Review Article:
Electrocardiographic localization of infarct related coronary artery in acute ST elevation myocardial infarction

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ABSTRACT

The electrocardiogram (ECG) remains a crucial tool in the identification and management of acute myocardial infarction (MI). A detailed analysis of patterns of ST-segment elevation may influence decisions regarding the use of reperfusion therapy. The early and accurate identification of the infarct-related artery on the ECG can help predict the amount of myocardium at risk and guide decisions regarding the urgency of revascularization. The specificity of the ECG in acute MI is limited by individual variations in coronary anatomy as well as by the presence of preexisting coronary artery disease, particularly in patients with a previous MI, collateral circulation, or previous coronary-artery bypass surgery. The ECG is also limited by its inadequate representation of the posterior, lateral, and apical walls of the left ventricle. Despite these limitations, the electrocardiogram can help in identifying proximal occlusion of the coronary arteries, which results in the most extensive and most severe myocardial infarctions.

Myocardium is usually supplied by three coronary arteries, although there are several variations in the number, origin, course and distribution of coronary arteries. Major contribution to left ventricular myocardial blood flow is by left anterior descending coronary artery (LAD) (50%), rest is equally contributed by right coronary artery (RCA) and left circumflex artery (LCx). In addition, most of the right ventricle is supplied by RCA. The need for a rapid reperfusion therapy is largely determined by how close the occlusion site is to the origin of the coronary artery, which corresponds to the area of ischaemic myocardium.

Each artery contributes its blood supply to specific regional areas in the heart. These areas are topographically represented by the following groups of leads:[1] (Table 1).

MYOCARDIAL DISTRIBUTION OF THREE MAIN CORONARY ARTERIES

Left anterior descending artery

The LAD travels along the anterior interventricular groove towards the apex of the heart. The major branches of LAD are septal and diagonal branches.

The septal branches arise perpendicularly from the LAD and pass into the interventricular septum. The diagonal branches of the LAD course over the anterolateral aspect of the heart. Considerable variations exist in the number and size of the diagonal branches. In most (80%) patients, the LAD courses around the apex of the left ventricle and terminates along the diaphragmatic aspect of the left ventricle. In the remaining patients, the LAD terminates either at or before the cardiac apex. In these patients, the left ventricular apical portion is supplied by the posterior descending branch (PDA) of the RCA or LCx, which is larger and longer than usual.

Left circumflex artery

The LCx artery passes within the left atrioventricular groove toward the inferior interventricular groove. The LCx artery is the dominant vessel in 15% of patients, supplying the left PDA from the distal continuation of the LCx. In the remaining patients, the distal LCx varies in size and length, depending on the number of posterolateral branches supplied by the distal RCA. The major

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branches of LCx are obtuse marginals, which vary from one to three in number and supply the lateral free wall of the left ventricle.

Right coronary artery

The RCA after originating from the right anterior aortic sinus, courses along the right atrioventricular groove toward the crux. The conus artery arises at the right coronary ostium and is usually the first branch of the RCA. The second branch of the RCA is usually the sinoatrial node artery. This vessel arises from the RCA in about 60% of patients, and from the LCx artery in under 40%, and from both arteries with a dual blood supply in the remaining cases. The midportion of the RCA usually gives rise to one or several medium-sized acute marginal branches which supply the anterior wall of the right ventricle. The RCA divides at the crux into a PDA and one or more right posterolateral branches.

Coronary artery dominance

The right dominant circulation is defined as RCA supplying the PDA and at least one posterolateral branch. This type is present in around 85% of patients. The nondominant RCA is seen in 15% of patients. One half of these patients have left dominant circulation which is defined as distal LCx supplying a left PDA and left posterolateral branches. In these cases, the RCA is very small, ends before reaching the crux, and does not supply any blood to the left ventricular myocardium. The remaining patients have balanced/codominant circulation

which is defined as RCA giving rise to the PDA and LCx artery providing all the postero-lateral branches.

BASIS OF ELECTROCARDIOGRAM CHANGES

The electrocardiogram (ECG) is a key investigation in diagnosing acute ST-segment elevation myocardial infarction (STEMI). During acute transmural ischaemia, one of the important determinant of the site of coronary artery occlusion is the direction of the vector of ST-segment deviation.[3] The injury vector is always oriented toward the injured area. The lead facing the injury vector head shows ST-segment elevation and the lead facing the vector tail (opposite leads) shows ST-segment depression.

