New criteria based on ST changes in 12-lead surface ECG to detect proximal versus distal right coronary artery occlusion in a case of acute inferoposterior myocardial infarction

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Discussion

S-T segment elevation in the inferior leads together with the clinical presentation indicates an evolving myocardial infarction of the infero-posterior wall ***(Fiol et al., 2004)***.

Inferior myocardial infarction in nearly 80% of cases is due to right coronary artery (RCA) occlusion and the rest is by occlusion of the left circumflex artery (LCx) ***(Berger and Ryan 1990).***

Previous autopsy studies indicated that among patients with fatal MI, the right coronary artery was the culprit vessel in (45% - 50%) of patients ***(Mikkelsson, et al., 2004).***

The outcome of patient with RCA as a culprit artery is determined mainly by the location of occlusion ***(Fiol et al., 2004).***

Right coronary artery occlusions proximal to the origin of the right ventricular branch are often associated with hypotension or bradycardia in the acute phase ***(Berger and Ryan, 1990).***

In 60% of individuals, the sino-atrial artery originates from proximal RCA, and sinus arrest is likely to occur in proximal RCA occlusions.

Also, the atria-ventricular (AV) node artery originates from proximal RCA in 86% of individuals ***(Mc Analty and Rahim toola, 1983)***.

Braat, et al. (1984) reported that the incidence of high degree AV block in patients with right ventricular infarction due to proximal RCA occlusion is high (48%).

Moreover, proximal RCA occlusion usually leads to RV involvement and this determines worse prognosis and higher mortality ***(Berger and Ryan, 1990).***

Therefore it's extremely important to recognize the site of RCA occlusion to determine the optimal treatment and management.

Once we have determined by ECG with high probability that RCA is the culprit artery, we may use other ECG criteria to predict proximal versus distal RCA occlusion.

Right ventricular (RV) infarction that usually accompanies the proximal RCA occlusion may be determined on the basis of ST changes in the right precordial leads ***(Wellens et al., 1999).***

ST- segment changes in these leads are very specific but they disappear in the early stage of the evolution of MI. Furthermore, no changes may occur in these leads due to the presence of concomitant lateral or posterior involvement ***(Kosuge et al., 2001)***.

Another limitation of using the right precordial leads is that these leads are not often recorded in the majority of Emergency Rooms. Thus the predictive value of ST changes in these leads is of limited value.

This study involved 50 patients (39 males, 11 females) with first acute inferior wall MI; patients with previous MI or bundle branch block which interfere with

the interpretation of ECG findings were excluded from this study. As well those who proved to have LCx as the culprit vessel were also excluded. Patients with previous coronary artery bypass graft were also excluded to assure that the inferior surface is supplied by the native vessel only.

In the present study, ECG criteria to detect site of occlusion in the right coronary artery in cases of inferior MI were studied.

Coronary angiography was done within one month from the onset of symptoms. The patients were classified into 2 groups:

Group I: in whom the RCA was occluded proximally (proximal to right ventricular artery)

Group II: in whom the RCA was occluded distally (distal to right ventricular artery).

Their ages ranged from 29 to 70 years, it was found that there is no significant difference regarding age and sex distribution among the two study groups.

Regarding the presence of cardiovascular risk factors, both groups were matched and none of the risk factors had any significant statistical difference on the location of occlusion in the RCA.

In a previous study, ***Turhan et al., in 2003***, evaluated the value of ST-segment depression in lead aVL in diagnosing RV infarction in patients with acute inferior MI. They found that ST-segment depression of over than 1mm in lead aVL was very sensitive and specific for RV involvement in patients with acute inferior MI when compared to ST elevation in V4R (the golden criteria).

In our study, this old criterion (ST segment depression in lead aVL $\geq$ 1mm) was examined and it was 88% sensitive and 20% specific in differentiating proximal versus distal RCA and it was non significant (P> 0.05).

As this criterion was found only in 36% of patients with proximal RCA versus 12% in patients with distal RCA and this was not statistically significant and this criterion was considered not valuable in predicting the site of occlusion in clinical practice.

The study done by ***Turhan et al., in 2003*** examined aVL for diagnosis of RV infarction in inferior MI and indirectly detects the location of RCA occlusion as it is proximal by the presence of RV infarction. Also coronary angiography was not done.

On the other hand; we also assessed the new criterion of simultaneous changes in leads I and aVL (sum of ST-segment depression in leads I and aVL $\geq$ 5.5 mm) and it was 36% sensitive and 88% specific and it was significant in differentiating proximal versus distal RCA (P<0.05) with PPV 75% and NPV 58%.

These results were comparable with the results of ***Fiol, et al. (2004*** who found that this criterion was 91% specific and 40% sensitive.

The criterion of ST-segment changes in lead V1 (Isoelectric, elevated or depressed) was also assessed and it had the highest accuracy as fallows:

Isoelectric or elevated ST-segment in lead V1 was found in 88% of patients with proximal RCA versus 16% in patients with distal RCA with 88% sensitivity & 84% specificity, PPV was 85%& NPV was 87%. And there was

a highly significant difference between the two groups regarding the presence or absence of this criterion ($P<0.001$).

