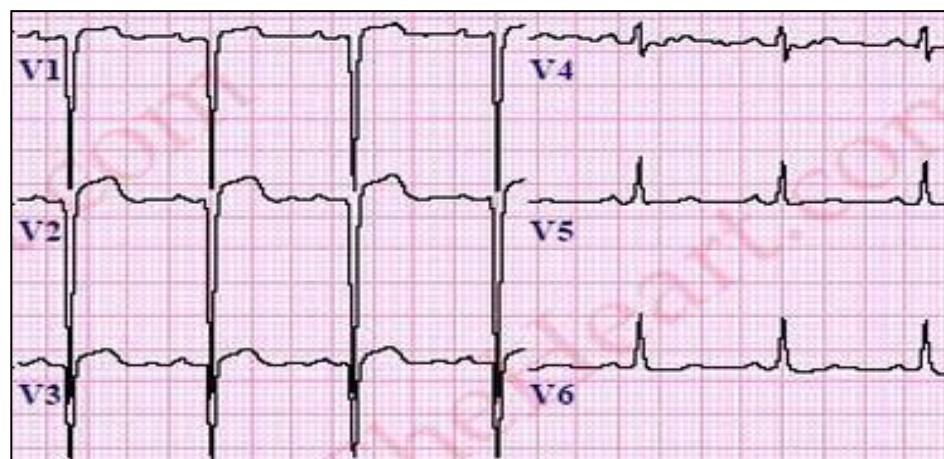


Fig 4: ECG old anterior myocardial infarction with a left ventricular aneurysm

Case Report

Diagnosis: Type 2 Diabetes Mellitus, Hypertension, Acs- Anterior Wall Mi (Non Thrombolysed), Nsr, Normal Lv Size & Function, Lvef ~ 51%

History & Examination

This patient diabetic (recently diagnosed), non hypertensive, with no family h/o CAD, presented with c/o typical chest pain of 2 hours duration on 08.08.2020 & consulted a nearby hospital, diagnosed as CAD– ACS, ANTERIOR WALL MI where loading dose was given and referred here for further management. Patient came to AIIMS Raipur on 10.08.2021, 2 days after the onset of ACS-AWMI (outside window period). No H/o syncope or palpitation. O/E BP= 114/68 mmHg, Pulse = 77 Bp/m, regular, JVP- Normal, CVS- S1,S2 normal, No S3/S4, no murmurs. R/S- NVBS present. No crackles. Patient was admitted for management and coronary angiogram.

ECG: NSR, NORMAL AXIS, short PR interval, Delta wave present in I, Avl, V2-V6, Negative wave in lead III, ST coved in V2-V3, T wave inversion I, Avl, V2-V6

Echo at Admission: Normal Lv Size & Function, Lvef ~ 51%, Apex Akinetic, Lv Thrombus Present At Apex (2.7 X 1.9cm), Distal Septum Hypokinetic, Mild To Moderate Mr, Trivial Tr (Rvsp 19 Mmhg), No Ar, Ra & Rv- Normal Size

ECHO AT Discharge: Normal LV size and contractility, No RWMA, LVEF – 62%, Mild MR (MR jet area – 3.9cm², Mild TR, TRvmax- 2.74 m/s, TRG- 30.04 mm Hg, RVSP – 35.04 mm Hg), NO LV APICAL CLOT AT DISCHARGE

Operation Details: CAG

CAG Report: TRIPLE VESSEL DISEASE

Hospital Course: Patient was managed with DAPT, Statin, antidiabetics, antihypertensive and other supportive measures.

Status AT Discharge: Stable

Future plan – OMT (Revascularisation, IF Symptomatic)

Investigations

Cardiac Viability Study: 1) Severely reduced to negligible perfusion with preserved metabolism is noted in the apex, apical anterior, apical septal and mid and basal antero, infero septal segments of LV myocardium- viable myocardium.

2) Severely reduced perfusion and metabolism is noted in the apical inferior segment of the LV myocardium- Non viable myocardium.

Table 1: Biochemistry Report

CBC		RFT		LFT		Thyroid profile		Viral marker	
TRBC	6.79million/ul	Urea	22mg/dl	AST	363U/L	TSH	4.2mIU/L	HIV	NR
HB	10.9gm%	Creat	1.17mg/dl	ALT	61U/L			HBsAg	NEG
TLC	13440/ul	UA	4.1mg/dl					HCV	NEG
Platelet	296000/ul	Na	136mEq/L					COVID-19	NEG
Urine R/M	WNL	K	4.3mEq/L						

Table 2: Lipid Profile

Lipid profile		Blood sugars		Coagulation profile		Others	
T. Chol	144mg/dl	RBS	145mg/dl	PT	14.5sec	TROP I	>50ng/mL
TG	104mg/dl	FBS	104mg/dl	INR	1.4		
HDL	28mg/dl	PPBS	158mg/dl	aPTT	40.3sec		
VLDL	21mg/dl	HBA1C	10.7%				
LDL	96mg/dl						

Systemic Examination

CNS- Conscious

RS- Within normal limits

P/A- Within normal limits

Discussion

- ST-elevation in leads V1-V6, I and aVL. Maximum elevation in V3, maximal depression in III
- Later: pathological Q-wave in the precordial leads V2 to V4-V5.

Conclusion

Studies have demonstrated that the prognosis of patients with anterior MI is worse when compared to those with inferior or posterior MI. Patients with anterior MI usually have a complicated hospital course as compared to inferior/posterior MI. Anterior MI is associated with an increased incidence of acute heart failure, ventricular fibrillation, and death. Following discharge, patients with

anterior MI correlated with poor long-term prognosis. Anterior MI associated with right bundle branch block (RBBB) predicts poor prognosis.

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