How to measure ST deviation?

Standard 12-lead ECG should be recorded at a paper speed of 25 mm/s and a voltage of 10 mm/mV at the time of admission. ST-changes should be measured 60 msec[4] or 80 msec from J point in all the leads. Measurements are to be taken to the nearest 0.5 mm (0.05 mV). The TP-segment should be used as the isoelectric line.

Inferior wall myocardial infarction

Inferior wall MI (IWMI) may be caused by occlusion in the course of either the right coronary artery (in 80% of the cases) or the LCx artery. The important features favouring right coronary artery rather than the LCx artery as the culprit artery in IWMI are ST-segment elevation in lead III

Table 1 : Coronary anatomy

Left anterior descending artery

The left anterior artery supplies anterior, anteroseptal or anterolateral wall of the LV (leads V1-V6, I, and aVL)

Right coronary artery

The right coronary artery supplies the inferior wall (leads II, III and aVF) and often the posterolateral wall of the LV (special leads V7-V9). The right coronary artery is the only artery that supplies the right ventricular free wall(special leads V3R to V6R)

Left circumflex artery

The left circumflex artery supplies the anterolateral (leads I, aVL, V5 and V6)and the posterolateral (leads V7-V9) walls of the LV. In 10%-15% of patients, it supplies the inferior wall of the LV.

LV = left ventricle

> lead II and more than 1 mm ST-segment depression in leads I and aVL[5,6] (Figure 1). In IWMI caused by RCA occlusion, injury vector is directed towards right (lead III), hence ST-segment elevation in lead III is greater than that in lead II.[7] An illustrative example of a patient with IWMI due to RCA occlusion is shown in (Figure 2).

One of the important clues that suggests proximal occlusion of the right coronary artery with associated right ventricular infarction in a case of IWMI is the additional finding of ST-segment elevation in lead V1.[8] Conversely, IWMI due to LCx occlusion produces an ST-segment vector directed toward the left (lead II). In this case, ST-segment elevation in lead III is not greater than that in lead II, and there is an isoelectric or elevated ST segment in lead aVL[9,10] (Figure 3). Associated ST-segment depression in leads V_1 and V_2 in a case of IWMI suggests concomitant posterior wall MI, which is usually caused by LCx occlusion but may also be seen in dominant RCA occlusion.[11]

It has been observed that various ECG and angiographic characteristics did have significant impact on sensitivity of ECG criteria for localising IWMI.[12] The ECG predicted RCA occlusion more reliably when more extensive ST-segment changes were present. Sensitivity was 93% among patients with a preprocedural summed ST-segment deviation >18.5 mm Vs 44% among patients with a
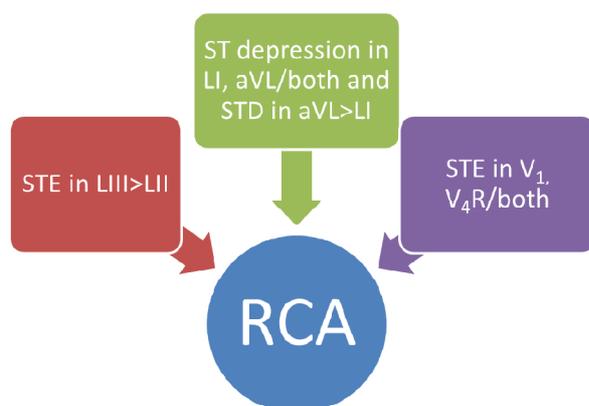

Figure 1: Electrocardiographic predictors of right coronary artery occlusion in inferior wall myocardial infarction(IWMI).
STE=ST-segment elevation;
STD=ST-segment depression

summed ST-segment deviation <18.5 mm (p<0.001). Patients with a dominant RCA showed a significantly higher sensitivity compared with patients with a non-dominant RCA (71 Vs 57%, p =0.04).[12]

In addition, it was observed that patients with acute RCA occlusion and less extensive ST-segment deviation showed significantly more often similar ST elevation in leads II and III, which thus resulted in a 'false-negative' ECG algorithm. In accordance with the higher algorithm sensitivity in patients with an extensive summed ST-segment deviation, the sensitivity of a 12-lead ECG was higher if recorded sooner after symptom onset. This is because of diminishing injury currents and decreasing ST-segment changes due to electrical uncoupling of individual cardiomyocytes in the affected area with persistent ischaemia.[13]