These results were comparable to the results by ***Fiol et al., 2004*** who found that this criterion was 87% specific and 70% sensitive for detection of proximal RCA occlusion.

These result were in agreement with ***Tuska et al., 2001*** who studied One hundred fifty-eight consecutive patients with acute Q-wave inferior wall myocardial infarction were classified into 3 groups on the basis of the initial ST-change in V1 (group 1 = 29 patients with ST elevation, group 2 = 97 patients with ST depression, and group 3 = 32 patients with no ST-segment change).The right coronary artery was the infarct-related artery in all the patients in group 1. Patients in group 1 had a significantly higher incidence of proximal lesion (86%) and right ventricular infarction (69%) than the other 2 groups did.

When combining the two new criteria of the sum of ST-segment depression in lead I and aVL $\geq$5.5mm, and iso-electric or elevated ST-segment in lead V1, the specificity and PPV increased to 100% to diagnose proximal RCA but the sensitivity decreased to 56%.

These results were comparable to the results by ***Fiol , et al. (2004)*** who found this combination 100% specific but 7% sensitive.

As regard the incidence of RV infarction, this study detected RV infarction as a complication of AIMI in a frequency of 22% these result were similar to that obtained by Bigrie with his co- workers reported 24% of patients with AIMI and RV involvement ***(Bigrie et al., 1983)***.

However, Buena and his group ***Buena et al., 1997*** and Lew and his workers recognized the presence of RV involvement in 48% of patients perhaps refer to the criteria used in the diagnosis of RV infarction ***Lew et al., 1985*** which include any of the following:
-Physical examination (JVP $\geq$ 3cm with either Kussmal" s sign or clear lung field).
-Echocardiography (RV dilatation or RV asynergy).
- Coronary angiography (occlusion or severe stenosis of the RCA proximal to RV branch.

As for the incidence of RV infarction in group I with proximal RCA occlusion there were 11 patients (44%), while in group II non had RV infarction, with significant difference between the two groups ($P<0.05$).

Regarding the incidence of AV block there was also a significant difference between the two groups with 8 patients (32%) in patients with proximal RCA occlusion versus o patients in the other group ($P<0.05$).

Which was comparable to the study done by ***Braat SH, et al 1984*** who studied 67 patients with inferior wall acute MI found that 29 patients had RV involvement, diagnosed by scintigraphy. 14 patients of these 29 patients had AV nodal conduction disturbance.

The incidence of high degree AV block in patients with right ventricular infarction due to proximal RCA occlusion is high (48%).

Patients with proximal RCA had higher peak creatine phosphokinase values with significant difference between the two study groups ($p< 0.001$).

From this study we found that the ECG can be a guide to diagnose the site of RCA occlusion in those with inferior MI. Patients with proximal RCA occlusion tends to have high CPK level, higher degree of AV block, and increased incidence of RV infarction.

The ECG in 88% of patients with proximal RCA occlusion showed isoeletric or elevated ST segment in lead V1with 84% specificity which increased to 100% when combining this criterion to sum of ST segment in leads I and aVL $\geq$5.5mm.

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Patients and Methods

The study was conducted on 50 patients who were admitted to (CCU) of National Heart Institute with the diagnosis of first time acute inferior myocardial infarction in whom the coronary angiography revealed single right coronary artery significant lesion.

The diagnosis of acute inferior myocardial infarction was established when the following criteria were present.

Inclusion criteria:-

1- Severe prolonged chest pain for more than 30minutes suggestive of MI.

2- Characteristic electrocardiographic changes in the form of ST segment elevation $\geq$1mm in at least two of inferior leads (II, III, aVF).

3- Serial elevation of specific cardiac enzyme "creatine phosphokinase and lactate dehydrogenase" >2 times of the upper normal limit.

Exclusion criteria:-

1- Patients with history or ECG evidence of previous myocardial infarction.

2- Late presentation "onset of symptoms more than 12 hours.

3- Patients with ECG evidence of left or right bundle branch block.

4- Patients with previous coronary artery bypass surgery.

5- Patients with right coronary artery stenosis of less than 70%of its luminal diameter.

6- Patients in whom the coronary angiography revealed evidence of significant stenosis in more than one vessel.

Methodology

All patients were submitted to the following:-

A- Full history analysis including:-

1- Personal history:
Age- gender –smoking.

2- Present history:
Presence of chest pain: onset, course, duration, site, typical or atypical.

3- Past history:
Ischemic events- myocardial infarction- CABG.

4- History of diabetes- hypertension- dyslipidemia

5- Family history of ischemic heart disease.

B- Complete general and local examination.

C- Routine laboratory investigations:-
Including blood sugar- lipid profile-serum cardiac enzymes (creatine phosphokinase and lactate dehydrogenase- serum aspartate aminotransferase) on admission, every 8 hours during 1st 24 hours, and daily till discharge.