**Right ventricular myocardial infarction**

When RCA occlusion occurs proximal to the right ventricular (RV) branch, the right ventricle will be in jeopardy and subsequently infarcted. This concept is the basis for using the ECG findings of RV infarction as an indicator of proximal RCA occlusion.[14] Hence, RVMI is always associated with occlusion of the proximal RCA. As discussed above, ST-segment elevation in lead V_1 in association with ST-segment elevation in inferior leads (with greater elevation in lead III than in lead II) is highly correlated with the presence of RVMI[5,15] (Figure 1).

ST-segment elevation of more than 1 mm in lead V_4R with an upright T wave in that lead is the most sensitive ECG sign of RVMI. This sign is rarely seen more than 12 hours after the infarction.[16,17] As this lead is positioned on the right hemithorax, it might better monitor injury currents from the right side of the heart when compared with extremity lead III and may thereby reduce the amount of patients with an RCA occlusion and a 'false-negative' ECG algorithm due to similar ST elevation in leads II and III. When using lead V_4R in inferior myocardial infarction, ST-segment de-
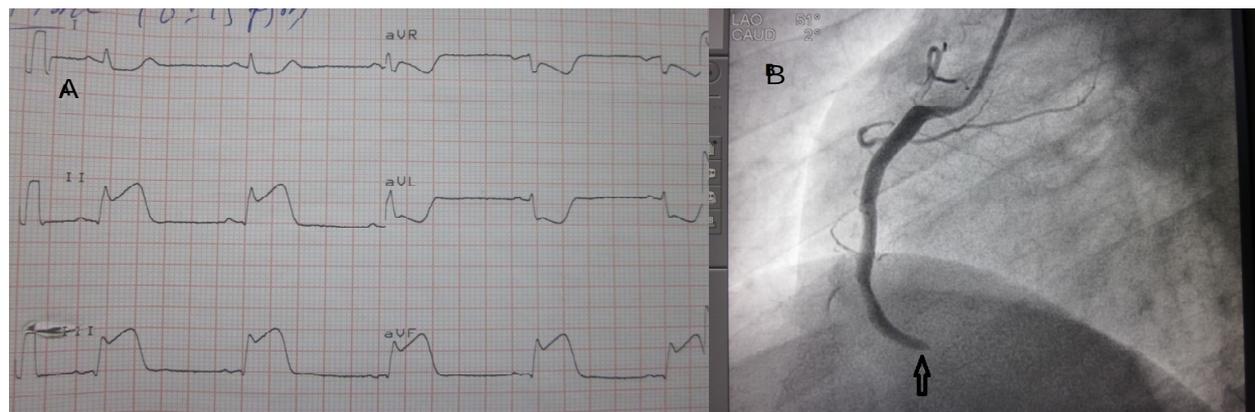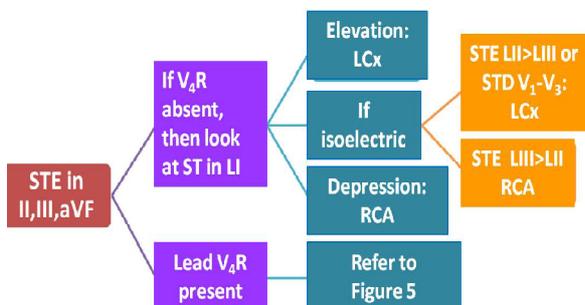
Figure 2: Inferior wall MI: ECG showing ST elevation in inferior leads with ST elevation in lead III>II and ST depression in LI and aVL suggestive of RCA occlusion
(**A**) Right coronary angiogram of the same patient showing total occlusion (arrow) of RCA in the distal part
(**B**) ECG=electrocardiogram; RCA=right coronary artery

viation in that lead does not last as long as in the standard extremity leads.[18] Hence, strong emphasis should be laid on recording lead V_4R as early as possible after the onset of symptoms. Unfortunately, although known for three decades, lead V_4R is rarely recorded in the real world in patients with STEMI.

Lead V4R is helpful in differentiating proximal versus distal RCA occlusion versus LCx occlusion by noting the presence or absence of convex upward ST-segment elevation in V_4R and by identifying whether the T wave is positive or negative in V_4R (Figure 4).[19] Occasionally, ST-segment elevation in leads V_2 and V_3 results from acute right ventricular infarction, thereby resembling anterior infarction; this appears to occur only when the injury to the left inferior wall is minimal.[20] Usually, the concurrent inferior wall injury suppresses this

anterior ST-segment elevation resulting from right ventricular injury. Similarly, right ven-tricular inf-arction appears to reduce the anterior ST-segment depression often observed with inferior wall myocardial infarction. A QS or QR pattern in leads V_3R and/or V_4R also suggests right ventricular myocardial necrosis but has less predictive accuracy than ST-segment elevation in these leads.[21]

Anterior wall myocardial infarction

The amount of LV myocardium at risk of infarction in a case of AWMI depends largely on the site of occlusion in the course of LAD. Therefore knowing the site of LAD occlusion with the help of ECG criteria in the emergency room is of immense help to the treating physician to decide about the plan of further management. It has been ob-
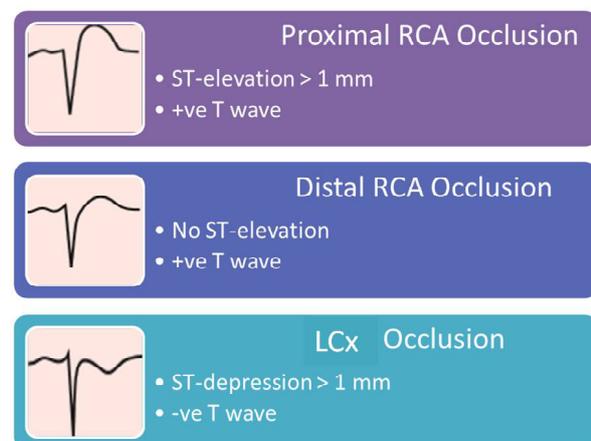

Figure 3: Algorithm for predicting infarct related artery in inferior wall myocardial infarction
STE=ST-segment elevation, STD=ST-segment depression, RCA=right coronary artery, LCX-left circumflex artery.

Figure: 4 ST-T changes in lead V_4R to predict infarct related artery
RCA=right coronary artery, LCx=left circumflex artery
Source: reference 19

served that quality of care in STEMI patients in the emergency room in whom high-risk ECG criteria were not identified was lower.[22] This highlights the importance of system changes to enhance the accuracy of ECG interpretation. Knowing that the occlusion is proximal or distal to first diagonal (D1) or first septal (S1) may be crucial for deciding on the best approach to treatment.

LAD occlusion may lead to a very extensive AWMI, or only septal, apical-anterior or mid-anterior MI depending on the location of occlusion.[23] Proximal LAD occlusion has been documented as an independent predictor of worse outcome related to increased mortality and recurrent MI,[24,25] and distal LAD occlusion is considered to have a favourable outcome.[25] Thus, an early identification of proximal LAD occlusion has crucial value not only from an academic standpoint, but also from a therapeutic point of view.
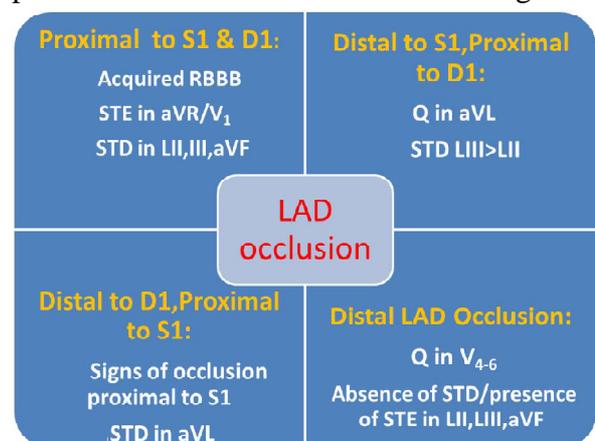
Precordial lead (V_1-V_6) ST-segment elevation in patients with symptoms suggestive of acute coronary syndrome (ACS) indicates STEMI due to LAD occlusion.[26-29] This information alone does not predict the extent of the jeopardized myocardium. ST segment changes in other precordial and frontal leads depends on the presence of ischaemia in three vectorially opposite areas, namely (i) basal septal area perfused by proximal septal branch; (ii) basolateral area perfused by 1st diagonal, and (iii) inferoapical area, when distal LAD wraps around apex

During acute AWMI, the maximal ST-segment elevation is best recorded in V_2 or V_3;[30] V_2 is the most sensitive lead to record ST-segment elevation (sensitivity 99%) and to identify the culprit lesion at the LAD. Lead V_1 captures electrical phenomena from the right paraseptal area, which has dual blood supply by the septal branches of the LAD and by a conal branch of the RCA. This is the reason why patients with AWMI usually have no ST-segment elevation in V_1.

The location of occlusion in the course of LAD, whether proximal or distal, can be identified by noting the changes in ST-segment of various leads

of surface ECG.[31-34] Indicators of proximal LAD occlusion on surface ECG are ST-segment elevation in V_1-V_3 and in lead aVL with added finding of ST-segment depression of more than 1 mm in lead aVF (Figure 5).[7,24,31] These findings are in correspondence with the direction of ST-segment vector which is upward, toward leads V1, aVL, and aVR, and away from the inferior leads. The most powerful predictors of proximal LAD occlusion include ST-segment elevation in aVL or aVR, concomitant ST-segment depression in inferior leads, ST-segment depression in V_5, and disappearance of preexisting septal Q waves in lateral leads.[24,31,35-38]

ST-segment elevation in leads V_1, V_2, and V_3 without significant inferior ST-segment depression suggests occlusion of the LAD beyond the origin of the first diagonal branch.[24] ST-segment elevation in leads V_1, V_2, and V_3 with concomitant elevation in the inferior leads suggests LAD occlusion distal to the origin of the first diagonal branch, in a vessel that wraps around the apex to supply the inferoapical region of the left ventricle. New right bundle-branch block with a Q wave preceding the R wave (QRBBB) in lead V_1 is a specific but insensitive marker of proximal LAD occlusion in association with AWMI[24] (Figure 5). An illustrative example of a patient with AWMI due to proximal LAD occlusion is shown in Figure 6.

Proximal to S1 & D1: Acquired RBBB STE in aVR/V_1 STD in LII,III,aVF	Distal to S1, Proximal to D1: Q in aVL STD LIII>LII
LAD occlusion	
Distal to D1, Proximal to S1: Signs of occlusion proximal to S1 STD in aVL	Distal LAD Occlusion: Q in V_{4-6} Absence of STD/presence of STE in LII,LIII,aVF

Figure 5 : Electrocardiographic predictors of location of occlusion in LAD in anterior myocardial infarction D1=first diagonal, S1=first septal, LAD=left anterior descending coronary artery, STD=ST segment depression, STE=ST segment elevation, RBBB=right bundle branch block.
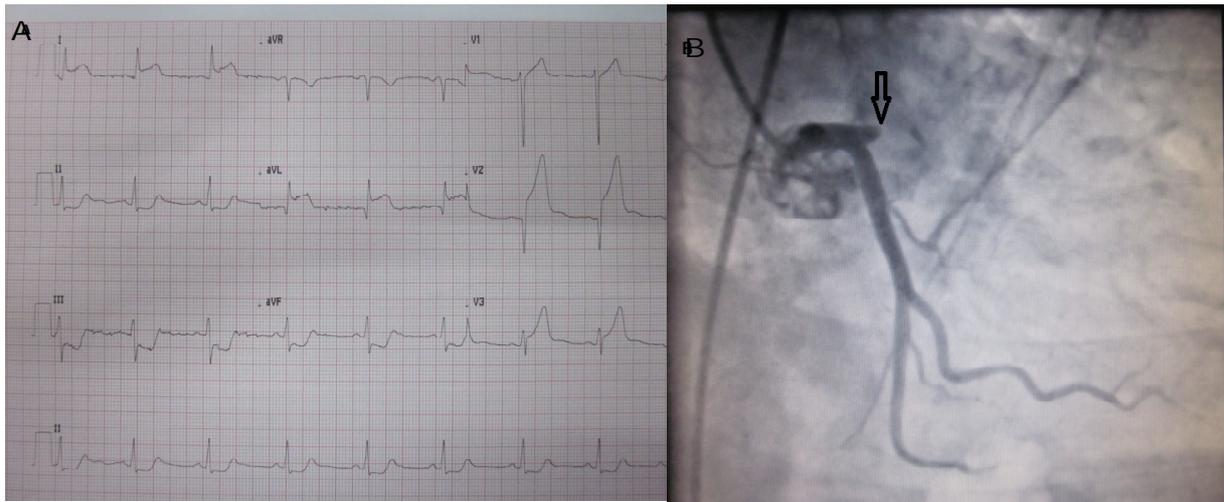
Figure: 6 Anterior wall MI. ECG showing ST elevation in leads V_2, V_3, I,aVL and ST depression in inferior leads suggestive of proximal LAD occlusion (**A**). Left coronary angiogram of the same patient confirming the site of occlusion (arrow) in the proximal LAD (**B**). ECG=electrocardiogram; LAD=left anterior descending artery
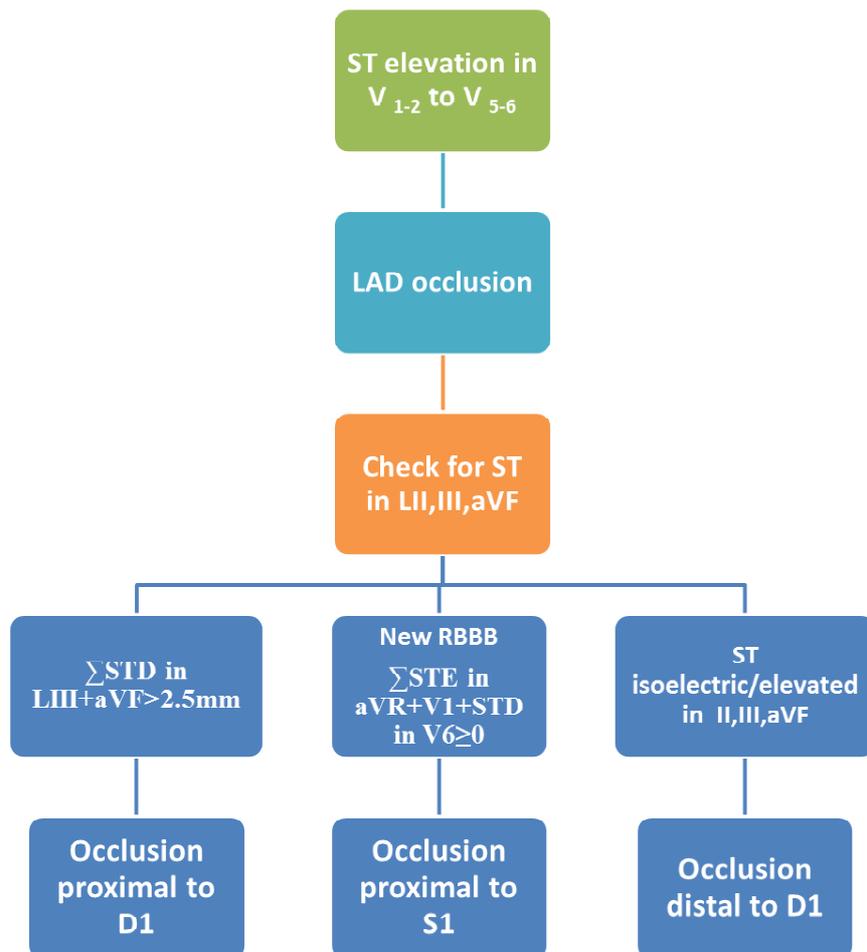
Figure: 7 Algorithm to precisely locate the site of Left anterior descending coronary artery occlusion in the case of evolving myocardial infarction with ST elevation in precordial leads
LAD=left anterior descending coronary artery, RBBB=right bundle branch block, D1=first diagonal, S1=first septal, STD=ST segment depression, STE-STsegment elevation, Σ = sum of
Source: reference 4

Nevertheless, it is better to have an algorithm based on deviations of ST segment in 12-lead ECG rather than to assess the ECG criteria separately. An easy to-use, simple and accurate algorithm (Figure 7) has been proposed by Fiol *et al*[4] based on the evaluation of ST-segment changes in 12-lead ECG which predicts the site of occlusion in LAD in patients with AWMI. This algorithm (Figure 7) correlated well with angiographic findings. This algorithm consists of 2 steps: first, to locate the occlusion in relation to diagonal 1(D1), and then, to locate the occlusion in relation to septal 1(S1). The ST-segment elevation in leads V_1, V_2, and V_4 through V_6 indicates occlusion in the LAD. To differentiate the occlusion in relation to D1 (proximal versus distal), ST-segment deviations are checked in inferior leads. The sum of ST depression in III and aVF leads 2.5 mm or more indicates an occlusion proximal to D1 (criterion A). The ST-segment, if isoelectric or elevated, indicates that the occlusion is distal to D1 (criterion C). To assess more precisely the location of LAD occlusion in relation to S1, ST-segment in aVR, V1, and V6 should then be checked. If sum of ST deviation in aVR + V_1 – V_6 is 0 or more, occlusion proximal to S1 is suggested. If less than 0, the occlusion is distal to S1 (criterion B). These data suggest that the equation is more useful when the result is less than 0, and presents a high specificity for an occlusion distal to S1. Cases with slight ST-segment depression in III + aVF (<2.5 mm) are difficult to classify in relation to D1, but in these cases the equation: sum of ST deviation in aVR + V_1 – V_6 less than 0 has high specificity for occlusion distal to S1.

Clinical-electrocardiographic correlation

Patients who presented with ECG criteria of proximal occlusion (any ST-depression in III + aVF>0.5 plus sum of ST-deviation in aVR + V_1 – V_6 ≥0) constitute a high-risk group.[4] This high-risk group had lower ejection fraction, higher peak of creatinine phophokinase (CPK) and its isoenzyme MB (CPK-MB), and worse Killip class, and included more patients with major adverse cardiac events

compared to the patients who did not meet all these criteria.

Posterior wall myocardial infarction

The standard 12-lead ECG is a relatively insensitive tool for detecting posterior wall MI (PWMI).[39,40] This is due to the absence of standard leads facing the posterior wall of LV. In upto 50% of patients with PWMI ST-segment elevation is not seen on the standard 12-lead ECG. During acute inferior infarction, ST-segment elevation in the posterior chest leads V_7 through V_9 identifies those patients with concomitant posterior wall involvement.

PWMI can be caused by lesions in the RCA or LCX which may be indirectly recognized by reciprocal ST depression in precordial leads V1-V3. Similar ST changes can also be the primary ECG manifestation of anterior subendocardial ischaemia. Posterior inferior wall infarction with reciprocal changes can be differentiated from primary anterior wall ischaemia by the presence of ST segment elevations in posterior leads, although these are not routinely recorded. Patients with an abnormal R wave in V_1 (0.04 in duration and/or R/S ratio ≥ 1 in the absence of preexcitation or right ventricular hypertrophy), with inferior or lateral Q waves, have an increased incidence of isolated occlusion of a dominant LCx without collateral circulation. These patients have a lower ejection fraction, increased end-systolic volume, and higher complication rate than patients with IWMI caused by isolated RCA occlusion.

LIMITATIONS OF ELECTROCARDIO-GRAM

Assessment of the site of occlusion of a coronary vessel is most reliable in case of a first episode of MI and is impaired by multivessel disease, an old MI, collateral circulation and when ventricular activation is altered as in pre-existent left bundle branch block, preexcitation and paced rhythm. The ST deviation changes with the window period. Hence, these criteria are not so reliable in patients presenting late after the onset of symptoms. The ST deviation may also change with antero-poste-

rior diameter of the chest and various other extracardiac conditions including chronic obstructive pulmonary disease.

These and other criteria proposed for localization of the site of coronary occlusion based on the initial ECG still require additional validation in larger populations. Current and future criteria will always be subject to limitations and exceptions based on variations in coronary anatomy, the dynamic nature of acute ECG changes, the presence of multivessel involvement and the presence of ventricular conduction delays. For example, in some cases, ischaemia can affect more than one region of the myocardium (e.g., inferolateral). Not uncommonly, the ECG will show the characteristic findings of involvement in each region. Sometimes, however, partial normalization can result from cancellation of opposing vectorial forces.

Despite several limitations described above, the ECG can help in identifying the location of occlusion of the coronary arteries in acute MI, thereby helping to predict the amount of myocardium at risk and guide decisions regarding the urgency of revascularization. It is better to apply an easy to-use, sequential algorithm based on deviations of ST in 12-lead ECG rather than to assess the ECG criteria separately.

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